San Francisco Bay Area Rapid Transit District

2150 Webster Street, P. O. Box 12688, Oakland, CA 94604-2688

COMMITTEE MEETING AGENDA

Monday, May 9, 2022

4:00 PM

You may join and listen to the BPCRB Meeting by calling 1 833 827 2778 and entering access code 157 362 755#
via Teleconference Only.

BART Police Citizen Review Board

SAN FRANCISCO BAY AREA RAPID TRANSIT DISTRICT
2150 Webster Street, P. O. Box 12688, Oakland, CA 94604-2688

NOTICE OF MEETING AND AGENDA
BART POLICE CITIZEN REVIEW BOARD
May 9, 2022
4:00 p.m.

A regular meeting of the BART Police Citizen Review Board (BPCRB) will be held on Monday, May 9, 2022, at 4:00 p.m., via teleconference only.

Please note, pursuant to all necessary findings having been made by the Board of Directors of the San Francisco Bay Area Rapid Transit District (for itself as well as all subordinate legislative bodies) to continue remote public meetings in the manner contemplated under urgency legislation Assembly Bill No. 361, public participation for this meeting will be via teleconference only.

Presentation materials will be available at least 72 hours prior to the BPCRB meeting at https://bart.legistar.com/Calendar.aspx (click on “Agenda”).

You may join and listen to the BPCRB Meeting by calling 1 833 827 2778 and entering access code 157 362 755#

You may join the Microsoft Teams meeting with this link:

https://teams.microsoft.com/l/meetup-join/19%3ameeting_YTRhN2ZlNzEtNDU3My00YzhlLTlmZTgtMDRmMzQ0NWUyZDMy%40thread.v2/0?context=%7b%22Tid%22%3a%2256c41995-d45f-4669-a533-30fdded094d9%22%2c%22Oid%22%3a%222abd3ac4-add6-4c1c-804d-063f9fc6b7b6%22%7d

We strongly encourage public comments to be submitted via email. You may submit comments via email to CitizenReviewBoard@bart.gov using “public comment” as the subject line. Your comment will be provided to the Board and will become a permanent part of the file. Please submit your comments as far in advance as possible. Emailed comments must be received before 9:00 a.m. in order to be included in the record.

Individuals may also be given an opportunity by the moderator to speak on any item on the agenda by calling 1 833 827 2778 and entering access code 157 362 755# in advance of the item. Public comment will be limited to three (3) minutes per person. Your phone will be muted until you are called upon.

1. Call to Order.
a. Roll Call.
b. Pledge of Allegiance.

2. Approval of Minutes of the Meeting of April 11, 2022. For Action.

Attachments: Approval of Minutes of the Meeting of April 11, 2022

3. Reporting Out Announcement from the Closed Session of April 11, 2022, if any.

4. Dissenting Opinion Memo for Office of the Independent Police Auditor (OIPA) Case #21-10 (Member Todd Davis' Request). For information.

Attachments: Dissenting Opinion Memo - Member Todd Davis' Request

5. Reporting Out Announcement from BART Police Citizen Review Board (BPCRB) Subcommittees. For Discussion.

6. Chief of Police's Report(s). For Discussion.
a. BART Police Department's Monthly Reports for March 2022.
b. BART Police Department's California Assembly Bill 481 - Military Equipment Funding, Acquisition and Use Policy (Verbal Report Only).

Attachments: BART Police Department's Monthly Reports for March 2022

7. Independent Police Auditor's Report (s). For Discussion.
a. Office of the Independent Police Auditor (OIPA) Monthly Report for April 2022.
b. Use of the Term of "Excited Delirium" and its effects on Racial Equity

Attachments: OIPA Monthly Report - April 2022
OIPA's Excited Delirium - Presentation
Attachment A - American Medical Association (AMA) New Policy Opposes "Exci
Attachment B - American College of Emergency Physicians (ACEP) Task Force
Attachment C - Police 1 Research Review Naked, but Dangerous
Attachment D - Force Science Article Sidestepping the Excited Delirium Debate
Attachment E - Physicians for Human Rights (PHR) Report Excited Delirium and
Attachment F - BPD Bulletin No. 22-103 Use of the Recovery Position

8. Public Comment. (Limited to 3 minutes per speaker.) (An opportunity for members of the public to address the BPCRB on matters under their jurisdiction and not on the agenda.)

9. Closed Session.
a. To Consider Public Employee Discipline/Dismissal/Release in OIPA Case #22-17. Govt. Code §54957.

All BPCRB closed session votes will be announced at the beginning of the next regular meeting.

10. Adjournment.

BART provides service/accommodations upon request to persons with disabilities and individuals who are limited English proficient who wish to address Board matters. A request must be made within one and five days in advance of Board meetings, depending on the service requested. Please contact the Office of the District Secretary at (510) 464-6083 for information.

Pursuant to Govt. Code §54953.5, the audio recording of the open session portions of this public meeting shall be subject to inspection pursuant to the California Public Records Act (CPRA). Requests for information under the CPRA should be filed with the BART Office of the District Secretary.

SAN FRANCISCO BAY AREA RAPID TRANSIT DISTRICT
2150 Webster Street, P.O. Box 12688, Oakland, CA 94604-2688
BART Police Citizen Review Board Meeting Minutes
Monday, April 11, 2022

A regular meeting of the BART Police Citizen Review Board (BPCRB) was held Monday, April 11, 2022, convening at 4:03 p.m. via teleconference, pursuant to Assembly Bill No. 361. The meeting was called to order by Vice Chairperson Pete Longmire; Mag Tatum, Recording Secretary.

Vice Chairperson Pete Longmire gave instructions on the virtual meeting, accessing the presentation materials online, Public Comment, and Members' remarks.

1. **Call to Order.**

   The regular meeting was convened at 4:03 p.m. by Chairperson Vice Chairperson Pete Longmire.

Members Present:	Members Todd Davis, Christina Gomez, Dana Lang, Pete Longmire, Les Mensinger, Laura Pagey, George Perezvelez, David Rizk. William White, and Erin Armstrong.
Absent:	Member Pedro Babiak.

   The Pledge of Allegiance was recited.

2. **Approval of Minutes of the Meeting of March 14, 2022.**

   Member Perezvelez moved that the Minutes of the Meeting March 14, 2022, be approved. Member Mensinger seconded the motion, which carried by roll call vote. Ayes – 9: Members Gomez, Lang, Longmire, Mensinger, Pagey, Perezvelez, Rizk, W. White, and Armstrong. Noes – 0. Abstain – 1: Member Davis. Absent – 1: Member Babiak.

3. **Reporting Out Announcement from the Closed Session of March 14, 2022**.

   Chairperson Armstrong announced that the Board voted unanimously to accept the findings in Office of the Independent Police Auditor (OIPA) Case #21-18, from the March 14, 2022, Closed Session.

4. **Reporting Out Announcement from BART Police Citizen Review Board (BPCRB) Subcommittees.**

   Chairperson Armstrong and Member Gomez presented reporting out information for various BART Police Citizen Review Board (BPCRB) Subcommittees. The item was discussed.

   Member David Rizk entered the meeting.

5. **Chief of Police's Reports.**
   a. **BART Police Department's Monthly Reports for February 2022.**

   Chief of Police Ed Alvarez presented the BART Police Department's Monthly Reports for February 2022. The reports were discussed.

   Aleta Dupree addressed the Board.

   b. **BART Police Department's Progressive Policing and Community Engagement Bureau Presentation.**

   Deputy Police Chief Angela Averiett presented the BART Police Department's Progressive Policing and Community Engagement Bureau. The item was discussed.

6. **Independent Police Auditor's (OIPA) Report.**
   a. **Office of the Independent Police Auditor Monthly Report for March 2022.**

   Independent Police Auditor Russell Bloom presented OIPA Monthly Report for March 2022. The report was discussed.

7. **Review of BPCRB Subcommittee Structure and Membership (Chair Armstrong's Request.)**

   Chairperson Armstrong presented the Review of BPCRB Subcommittee Structure and Membership. The item was discussed.

   Member Rizk presented information for the BART Ad Hoc Working Group on Fare Evasion.

   Chairperson Armstrong presented information for the BART Citizen Oversight Model Standing Subcommittee.

8. **Public Comment.**

   Chairperson Armstrong called for general Public Comment.

   Aleta Dupree addressed the Board.

9. **Closed Session.**

   a. To Consider Public Employee Discipline/Dismissal/Release in OIPA Case Number #21-10. Govt. Code §54957.

   Chairperson Armstrong announced that the Board would enter closed session under Item 9-A (Public Employee Discipline/Dismissal/Release in OIPA Case Numbers #21-10) of the Regular Meeting agenda and that the closed session votes, if any, would be announced in open session at the beginning of the next regular BPCRB meeting.

10. **Adjournment.**

    The Meeting was adjourned at 5:55 p.m.

Dear Bart Board of Directors:

I appreciate the opportunity to address this agency regarding the recent OIPA case 21-10. I want to explain why I dissented from the Bart Police's Citizen Review Board acceptance of the findings from the OIPA. In this case, the complainants' allegations included Biased Based Policing by the Bart Police officers towards the detained juveniles. OIPA did not agree with this assertion and found that it did not have merit. I respect the purpose and intent of the office to find out the facts of the incident, identify areas of improvement, and bring to light/accountability the departments missteps. However, I believe they overlooked the gravity of handcuffing the juveniles. According to policy, there are special considerations that should be taken when deciding to handcuff someone, especially minors. The policy lists multiple requirements when deciding to handcuff and I want to highlight the considerations brought up during the interviews: compliance and flight risk.

The non-compliance standard for handcuffing was not met.

- The officers that interacted with the youth described the youth as compliant and not posing a threat.
- Based on body camera footage, the kids answered all the officers' questions. and for most part truthfully. They provided the contact info for their parents so that officers could inform them of the issue and verify their statements.
- The officers stated that the youths' behavior did not change until AFTER they were handcuffed for at least forty-five minutes later.
- They all agreed that the youth behavior did not change until much later (roughly 45min later) once a crowd developed to observe their actions. I posit, how many of us would not have been as cooperative as we were in the beginning of an interaction with the police once they not only detained us but put tight, uncomfortable restricting objects on our wrists for almost an hour. [POSSIBLE REMOVAL]
-

The flight risk standard for handcuffing was not met.

- An officer incorrectly applied the "flight risk" standard to the youths based on his belief that they fled the scene.
- In this case, leaving the BART station after completing a trip was equated to "fleeing the scene".
- The policy defines "fleeing the scene" as person(s) posing a risk of leaving the officers before they can determine whether they have enough evidence to demonstrate the suspect committed the crime.
- In this case, no officers suggested that the kids attempted to leave before they asked their question. The officers all agreed the kids were compliant.

Based on the available information and reading of the handcuffing policy, there did not appear a valid reason for why the officers handcuffed the youths. The action was incorrect, and therefore I dissented from the Bart Police's Citizen Review Board acceptance of the findings from the OIPA.

I feel it is important to explain this decision, and how the incorrect application of the handcuffing policy initiated a tense situation with the community and further eroded the trust between the community and BART police officers.

So once again I ask, why the need for handcuffs. It seems to me the reason stemmed from the later answers that the officers provided regarding the incident. When asked whether the kids posed a danger to the officers they stated no; however, multiple officers suggested that the crowd that began to form (ironically because of the handcuffed youth) and their unfamiliarity of the area exacerbated their fear.

However, these are not valid reasons to handcuff minors for almost an hour. Moreover, why were the officers afraid? Like the youth, the community did not threaten or physically harm the officers. A decent number of concerned cooperative community members, calmly spoke with the officers, discussed their, concerns and even thanked the officers for answering questions.

These aren't actions of youth or adults that should warrant fear unless the officers already held predetermined beliefs about this specific group of community members and youth.

I assert their bias against African Americans contributed to their fear; and despite the cooperation, willingness to answer questions and patience of the African American adults and youth during a tense ordeal made no difference to the members of Bart Police present on that day; the officers blinded by color decided to handcuff the kids and further fan the anger of the community.

-Todd Davis

Bart Citizen Review Board Member

BART POLICE DEPARTMENT

March 2022

MONTHLY REPORT

Bay Area Rapid Transit Police Department

101 8th St, Oakland, CA, 94607 (510) 464-7000 www.bart.gov/police

March 2022 *Performance Measurement Review - Systemwide*

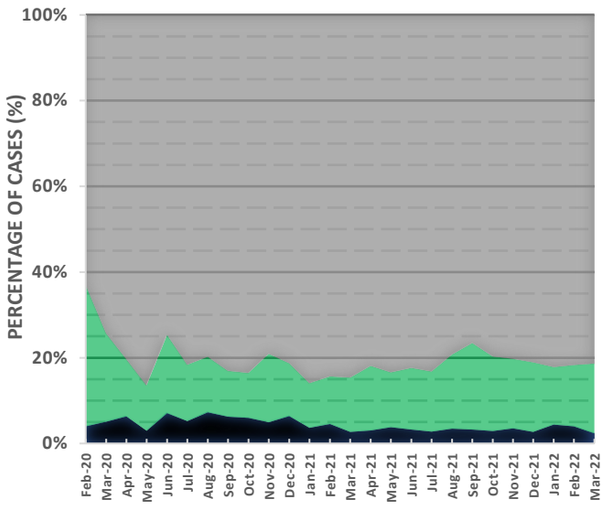
PART 1 UCR Crime	2017	2018	2019	2020	2021	YTD 2021	YTD 2022	PCT %
Homicide	0	3	2	0	0	0	2	-%
Rape	8	3	7	5	8	0	2	-%
Robbery	290	345	378	252	143	42	39	-7%
Aggravated Assault	125	130	112	95	71	10	17	+70%
Violent Crime Subtotal	423	481	499	352	222	52	60	+15%
Burglary (Structural)	15	18	16	12	11	0	4	-%
Larceny & Auto Burglary	2,593	2,565	3,177	1,038	882	156	203	+30%
Auto Theft	420	348	247	100	134	12	41	+242%
Arson	4	4	4	4	5	0	3	-%
Property Crime Subtotal	3,032	2,935	3,444	1,154	1,032	168	251	+49%
TOTAL	3,455	3,416	3,943	1,506	1,254	220	311	+41%

Part 1 Crimes: Top Five Stations

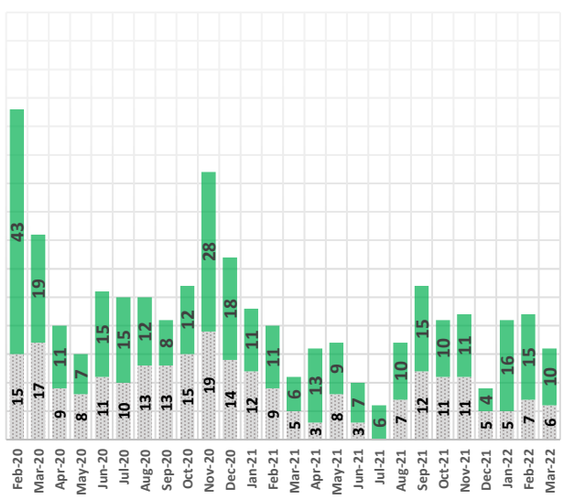
March 2022 K30/MAS A30/COS A40/SLS A70/SHS A20/FVS

Full Year 2021 A30/COS A60/HAS M10/OWS A10/LMS A20/FVS

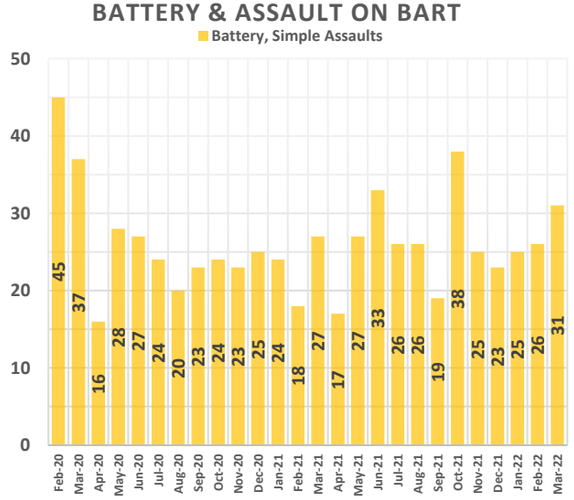
PART 1 CRIME OVERALL



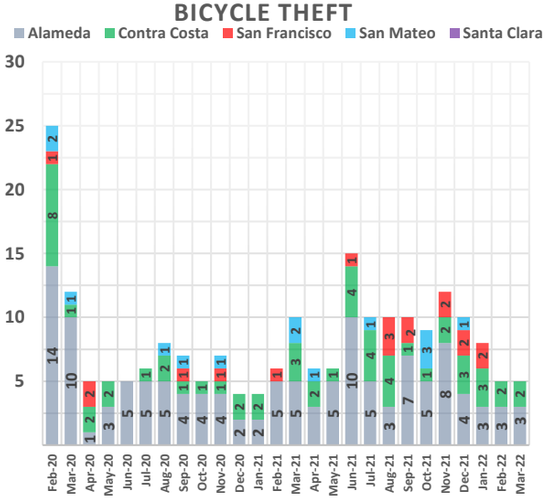
ELECTRONIC THEFT



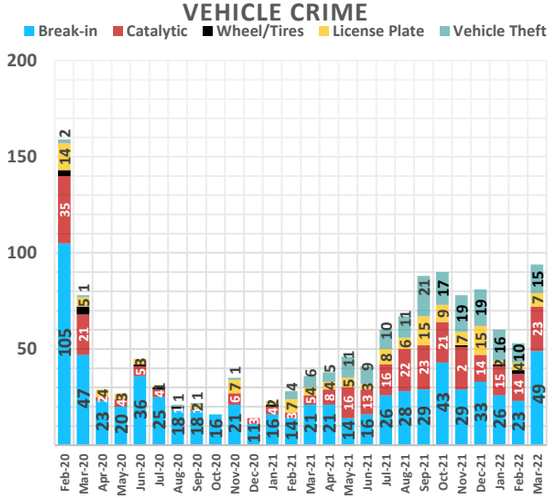
BATTERY & ASSAULT ON BART



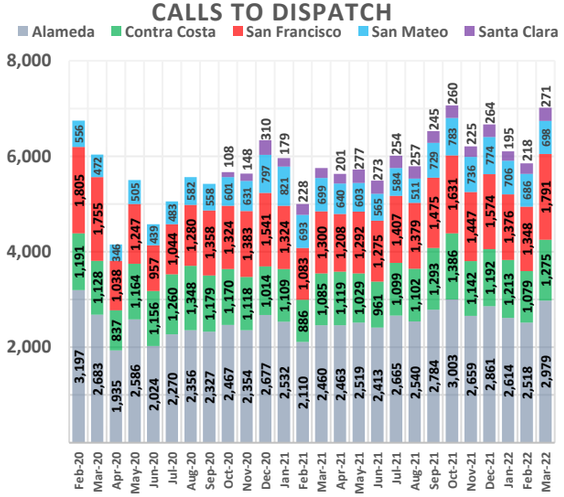
BICYCLE THEFT



VEHICLE CRIME



CALLS TO DISPATCH



Preface: *The data is retrieved from the BART Police Database and remains unaudited until corrections. Numbers may differ from the reported data in the Uniform Crime Reporting (UCR) program.*

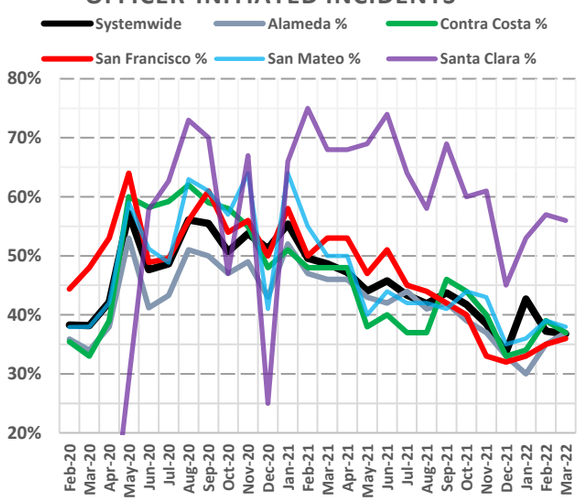
Late reporting, the reclassification or unfounding of crimes, can affect crime statistics. Overtime costs are projected numbers. Information provided on the reports are subject to change.

Bay Area Rapid Transit Police Department

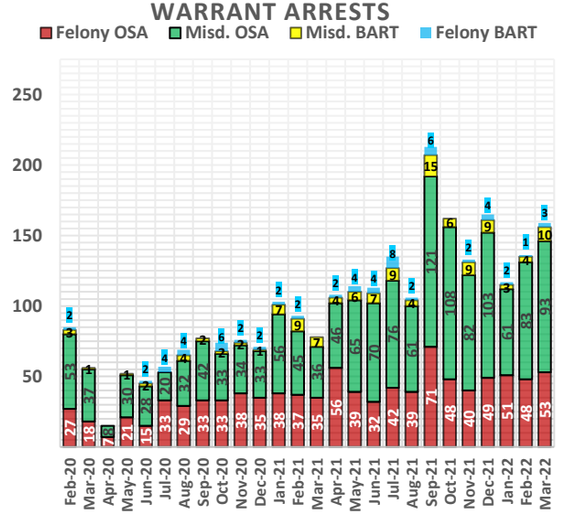
101 8th St, Oakland, CA, 94607 (510) 464-7000 www.bart.gov/police

March 2022 *Performance Measurement Review - **Systemwide***

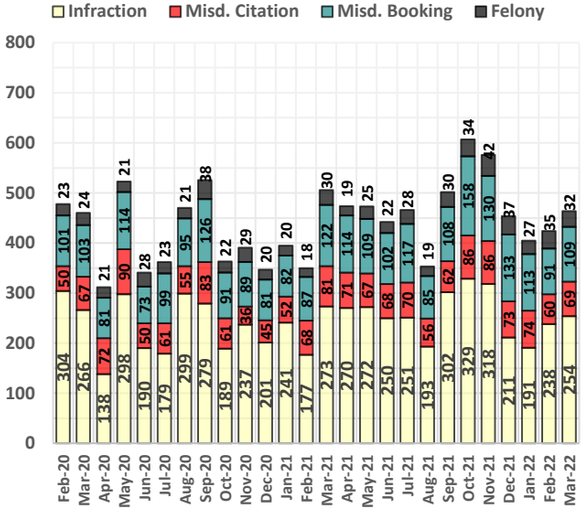
OFFICER-INITIATED INCIDENTS



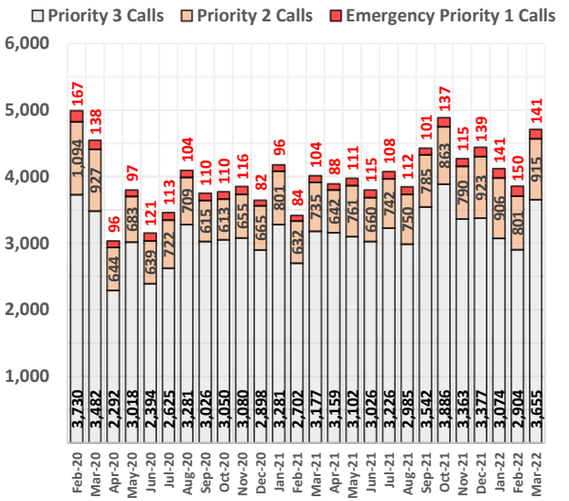
WARRANT ARRESTS



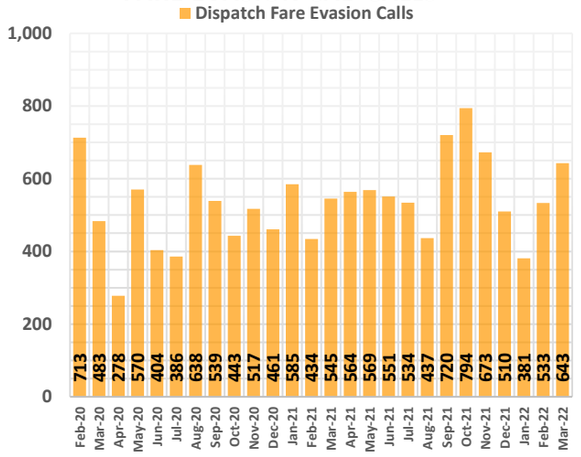
ALL BOOKINGS & ARRESTS



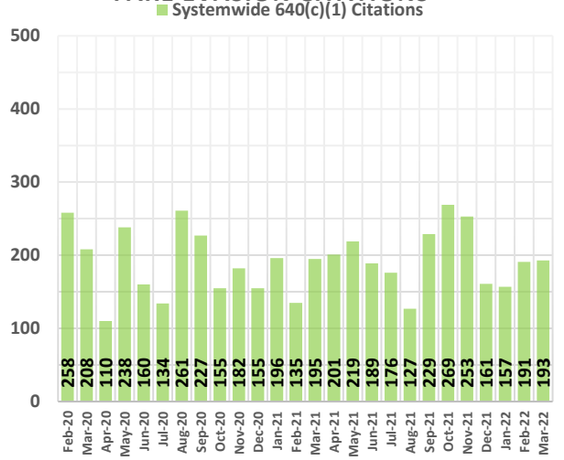
PRIORITY TYPE 1 - 3 CALLS



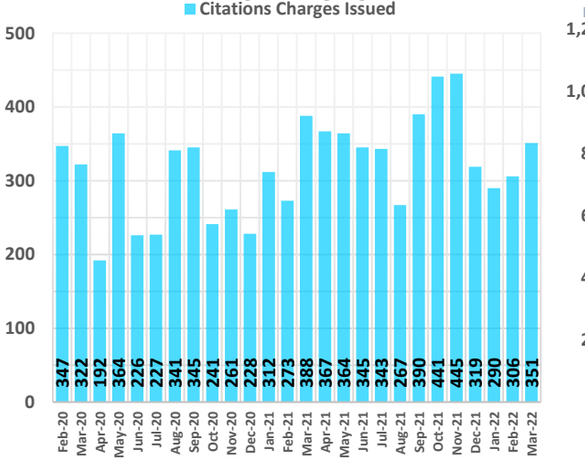
FARE EVASION CAD CALLS



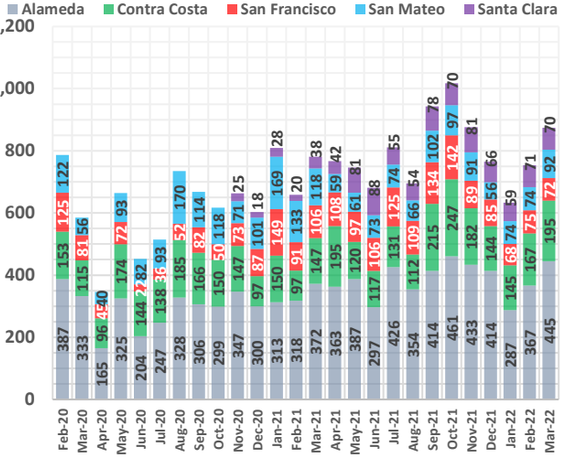
FARE EVASION CITATIONS



ALL CITATIONS



FIELD INTERVIEWS



Preface: *The data is retrieved from the BART Police Database and remains unaudited until corrections. Numbers may differ from the reported data in the Uniform Crime Reporting (UCR) program.*

Late reporting, the reclassification or unfounding of crimes, may affect statistics. Infraction citations consist of all individual charges. Felony arrests, misdemeanor arrests and citations are based on each instance per individual

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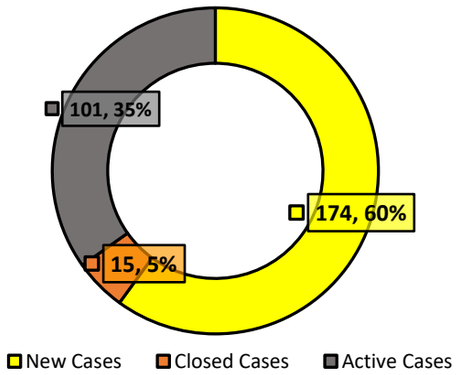
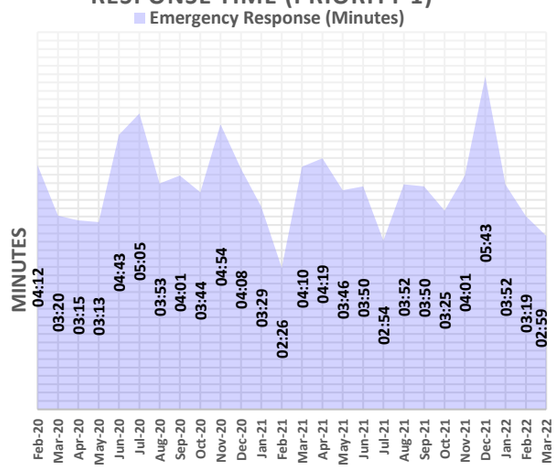
March 2022 *Performance Measurement Review - **Systemwide***

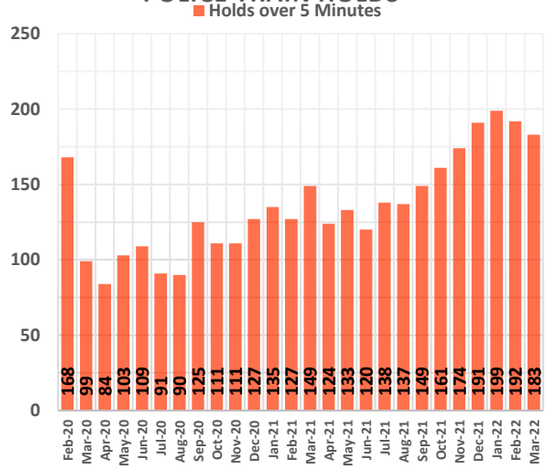
INVESTIGATIVE ASSIGNMENTS



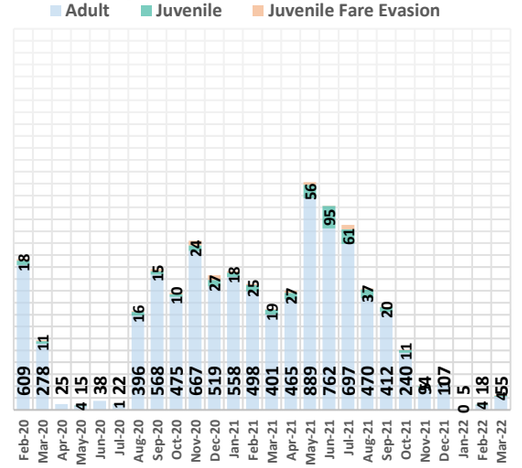
RESPONSE TIME (PRIORITY 1)



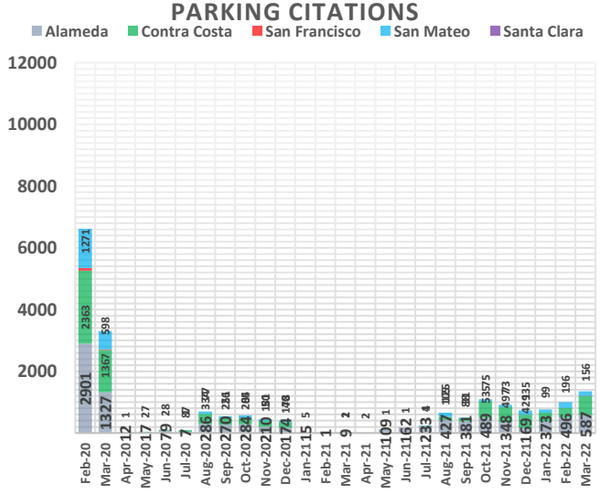
POLICE TRAIN HOLDS



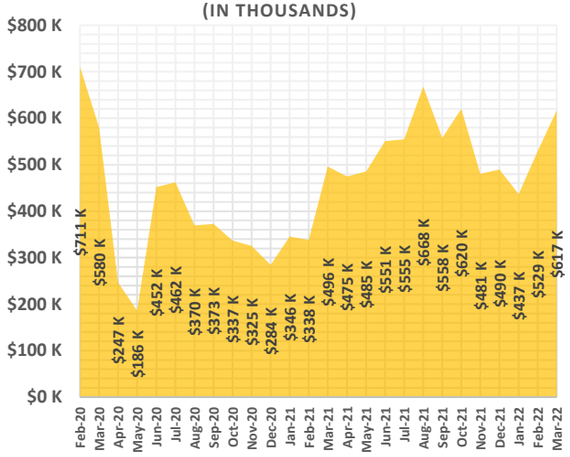
PROOF OF PAYMENT CITATIONS



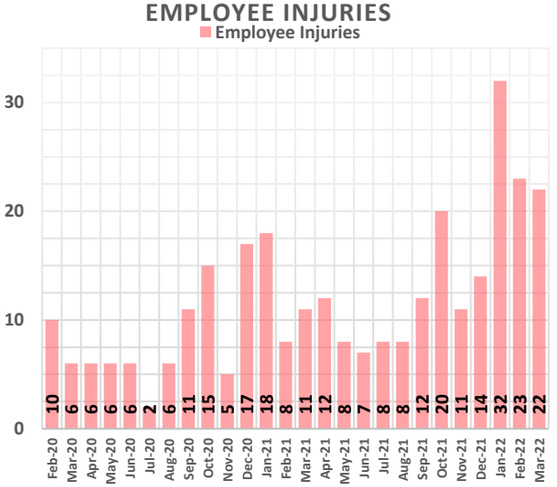
PARKING CITATIONS



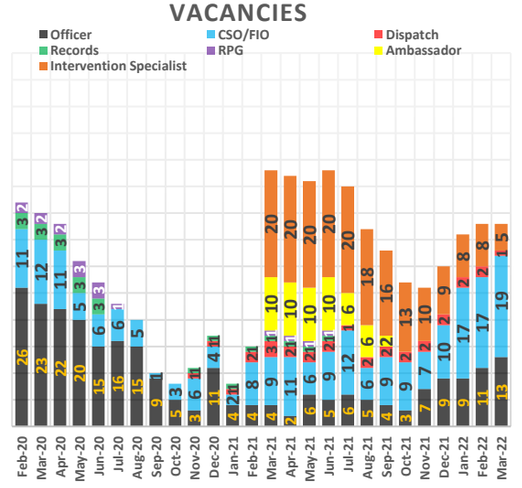
OVERTIME UTILIZATION (IN THOUSANDS)



EMPLOYEE INJURIES



VACANCIES



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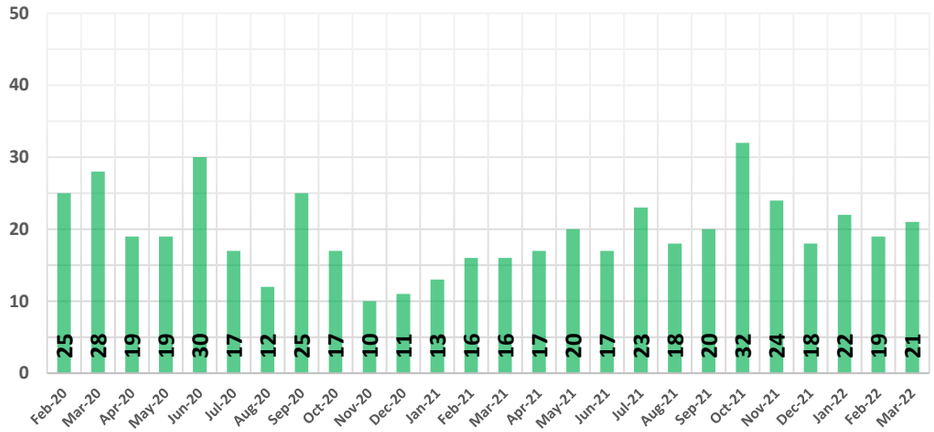
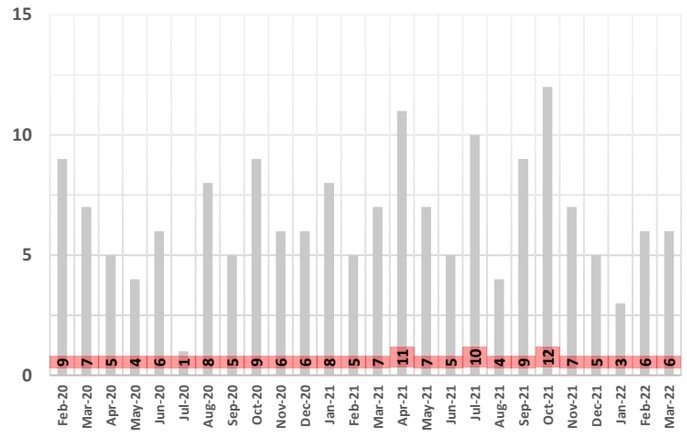
March 2022 *Performance Measurement Review - Systemwide*

March 2022

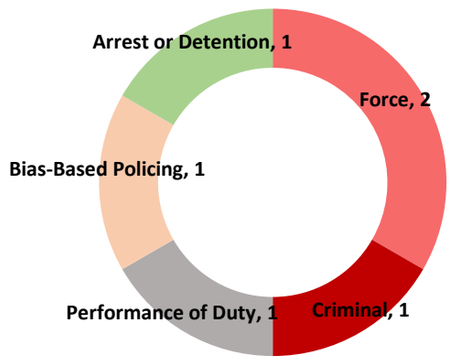
AB716 - PROHIBITION ORDERS



INTERNAL AFFAIRS COMPLAINTS

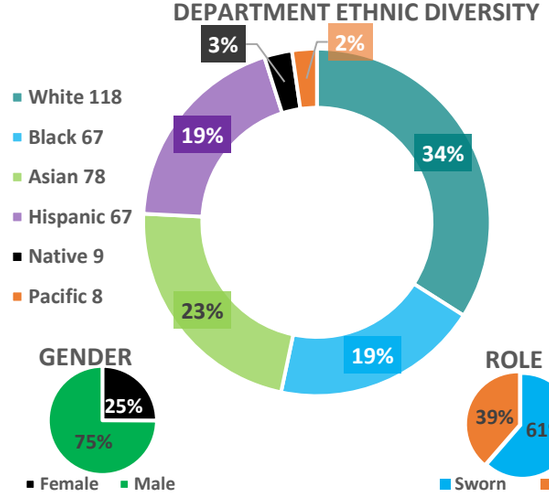


IA COMPLAINTS RECEIVED[1]

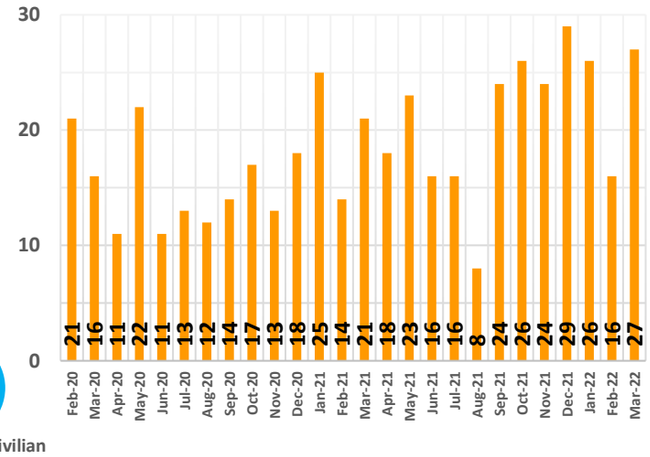


1: Each incident could contain more than one allegation. This chart reflects the most significant allegation per

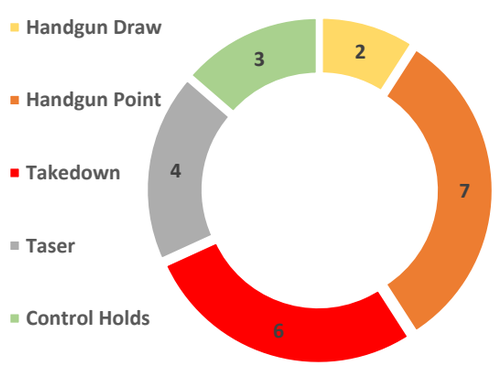
DEPARTMENT ETHNIC DIVERSITY



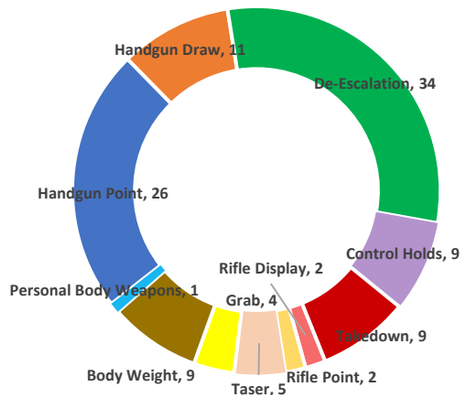
USE OF FORCE INCIDENTS



INCIDENT FORCE OPTIONS USED[2]



TYPES OF FORCE USED[3]



2: Each incident could contain more than one force option. This chart reflects most significant force options used per incident.

3: Some incidents involved the use of multiple force options. If two officers involved in the same incident used the same force option, this data would reflect both officers. As an example, if two officers used control holds in the same incident, this data would reflect two separate control holds.

Bay Area Rapid Transit Police Department

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March 2022 *Performance Measurement Review -* ***Systemwide***

BART PD Uses of Force													
Use of Force Incidents	**Jan**	**Feb**	**Mar**	**Apr**	**May**	**Jun**	**Jul**	**Aug**	**Sep**	**Oct**	**Nov**	**Dec**	**Total**
2022 CY	26	16	27										69
2021 CY	25	14	21	18	23	16	16	8	24	26	24	29	244
2020 CY	22	21	16	11	22	11	13	12	14	17	13	18	190
2019 CY	27	20	17	30	21	19	27	27	26	21	14	28	277
2022 YTD	26	42	69										
2021 YTD	25	39	60	78	101	117	133	141	165	191	215	244	
2020 YTD	22	43	59	70	92	103	116	128	142	159	172	190	
2019 YTD	27	47	64	94	115	134	161	188	214	235	249	277	

BART PD Dispatch Communications Center													
Dispatch Center Calls	**Jan**	**Feb**	**Mar**	**Apr**	**May**	**Jun**	**Jul**	**Aug**	**Sep**	**Oct**	**Nov**	**Dec**	**Total**
2022 Calls for Service	6,087	5,802	7,138										19,027
2022 Priority 1 Calls	141	150	141										432
2022 Medical Emergencies	302	255	286										843
2022 Avg P1 Response Time	03:52	03:19	02:59										03:23
2021 Calls for Service	5,966	5,076	5,827	5,706	5,756	5,491	6,017	5,807	6,530	7,031	6,184	6,670	72,061
2021 Priority 1 Calls	96	84	104	88	111	115	108	112	101	137	115	139	1,310
2021 Medical Emergencies	266	191	193	193	197	181	209	195	228	232	199	294	2,578
2021 Avg P1 Response Time	03:29	02:26	04:10	04:19	03:46	03:50	02:54	03:52	03:50	03:25	04:01	05:43	03:49
2020 Calls for Service	7,470	6,753	6,086	4,242	5,583	4,770	5,319	6,008	5,715	5,835	5,678	5,499	68,958
2020 Priority 1 Calls	191	167	138	96	97	121	113	104	110	110	116	82	1,445
2020 Medical Emergencies	306	295	273	173	168	166	176	156	154	125	160	178	2,330
2020 Avg P1 Response Time	04:02	04:12	03:20	03:15	03:13	04:43	05:05	03:53	04:01	03:44	04:54	04:08	04:02
2019 Calls for Service	7,523	7,785	7,829	7,698	7,449	6,676	6,746	7,182	6,770	7,138	6,301	7,643	86,740
2019 Priority 1 Calls	203	181	202	204	202	213	205	199	222	205	192	192	2,420
2019 Medical Emergencies	361	310	370	321	396	360	318	323	339	329	329	381	4,137
2019 Avg P1 Response Time	05:16	05:10	04:49	04:40	03:52	06:17	04:38	06:45	05:34	05:30	04:57	03:47	05:06

Bay Area Rapid Transit Police Department

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March 2022 Performance Measurement Review - **Systemwide**

BART PD Enforcement Contacts													
Felony Arrests	**Jan**	**Feb**	**Mar**	**Apr**	**May**	**Jun**	**Jul**	**Aug**	**Sep**	**Oct**	**Nov**	**Dec**	**Total**
2022 Felony Arrest	27	35	32										94
2021 Felony Arrest	20	18	30	19	25	22	28	19	30	34	42	37	324
2020 Felony Arrest	51	23	24	21	21	28	23	21	38	22	29	20	321
2019 Felony Arrest	35	41	27	29	35	25	33	43	38	25	32	22	385
2022 Felony Arrest YTD	27	62	94										
2021 Felony Arrest YTD	20	38	68	87	112	134	162	181	211	245	287	324	
2020 Felony Arrest YTD	51	74	98	119	140	168	191	212	250	272	301	321	
2019 Felony Arrest YTD	35	76	103	132	167	192	225	268	306	331	363	385	
Misdemeanor Arrests	**Jan**	**Feb**	**Mar**	**Apr**	**May**	**Jun**	**Jul**	**Aug**	**Sep**	**Oct**	**Nov**	**Dec**	**Total**
2022 Misd. Arrest	113	91	109										313
2021 Misd. Arrest	82	87	122	114	109	102	117	85	108	158	130	133	1,347
2020 Misd. Arrest	124	101	103	81	114	73	99	95	126	91	89	81	1,177
2019 Misd. Arrest	138	169	147	142	127	101	108	119	92	115	89	118	1,465
2022 Misd. Arrest YTD	113	204	313										
2021 Misd. Arrest YTD	82	169	291	405	514	616	733	818	926	1,084	1,214	1,347	
2020 Misd. Arrest YTD	124	225	328	409	523	596	695	790	916	1,007	1,096	1,177	
2019 Misd. Arrest YTD	138	307	454	596	723	824	932	1,051	1,143	1,258	1,347	1,465	
Cite & Releases	**Jan**	**Feb**	**Mar**	**Apr**	**May**	**Jun**	**Jul**	**Aug**	**Sep**	**Oct**	**Nov**	**Dec**	**Total**
2022 Cite & Release	74	60	69										203
2021 Cite & Release	52	68	81	71	67	68	70	56	62	86	86	73	840
2020 Cite & Release	68	50	67	72	90	50	61	55	83	61	36	45	738
2019 Cite & Release	107	138	112	83	64	72	62	74	47	81	52	83	975
2022 Cite & Release YTD	74	134	203										
2021 Cite & Release YTD	52	120	201	272	339	407	477	533	595	681	767	840	
2020 Cite & Release YTD	68	118	185	257	347	397	458	513	596	657	693	738	
2019 Cite & Release YTD	107	245	357	440	504	576	638	712	759	840	892	975	
Field Interviews	**Jan**	**Feb**	**Mar**	**Apr**	**May**	**Jun**	**Jul**	**Aug**	**Sep**	**Oct**	**Nov**	**Dec**	**Total**
2022 Field Interview	633	754	874										2,261
2021 Field Interview	809	659	781	767	746	681	811	695	943	1,017	876	765	9,550
2020 Field Interview	719	787	585	346	665	464	538	766	696	629	663	603	7,461
2019 Field Interview	841	1,011	868	945	765	571	608	714	696	810	561	699	9,089
2022 Field Interview YTD	633	1,387	2,261										
2021 Field Interview YTD	809	1,468	2,249	3,016	3,762	4,443	5,254	5,949	6,892	7,909	8,785	9,550	
2020 Field Interview YTD	719	1,506	2,091	2,437	3,102	3,566	4,104	4,870	5,566	6,195	6,858	7,461	
2019 Field Interview YTD	841	1,852	2,720	3,665	4,430	5,001	5,609	6,323	7,019	7,829	8,390	9,089	
Combined Contacts	**Jan**	**Feb**	**Mar**	**Apr**	**May**	**Jun**	**Jul**	**Aug**	**Sep**	**Oct**	**Nov**	**Dec**	**Total**
2022 Monthly Enf. Contacts	847	940	1,084										2,871
2021 Monthly Enf. Contacts	963	832	1,014	971	947	873	1,026	855	1,143	1,295	1,134	1,008	12,061
2020 Monthly Enf. Contacts	962	961	779	520	890	615	721	937	943	803	817	749	9,697
2019 Monthly Enf. Contacts	1,121	1,359	1,154	1,199	991	769	811	950	873	1,031	734	922	11,914
2022 Enf. Contacts YTD	847	1,787	2,871										
2021 Enf. Contacts YTD	963	1,795	2,809	3,780	4,727	5,600	6,626	7,481	8,624	9,919	11,053	12,061	
2020 Enf. Contacts YTD	962	1,923	2,702	3,222	4,112	4,727	5,448	6,385	7,328	8,131	8,948	9,697	
2019 Enf. Contacts YTD	1,121	2,480	3,634	4,833	5,824	6,593	7,404	8,354	9,227	10,258	10,992	11,914	

Bay Area Rapid Transit Police Department
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March 2022 *Performance Measurement Review -* ***Systemwide***

BART PD Warrant Arrests													
Warrant Arrests	**Jan**	**Feb**	**Mar**	**Apr**	**May**	**Jun**	**Jul**	**Aug**	**Sep**	**Oct**	**Nov**	**Dec**	**Total**
2022 BART Felony	2	1	3										6
2022 BART Misdemeanor	3	4	10										17
2022 Outside Felony	51	48	53										152
2022 Outside Misdemeanor	61	83	93										237
2022 Monthly Total	117	136	159										412
2022 YTD Total	117	253	412										
2021 BART Felony	2	2	0	2	4	4	8	2	6	0	2	4	36
2021 BART Misdemeanor	7	9	7	4	6	7	9	4	15	6	9	9	92
2021 Outside Felony	38	37	35	56	39	32	42	39	71	48	40	49	526
2021 Outside Misdemeanor	56	45	36	46	65	70	76	61	121	108	82	103	869
2021 Monthly Total	103	93	78	108	114	113	135	106	213	162	133	165	1,523
2021 YTD Total	103	196	274	382	496	609	744	850	1,063	1,225	1,358	1,523	
2020 BART Felony	0	2	0	0	0	2	4	4	0	6	2	2	22
2020 BART Misdemeanor	4	3	1	0	1	2	0	4	2	2	2	1	22
2020 Outside Felony	21	27	18	7	21	15	33	29	33	33	38	35	310
2020 Outside Misdemeanor	75	53	37	8	30	28	20	32	42	33	34	33	425
2020 Monthly Total	100	85	56	15	52	47	57	69	77	74	76	71	779
2020 YTD Total	100	185	241	256	308	355	412	481	558	632	708	779	
2019 BART Felony	4	2	2	4	0	2	0	0	0	6	0	0	20
2019 BART Misdemeanor	2	12	3	5	0	3	7	10	2	1	3	7	55
2019 Outside Felony	21	37	27	19	16	14	29	15	18	25	25	20	266
2019 Outside Misdemeanor	72	75	87	76	61	43	60	56	69	71	86	65	821
2019 Monthly Total	99	126	119	104	77	62	96	81	89	103	114	92	1,162
2019 YTD Total	99	225	344	448	525	587	683	764	853	956	1,070	1,162	

Bay Area Rapid Transit Police Department

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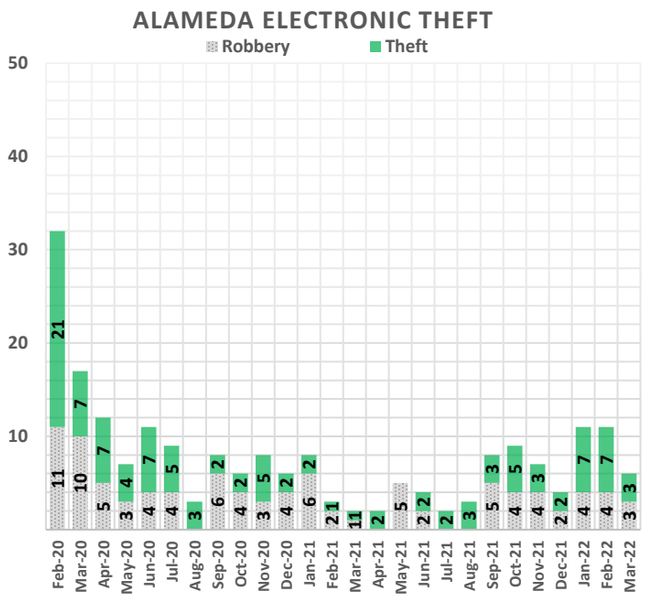
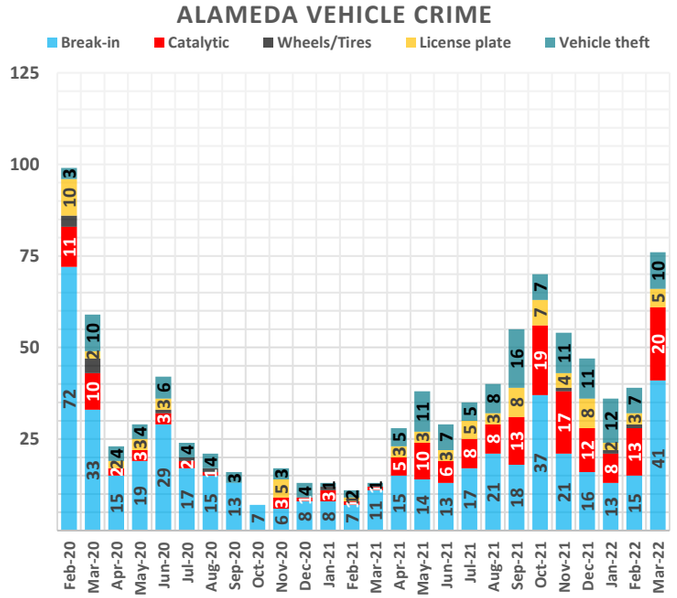
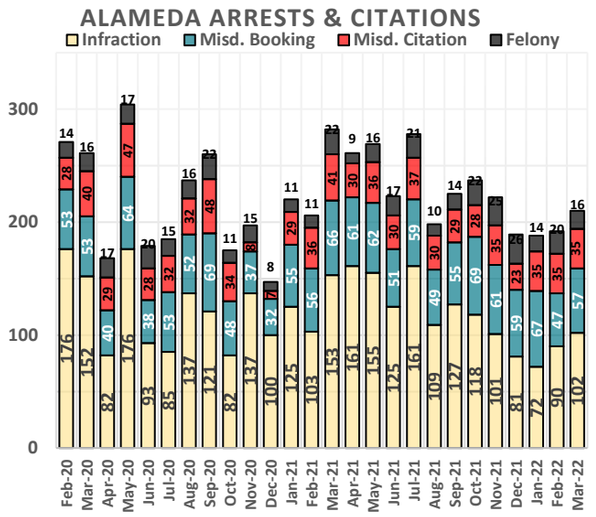
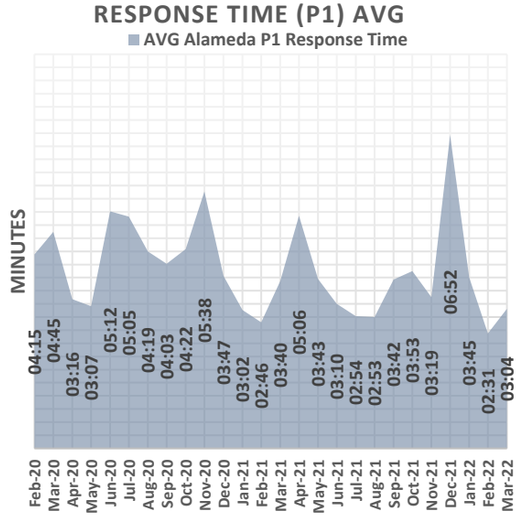
March 2022 *Performance Measurement Review - Alameda County*

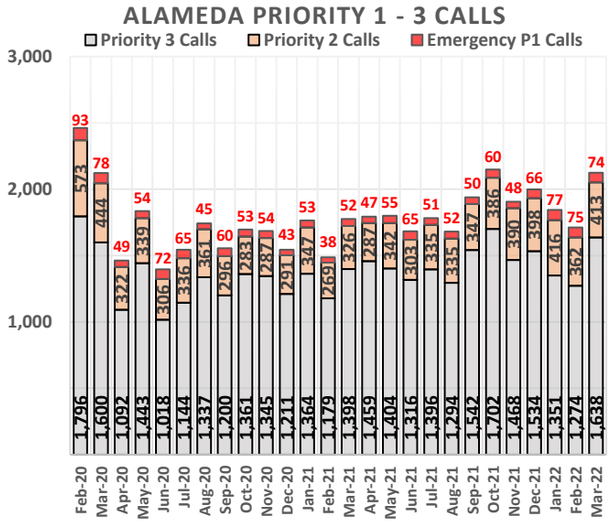
PART 1 UCR Crime	2018	2019	2020	2021	YTD 2021	YTD 2022	PCT %
Homicide	2	1	0	0	0	1	-%
Rape	3	2	3	2	0	0	-%
Robbery	211	229	122	64	16	25	+56%
Aggravated Assault	87	52	54	34	4	8	+100%
Violent Crime Subtotal	*303*	*284*	*179*	*100*	*20*	*34*	*+70%*
Burglary (Structural)	11	13	9	6	0	1	-%
Larceny & Auto Burglary	1,262	1,634	577	472	65	128	+97%
Auto Theft	201	149	56	85	4	29	+625%
Arson	3	5	2	2	0	1	-%
Property Crime Subtotal	*1,477*	*1,801*	*644*	*565*	*69*	*159*	*+130%*
TOTAL	*1,780*	*2,085*	*823*	*665*	*89*	*193*	*+117%*

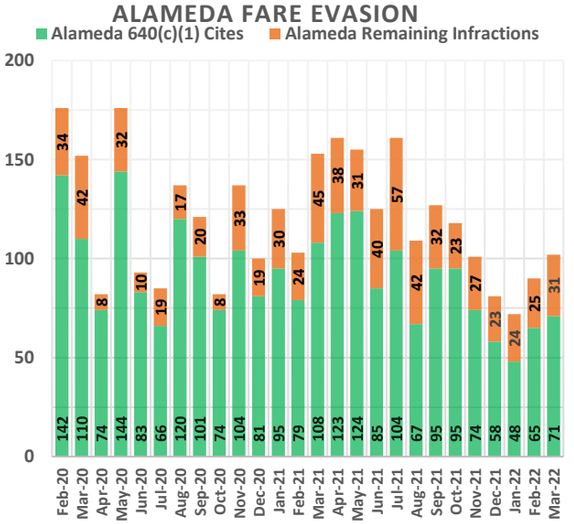












Preface: *The data is retrieved from the BART Police Database and remains unaudited until corrections. Numbers may differ from the reported data in the Uniform Crime Reporting (UCR) program.*

Late reporting, the reclassification or unfounding of crimes, may affect statistics. Infraction citations consist of all individual charges. Felony arrests, misdemeanor arrests and citations are based on each instance per individual

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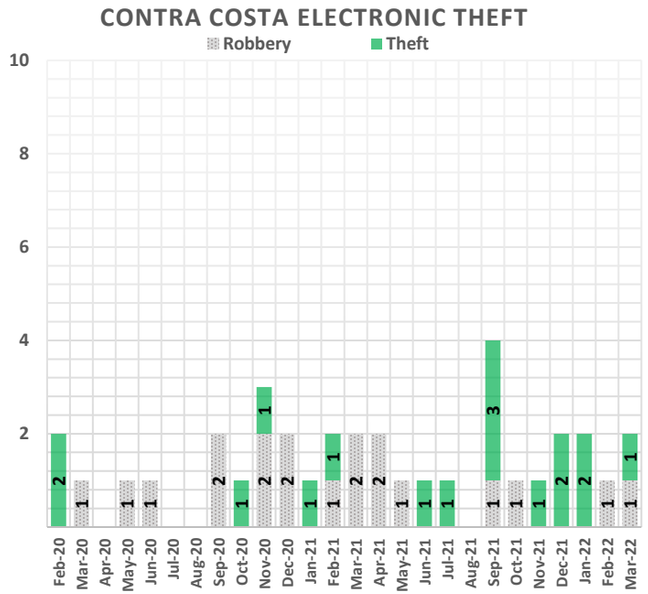
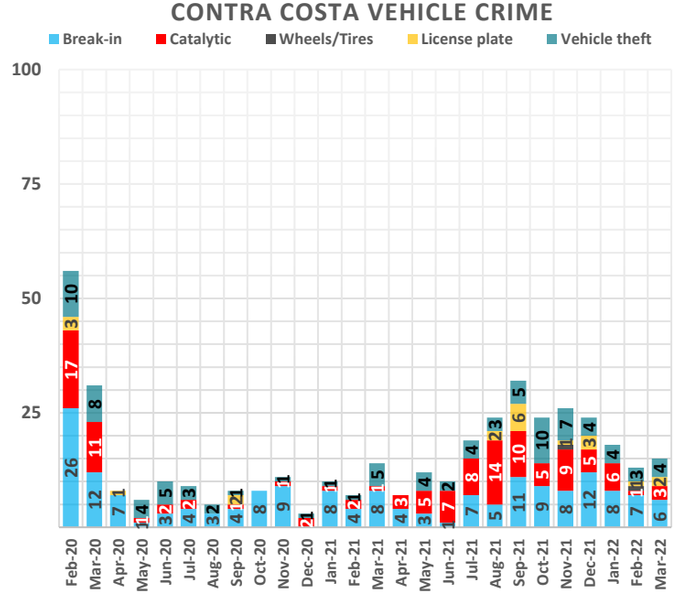
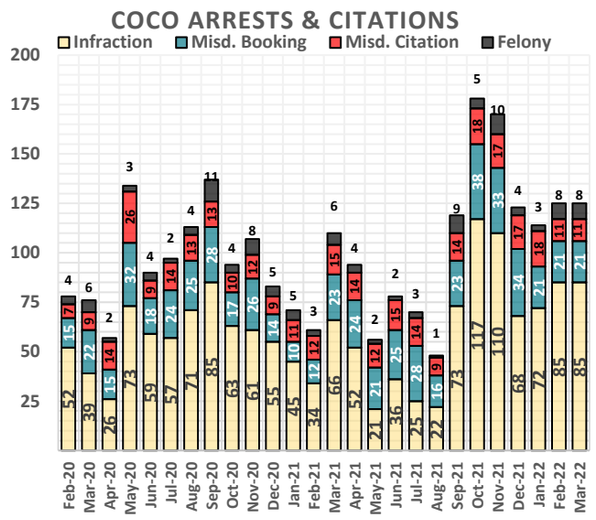
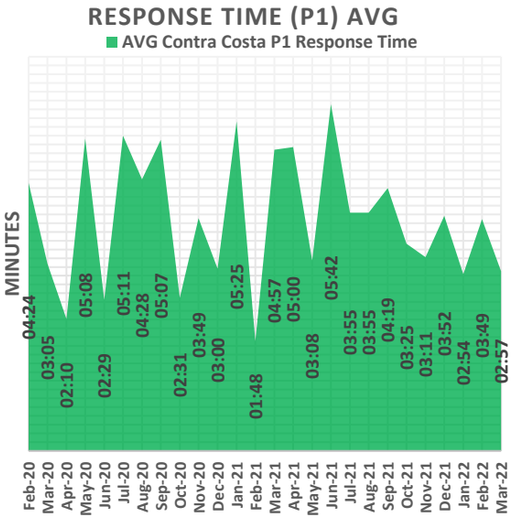
March 2022 Performance Measurement Review - **Contra Costa County**

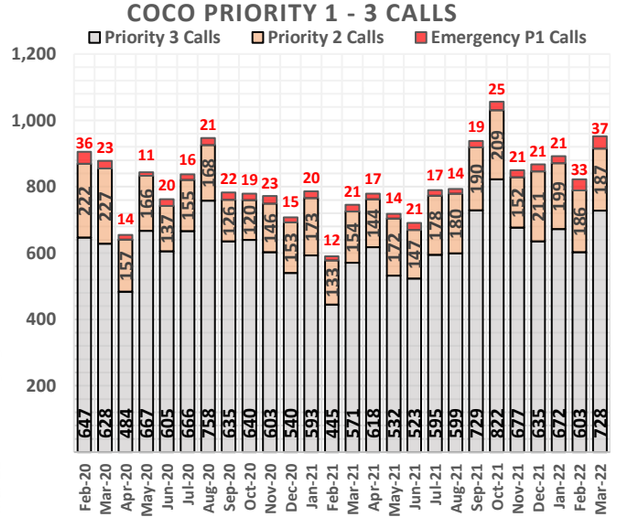
PART 1 UCR Crime	2018	2019	2020	2021	YTD 2021	YTD 2022	PCT %
Homicide	1	0	0	0	0	0	-%
Rape	0	4	0	4	0	2	-%
Robbery	29	34	23	19	7	4	-43%
Aggravated Assault	20	23	17	19	5	3	-40%
Violent Crime Subtotal	50	61	40	42	12	9	-25%
Burglary (Structural)	1	2	1	1	0	1	-%
Larceny & Auto Burglary	669	592	202	226	40	54	+35%
Auto Theft	124	81	40	46	7	11	+57%
Arson	1	0	0	0	0	2	-%
Property Crime Subtotal	795	675	243	273	47	68	+45%
TOTAL	845	736	283	315	59	77	+31%

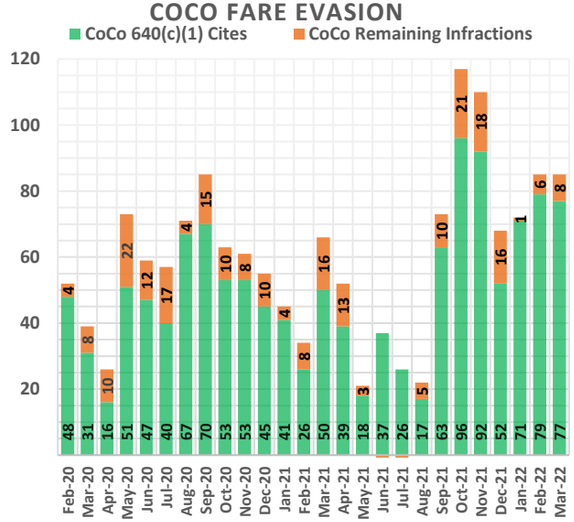












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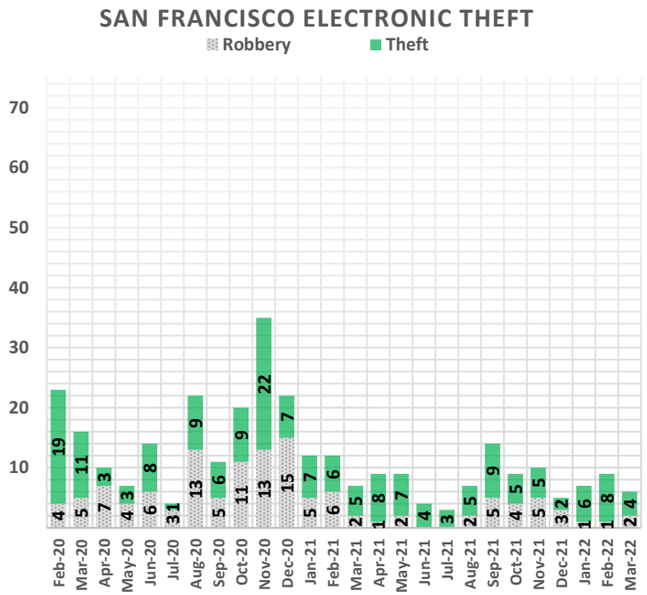
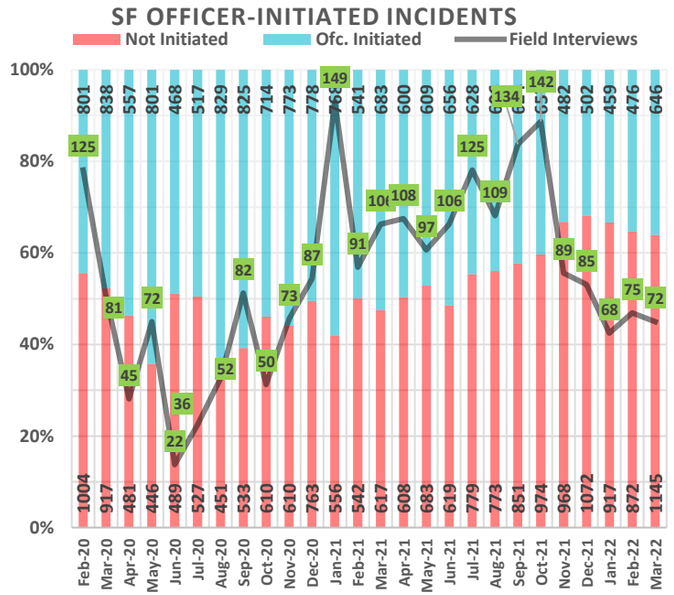
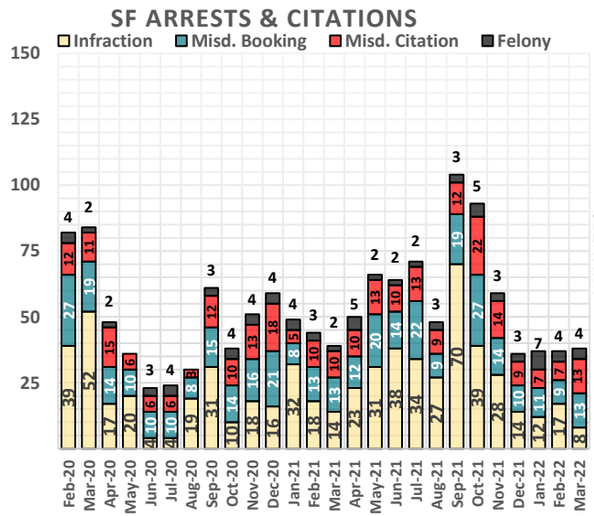
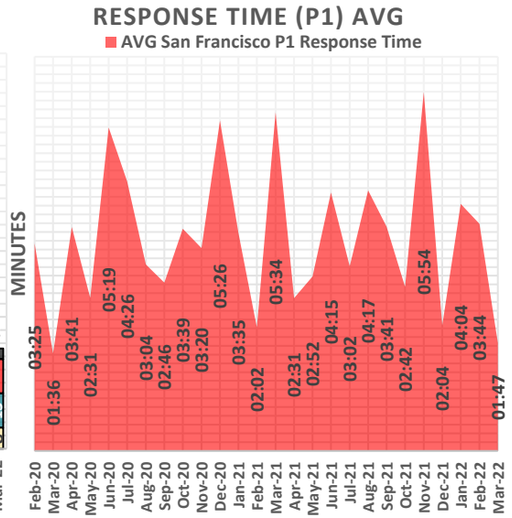
March 2022 *Performance Measurement Review - San Francisco County*

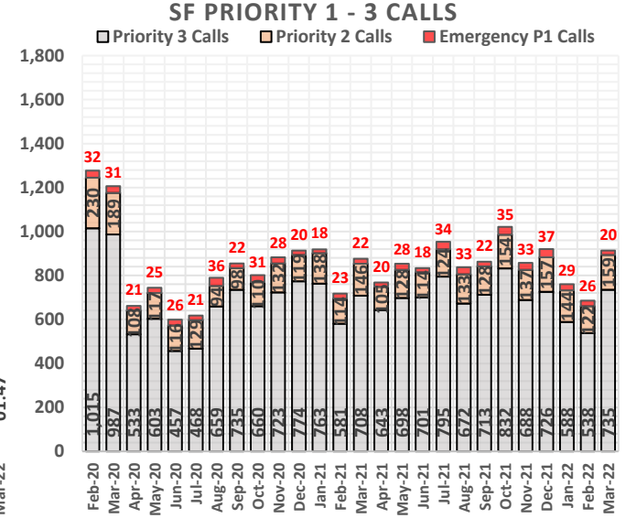
PART 1 UCR Crime	2018	2019	2020	2021	YTD 2021	YTD 2022	PCT %
Homicide	0	0	0	0	0	1	-%
Rape	0	0	4	1	0	0	-%
Robbery	97	104	101	47	16	6	-63%
Aggravated Assault	18	28	19	16	2	3	+50%
Violent Crime Subtotal	*115*	*132*	*124*	*64*	*18*	*10*	*-44%*
Burglary (Structural)	6	4	1	3	0	1	-%
Larceny & Auto Burglary	473	619	200	103	27	21	-22%
Auto Theft	1	1	0	0	0	0	-%
Arson	0	0	1	2	0	0	-%
Property Crime Subtotal	*480*	*624*	*202*	*108*	*27*	*22*	*-19%*
TOTAL	*595*	*756*	*326*	*172*	*45*	*32*	*-29%*

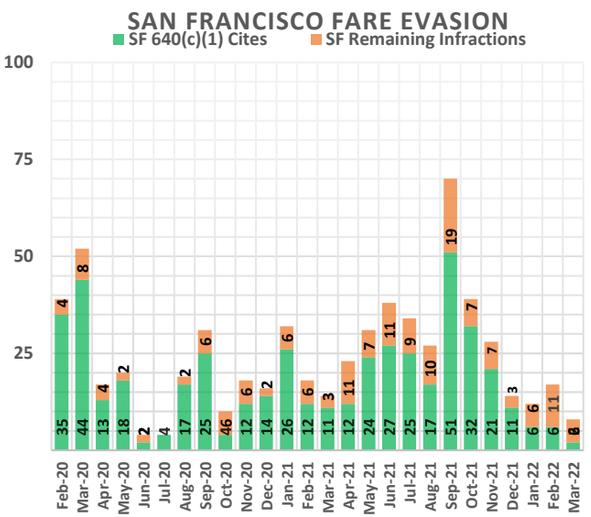












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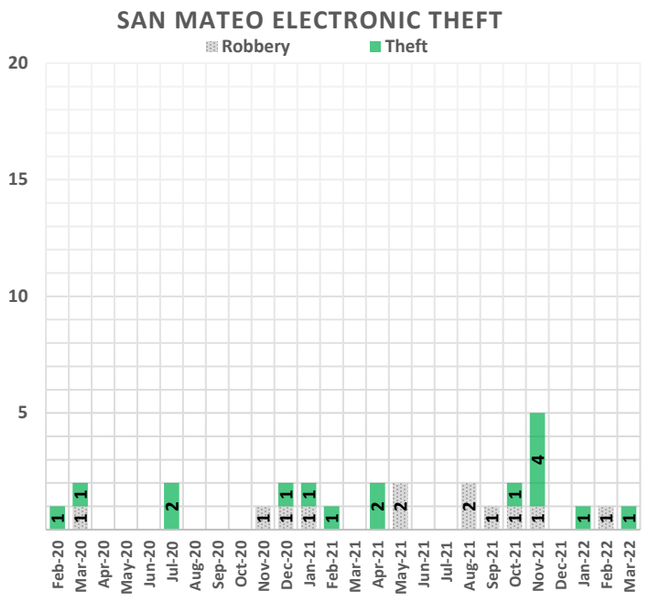
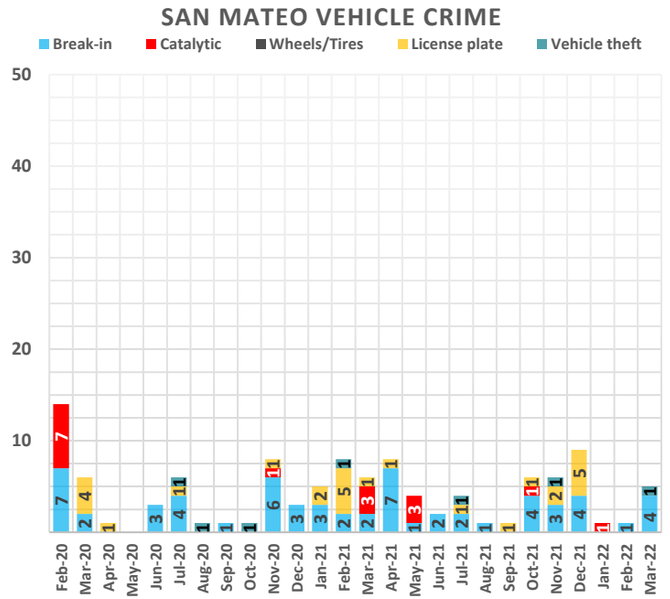
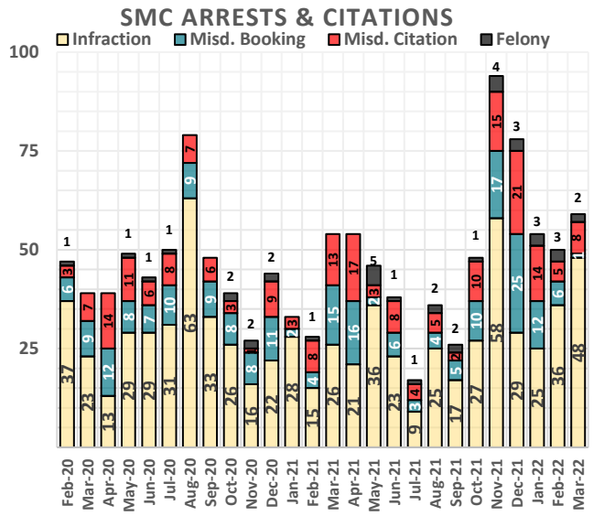
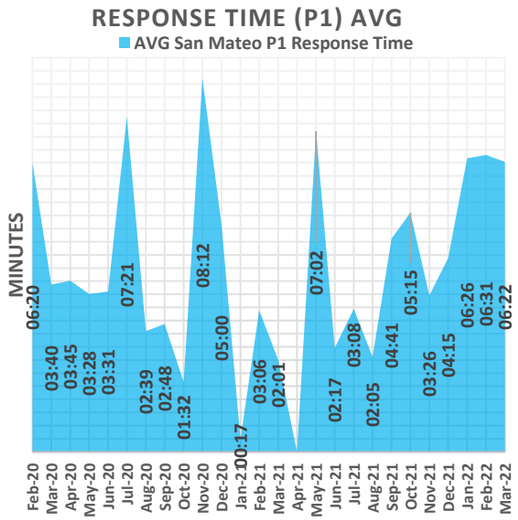
March 2022 *Performance Measurement Review - San Mateo County*

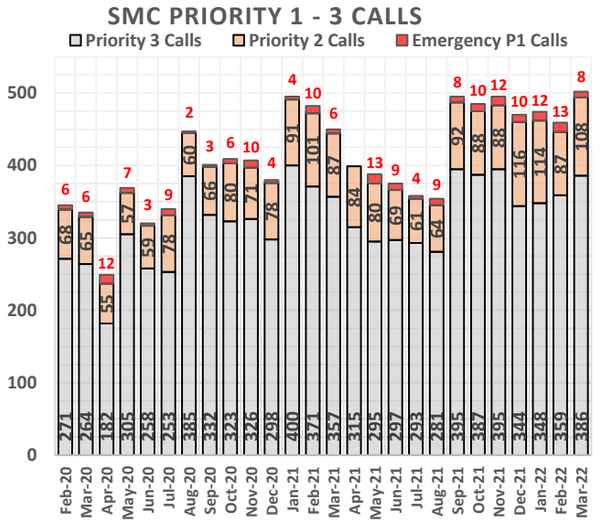
PART 1 UCR Crime	2018	2019	2020	2021	YTD 2020	YTD 2021	PCT %
Homicide	0	0	0	0	0	0	-%
Rape	0	1	1	1	0	0	-%
Robbery	8	13	6	10	2	4	+100%
Aggravated Assault	5	8	4	2	2	2	0%
Violent Crime Subtotal	13	22	11	13	4	6	+50%
Burglary (Structural)	0	0	1	1	0	0	-%
Larceny & Auto Burglary	161	332	75	81	24	7	-71%
Auto Theft	19	13	4	3	1	1	0%
Arson	0	0	1	1	0	0	-%
Property Crime Subtotal	180	345	81	86	25	8	-68%
TOTAL	193	367	92	99	29	14	-52%

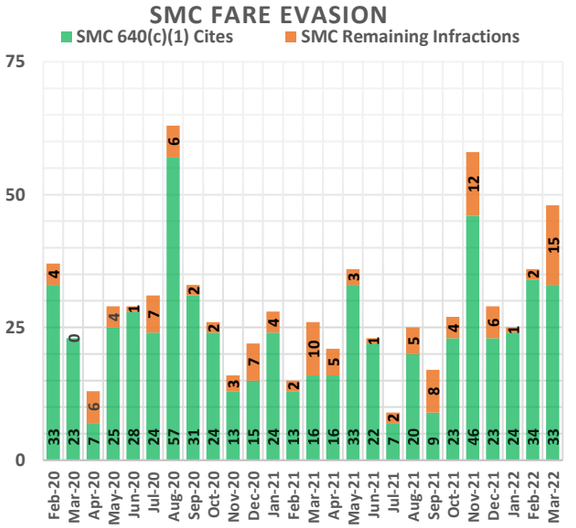












Preface: *The data is retrieved from the BART Police Database and remains unaudited until corrections. Numbers may differ from the reported data in the Uniform Crime Reporting (UCR) program.*

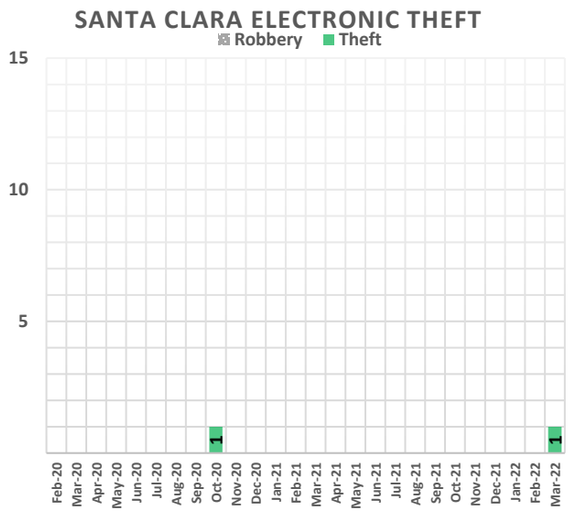
Late reporting, the reclassification or unfounding of crimes, may affect statistics. Infraction citations consist of all individual charges. Felony arrests, misdemeanor arrests and citations are based on each instance per individual.

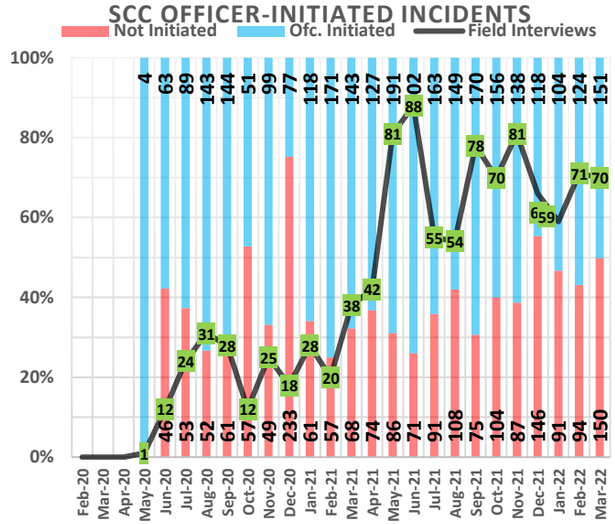
Bay Area Rapid Transit Police Department

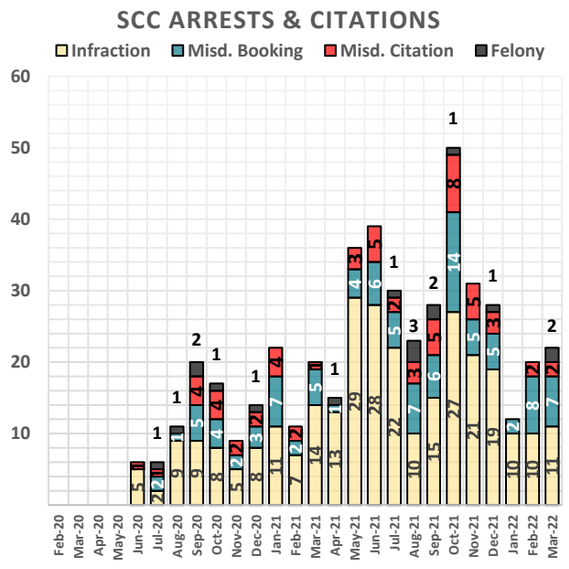
101 8th St, Oakland, CA, 94607 (510) 464-7000 www.bart.gov/police

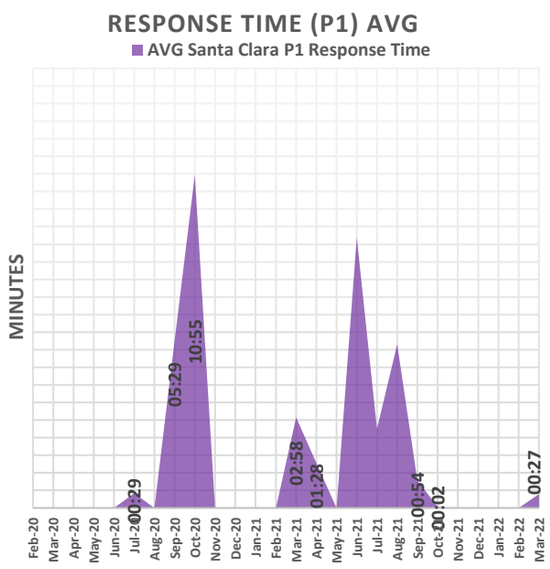
March 2022 *Performance Measurement Review - Santa Clara County*

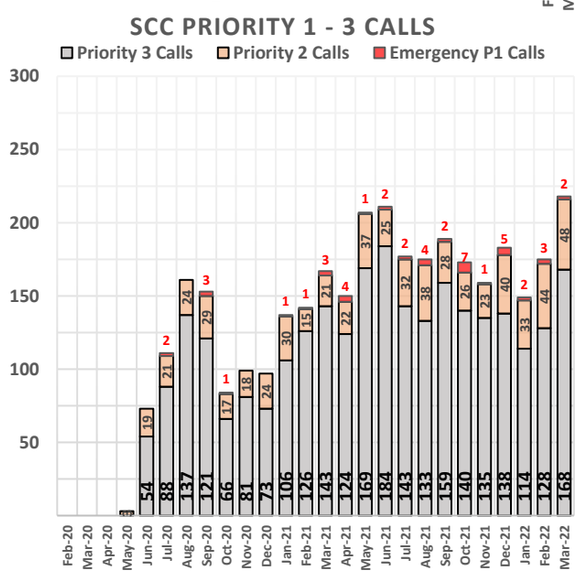
PART 1 UCR Crime	2018	2019	2020	2020	YTD 2020	YTD 2021	PCT %
Homicide	-	-	-	-	0	0	-%
Rape	-	-	-	-	0	0	-%
Robbery	-	-	-	2	0	0	-%
Aggravated Assault	-	-	1	0	0	1	-%
Violent Crime Subtotal	0	0	1	2	0	1	-%
Burglary (Structural)	-	-	-	-	0	0	-%
Larceny & Auto Burglary	-	-	1	-	0	0	-%
Auto Theft	-	-	-	-	0	0	-%
Arson	-	-	-	-	0	0	-%
Property Crime Subtotal	0	0	1	0	0	0	-%
TOTAL	0	0	2	2	0	1	-%

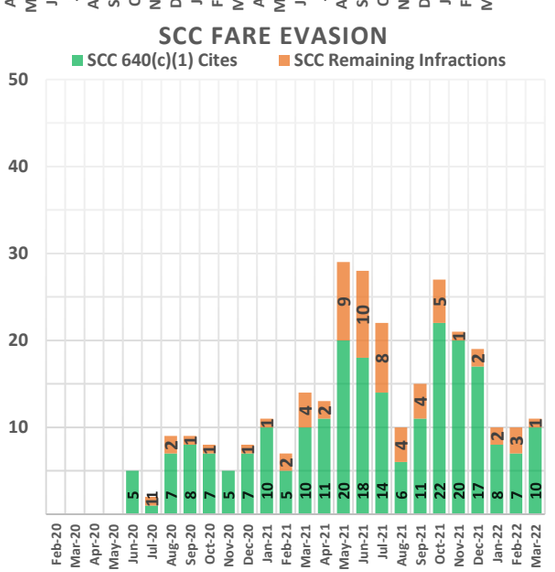












Preface: *The data is retrieved from the BART Police Database and remains unaudited until corrections. Numbers may differ from the reported data in the Uniform Crime Reporting (UCR) program. Late reporting, the reclassification or unfounding of crimes, may affect statistics. Infraction citations consist of all individual charges. Felony arrests, misdemeanor arrests and citations are based on each instance per individual.*

Internal Affairs Division Acronyms

Admin: Administrative

OIPA Investigation: Office of the Independent Police Auditor is conducting the investigation

S.R.: Supervisor Referral

Tolled: Internal Affairs Investigation is on hold (paused)

Allegation Types

Arrest or Detention

BBP: Bias Based Policing

CUBO: Conduct Unbecoming an Officer

Courtesy

Force

POD: Performance of Duty

Policy Complaint

Policy/Procedure

Racial Animus

Reporting Misconduct

Search or Seizure

Service Review

Supervision

Truthfulness

Workplace Discrimination/Harassment

BART Police Department - Office of Internal Affairs

Investigation Log

IA#:	DATE OCC'D	DATE REC'D	ALLEGATION	MISC	INVESTIGATOR	STATUS	5 Month Date	Due Date
IA2018-001	1/3/2018	1/3/2018	Force (OIS)		Lt. Salas	Tolled	6/4/2018	
IA2018-060	7/22/2018	7/23/2018	Service Review		Lt. Salas	Tolled	12/23/2018	
IA2020-017	2/15/2020	2/15/2020	Force		Sgt. Spears	Tolled	7/16/2020	2/15/2021
IA2020-057	8/12/2020	8/27/2020	CUBO	Retained By OIPA #20-22	OIPA	OIPA Investigation	1/26/2021	8/27/2021
IA2021-008	1/8/2021	1/11/2021	Unk	Admin Closure	Sgt. Turner	Tolled	6/12/2021	1/11/2022
IA2021-032	4/19/2021	4/19/2021	POD		Lt. Salas	In Progress	9/18/2021	4/19/2022
IA2021-033					Lt. Salas	In Progress	9/20/2021	12/31/1900
IA2021-036	6/4/2021	6/4/2021	POD		Sgt. Turner	In Progress	11/3/2021	6/4/2022
IA2021-037	1/24/2021	5/11/2021	POD	Admin Closure	Lt. Salas	In Progress	10/10/2021	5/11/2022
IA2021-040	6/11/2021	6/11/2021	Force, CUBO		Sgt. Turner	In Progress	11/10/2021	6/11/2022
IA2021-042	5/23/2021	5/23/2021	BBP, CUBO		Sgt. Spears	In Progress	10/22/2021	5/23/2022
IA2021-043	5/23/2021	5/26/2021	Force, Arrest/Detention	Retained by OIPA #21-10	OIPA	OIPA Investigation	10/25/2021	5/26/2022
IA2021-044	5/28/2021	5/28/2021	Bias Based Policing		Sgt. Spears	In Progress	10/27/2021	5/28/2022

IA2021-045	6/12/2021	6/12/2021	CUBO		Sgt. Turner	In Progress	11/11/2021	6/12/2022
IA2021-046	6/19/2021	6/22/2021	Force, BBP	Retained by OIPA #21-15	OIPA	OIPA Investigation	11/21/2021	6/22/2022
IA2021-047	6/16/2021	6/22/2021	CUBO, Policy/Procedure		Sgt. Spears	In Progress	11/21/2021	6/22/2022
IA2021-048	2/1/2021	7/1/2021	Arrest/Detention Force/POD(Sup)		Sgt. Turner	In Progress	11/30/2021	7/1/2022
IA2021-049	5/19/2021	7/5/2021	Force		Sgt. Spears	In Progress	12/4/2021	7/5/2022
IA2021-050	7/4/2021	7/5/2021	Policy/Procedure, BBP, Arrest/Detention		Sgt. Spears	In Progress	12/4/2021	7/5/2022
IA2021-053	7/16/2021	7/16/2021	Force		Sgt. Turner	In Progress	12/15/2021	7/16/2022
IA2021-054	7/19/2021	7/19/2021	POD		Sgt. Turner	In Progress	12/18/2021	7/19/2022
IA2021-055	7/21/2021	7/21/2021	CUBO		Sgt. Turner	In Progress	12/20/2021	7/21/2022
IA2021-056	7/18/2021	7/27/2021	Force/ Arrest & Detention		Sgt. Spears	In Progress	12/26/2021	7/27/2022
IA2021-057	7/28/2021	7/28/2021	Bias Based Policing		Sgt. Spears	In Progress	12/27/2021	7/28/2022
IA2021-058	7/16/2021	8/2/2021	Force, BBP, POD, CUBO		Sgt. Turner	Tolled	1/1/2022	8/2/2022
IA2021-059	8/16/2021	8/16/2021	Force		Sgt. Turner	In Progress	1/15/2022	8/16/2022
IA2021-060	8/14/2021	8/14/2021	Force, CUBO		Sgt. Turner	In Progress	1/13/2022	8/14/2022

IA2021-061	8/19/2021	8/19/2021	CUBO		Sgt. Spears	In Progress	1/18/2022	8/19/2022
IA2021-062	4/6/2021	9/2/2021	Force, POD		Sgt. Turner	In Progress	2/1/2022	9/2/2022
IA2021-063	9/16/2021	9/16/2021	Force		Sgt. Turner	In Progress	2/15/2022	9/16/2022
IA2021-064	9/16/2021	9/20/2021	Force		Sgt. Turner	In Progress	2/19/2022	9/20/2022
IA2021-065	9/15/2021	9/20/2021	Force		Sgt. Spears	In Progress	2/19/2022	9/20/2022
IA2021-066	9/22/2021	9/22/2021	Bias-Based Policing	clear by video request sent to oipa on 11/1/21	Sgt. Spears	Pending Approval	2/21/2022	9/22/2022
IA2021-067	9/22/2021	9/22/2021	Bias-Based Policing		Sgt. Turner	In Progress	2/21/2022	9/22/2022
IA2021-068	9/12/2021	9/14/2021	CUBO		Sgt. Spears	In Progress	3/1/2022	9/14/2022
IA2021-069	9/23/2021	9/27/2021	Bias-Based Policing		Sgt. Spears	In Progress	3/1/2022	9/27/2022
IA2021-070	9/24/2021	9/24/2021	Force		Sgt. Turner	In Progress	2/23/2021	9/24/2022
IA2021-072	10/3/2021	10/4/2021	CUBO	SR	Sgt. Spears	In Progress	3/5/2022	10/4/2022
IA2021-073	3/29/2021	10/4/2021	Force		Sgt. Spears	In Progress	3/5/2022	10/4/2022
IA2021-074	9/25/2021	10/1/2021	Force, Arrest/Detention, Policy/Procedure	Retained by OIPA #21-19	OIPA	OIPA Investigation	3/2/2022	10/1/2022

IA2021-076	10/13/2021	10/13/2021	Force		Sgt. Turner	In Progress	3/14/2022	10/13/2022
IA2021-077	10/8/2021	10/12/2021	Bias Based Policing		Sgt. Turner	In Progress	3/13/2021	10/12/2022
IA2021-078	10/15/2021	10/15/2021	BBP, CUBO		Sgt. Turner	In Progress	3/16/2021	10/15/2022
IA2021-079	10/11/2021	10/13/2021	Arrest/Detention		Sgt. Spears	In Progress	3/22/2022	10/13/2022
IA2021-080	10/14/2021	10/20/2021	CUBO		Sgt. Spears	In Progress	3/22/2022	10/20/2022
IA2021-081	12/31/2019	10/13/2021	Force		Sgt. Turner	Tolled	3/14/2022	10/13/2022
IA2021-083	10/15/2021	10/15/2021	Force		Sgt. Spears	In Progress	3/16/2021	10/15/2022
IA2021-084	10/21/2021	10/21/2021	POD		Sgt. Turner	In Progress	3/22/2022	10/21/2022
IA2021-085	10/26/2021	10/26/2021	Force, BBP Arrest/Detention		Sgt. Spears	In Progress	3/27/2022	10/26/2022
IA2021-086	10/27/2021	11/1/2021	Force	Retained by OIPA #21-24	OIPA	OIPA Investigation	4/2/2022	11/1/2022
IA2021-087		11/1/2021	POD, Truthfulness, Policy/Procedure, CUBO		Sgt. Turner	In Progress	4/2/2022	11/1/2022
IA2021-088	10/31/2021	10/31/2021	Force		Sgt. Spears	In Progress	4/1/2022	10/31/2022
IA2021-089	11/1/2021	11/1/2021	Force		Sgt. Spears	In Progress	4/2/2022	11/1/2022
IA2021-090	11/4/2021	11/8/2021	Force		Sgt. Spears	In Progress	4/9/2022	11/8/2022

IA2021-091	11/10/2021	11/12/2021	POD	OIPA Intake #21-24, Possible Admin Closure	Sgt. Turner	In Progress	4/13/2022	11/12/2022
IA2021-092	11/12/2021	11/16/2021	Force, Arrest/Detention		Sgt. Spears	In Progress	4/17/2022	11/16/2022
IA2021-093	11/2/2021	11/10/2021	Arrest/Detention		Sgt. Spears	In Progress	4/30/2022	11/10/2022
IA2021-095	6/29/2016	10/25/2021	POD	Possible Admin Clourse	Sgt. Spears	In Progress	3/26/2022	10/25/2022
IA2021-096	12/5/2021	12/8/2021	Force		Sgt. Turner	In Progress	5/9/2022	12/8/2022
IA2021-097	12/3/2021	12/17/2021	POD, CUBO Policy/Procedure (Report Writing), Axon Camera		Sgt. Spears	In Progress	5/23/2022	12/17/2022
IA2021-099	11/24/2021	11/26/2021	CUBO		Sgt. Spears	In Progress	5/28/2022	11/26/2022
IA2021-102	10/2/2021	10/15/2021	POD, Policy/Procedure (Report Writing)	OIPA Intake #21-21	Sgt. Spears	In Progress	3/16/2022	10/15/2022
IA2022-001	1/13/2022	1/18/2022	Force		Sgt. Turner	In Progress	6/19/2022	1/18/2023
IA2022-002	1/1/2022	1/4/2022	POD	OIPA Intake #22-01	OIPA	In Progress	6/5/2022	1/4/2023
IA2022-003	1/24/2022	1/26/2022	CUBO		Sgt. Spears	In Progress	6/28/2022	1/26/2023
IA2022-004	2/3/2022	2/4/2022	Force, CUBO, POD		Sgt. Turner	In Progress	7/6/2022	2/4/2023

IA2022-005	2/8/2022	2/8/2022	Search/Seizure, CUBO, Axon		Sgt. Spears	In Progress	7/10/2022	2/8/2023
IA2022-006	2/8/2022	2/8/2022	POD, CUBO, Axon		Sgt. Spears	In Progress	7/10/2022	2/8/2023
IA2022-008	12/4/2020	2/10/2022	Arrest/Detention, Policy/Procedure	Retained by OIPA #22-04	OIPA	OIPA Investigation	7/12/2022	2/10/2023
IA2022-009	2/8/2022	2/9/2022	Arrest/Detention, Force, Criminal (Sexual Assault)	OIPA deferred to IA #22-03	Sgt. Turner	In Progress	7/11/2022	2/9/2023

BART Watch - 2022

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	YTD
Crime in Progress	23	15	31										69
Disruptive Behavior	277	253	332										862
Drug Use	150	121	202										473
Human Trafficking	2	2	0										4
Illegally Parked Vehicle	2	2	2										6
Aggressive Panhandling	6	5	10										21
Report a Crime Tip	7	9	8										24
Robbery/Theft	5	6	7										18
Sexual Assault/Lewd Behavior	6	2	8										16
Suspicious Activity	45	29	51										125
Unattended Bag or Package	6	10	17										33
Unsecure Door	8	5	4										17
Vandalism	20	32	41										93
Welfare Check	64	62	90										216
Unwanted Sex Harassment	5	3	3										11
Total	**626**	**556**	**806**										**1,988**
Text-a-Tip	1,557	1,511	1,970										5,038

Total Downloads: **98,342**

Office of the
INDEPENDENT
POLICE AUDITOR

BAY AREA RAPID TRANSIT DISTRICT

MONTHLY REPORT

April 2022

Issue date: May 9, 2022

This report is filed pursuant to the BART Citizen Oversight Model, Chapter 1-05 (B), which requires the Office of the Independent Police Auditor (OIPA) to submit reports to the BART Police Citizen Review Board (BPCRB). This report provides information for the period **April 1, 2022 through April 30, 2022.**[1] ***(The Quantitative Report includes all complaints received and administrative investigations initiated by both OIPA and the BART Police Department (BPD) Internal Affairs Bureau (IAB)).***

QUANTITATIVE REPORT

	Cases Filed[2]	Open Cases[3]	Investigations Resolved	OIPA Investigations Concluded[4]	Cases Appealed to OIPA[5]	Cases Appealed by BPCRB[6]
April 2021	13	65	9	1	0	0
May 2021	9	69	4	1	0	0
June 2021	5	74	1	1	0	0
July 2021	10	81	3	0	0	0
August 2021	4	78	7	1	0	0
September 2021	10	81	8	2	0	0
October 2021	15	88	7	0	0	0
November 2021	8	87	11	1	0	0
December 2021	6	87	6	0	1	0
January 2022	4	84	7	1	0	0
February 2022	6	81	9	1	0	0
March 2022	6	73*	14	1	0	0
April 2022	10	79	6	1	0	0

TYPES OF CASES FILED

Citizen Complaints (Formal)	10
Informal Complaints[7]	0
Administrative Investigations	0
Inquiries[8]	0
TOTAL	**10**

CITIZEN COMPLAINTS RECEIVED PER DEPARTMENT[9]

OIPA	5
BART Police Department	5
TOTAL	**10**

* This total was adjusted to reflect the closure of IA2021-071/OIPA #21-18, which was completed in March 2022 but not listed by BPD as completed until April 2022.

COMPLAINTS/INVESTIGATIONS INITIATED DURING REPORTING PERIOD

During April 2022, **5 Citizen Complaints** were received by OIPA:

Complaint # (OIPA Case #) (IA Case #)	Nature of Complaint	Action Taken	Days Elapsed Since Complaint Filed
1 (OIPA #22-12) (IA2022-016)	Unknown BPD Officers #1-2: • Performance of Duty • Conduct Unbecoming an Officer	OIPA notified BPD which initiated an investigation.	32
2 (OIPA #22-15) (IA2022-018)	Officers #1-3: • Arrest/Detention • Force • Policy/Procedure • Conduct Unbecoming an Officer	OIPA notified BPD which initiated an investigation.	29
3 (OIPA #22-14) (IA2022-019)	Unknown BPD Officers #1-2: • Bias-Based Policing	OIPA notified BPD which initiated an investigation.	28
4 (OIPA #22-16) (IA2022-020)	Officers #1-2: • Bias-Based Policing • Policy/Procedure	OIPA notified BPD which initiated an investigation.	27
5 (OIPA #22-17) (IA2022-021)	Officer #1: • Bias-Based Policing • Racial Animus • Conduct Unbecoming an Officer	OIPA notified BPD which initiated an investigation.	25

During April 2022, **4 Citizen Complaints (Formal)** were received by BPD:

Complaint # IA Case #	Nature of Complaint	Action Taken	Days Elapsed Since Complaint Filed
1 (IA2022-017)	Officer #1: • Performance of Duty	BPD initiated an investigation.	27
2 (IA2022-022)	Officer #1: • Bias-Based Policing • Conduct Unbecoming an Officer • Policy/Procedure (AXON Camera)	BPD initiated an investigation.	22
3 (IA2022-024)	Unknown Officers #1-2: • Conduct Unbecoming an Officer	BPD initiated an investigation.	17
4 (IA2022-025)	Officers #1-2: • Bias-Based Policing • Performance of Duty	BPD initiated an investigation.	14

COMPLAINTS/INVESTIGATIONS RECEIVED DURING PRIOR REPORTING PERIOD

During March 2022, **1 Citizen Complaint (Formal)** was received by BPD but not previously reported:

Complaint # IA Case #	Nature of Complaint	Action Taken	Days Elapsed Since Complaint Filed
1 (IA2022-023)	Officer #1: • Force • Conduct Unbecoming an Officer	BPD initiated an investigation.	56

COMPLAINTS/INVESTIGATIONS CONCLUDED DURING REPORTING PERIOD

During April 2022, **1 Citizen Complaint** was concluded by OIPA:

Complaint # (IA Case #)	Nature of Complaint	Disposition	Days Elapsed Since Complaint Filed	Days Taken to Complete Investigation
1 (OIPA #21-10) (IA2021-043)	Officers improperly detained subjects and used excessive force and did so because of the subjects' race.	Officers #1-5: • Arrest/Detention – Exonerated • Force – Exonerated • Bias-Based Policing – Unfounded	347	313

During April 2022, **2 Citizen Complaints** were concluded by BPD:

Complaint # (IA Case #)	Nature of Complaint	Disposition	Days Elapsed Since Complaint Filed	Days Taken to Complete Investigation
1 (IA2021-008)	Officers did not take enforcement action against fare evaders and attempted intimidate complainant.	Officers #1-5: • Performance of Duty – Administratively Closed • Conduct Unbecoming an Officer – Administratively Closed	483	469[†]
2 (IA2021-042)	Officer harassed complainant during a law enforcement contact.	Officers #1-2: • Conduct Unbecoming an Officer – Unfounded	351	338

[†] BPD reported that this case was tolled due to a subject officer's unavailability from May 28, 2021 to April 1, 2022 (308 days).

During April 2022, **2 Administrative Investigations** were concluded by BPD:

Nature of Allegations	Disposition	Days Elapsed Since Investigation Initiated	Days Taken to Address Allegation(s)	
1 (IA2021-032)	Officer was impaired while on duty.	Officer #1: • Performance of Duty – Sustained • Policy/Procedure – Not Sustained	385	357
2 (IA2021-037)	Complainant stated that officer should be held accountable for misconduct during an arrest/detention and use of force. Supervisor did not forward potential misconduct complaint to Internal Affairs.	Officer #1: • Force – Administratively Closed Officer #2: • Supervision – Training Recommendation	363	349

DISCIPLINE ISSUED DURING REPORTING PERIOD

During April 2022, BPD took the following actions in cases where one or more allegations of misconduct were sustained:

Case #	Nature of Sustained Allegation(s) ‡	Classification of Sustained Allegation(s)	Action Taken
1	Officer used racist language in an email to BART staff.	Officer #1: • Racial Animus • Conduct Unbecoming an Officer	Officer #1: • No Action§
2	Officer was impaired while on duty.	Officer #1: • Performance of Duty – Sustained	Officer #1: • Written Reprimand[10]
3	Officer failed to properly document a law enforcement contact and did not properly review the use of force.	Officer #2: • Policy/Procedure (AXON Camera) – Sustained • Policy/Procedure (Use of Force Review) – Sustained	Officer #1: • Letter of Discussion[11]
4	Officer did not properly document a law enforcement contact.	Officer #1: • Policy/Procedure (AXON Camera)	Officer #1: • Letter of Discussion

‡Some details regarding the nature of sustained allegations may be withheld to avoid unintentionally breaching mandatory confidentiality requirements. In some instances, the relative infrequency of the alleged misconduct may tend to allow for identification of the subject officer in violation of the applicable CA Penal Code section (832.7).

§ Subject officer separated from the Department prior to the imposition of any discipline for the sustained allegations.

5	Officer did not properly document a law enforcement contact.	Officer #1: • Policy/Procedure (AXON Camera)	Officer #1: • Letter of Discussion
6	Officer did not properly document a law enforcement contact.	Officer #1: • Policy/Procedure (AXON Camera)	Officer #1: • Letter of Discussion

ADDITIONAL NOTES

In accordance with the BART Citizen Oversight Model (Model), OIPA investigates certain complaints, conducts complainant-initiated appeals, and monitors and/or reviews complaint investigations conducted by BPD. Though potentially work-intensive, some complaint investigation reviews are completed informally, with any concerns being addressed through a conference with BPD's Internal Affairs investigators. Noting the various kinds of work that OIPA undertakes with regard to complaints and investigations, the following chart includes some of the pending cases in which OIPA is involved as of the end of this reporting period.

Investigations Being Conducted	8
Complainant-Initiated Appeals	1
BPD-Initiated Appeals	0
Investigations Being Monitored	71
Investigations Reviewed During Current Month	10†

†This number does not include all OIPA reviews, as OIPA commonly looks at a variety of cases in the Internal Affairs database to obtain updates on both pending and completed investigations.

The Model provides that OIPA shall have authority to require follow-up investigation into any citizen complaint or allegation that is handled by BPD. The OIPA Monthly Report will reflect information regarding monitored cases with detail not to exceed that which is allowable under state law. The investigations reviewed by OIPA during the period did not generate any notable recommendations for revisions or additional investigation.[1][2]

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[1] In addition to reporting on complaints received by the BART Police Department, the Citizen Oversight Model requires reporting on all complaints received by the "Citizen Board, Office of the District Secretary, and other District departments." As complaints received by the BART Police Citizen Review Board are customarily directed to OIPA for further action, such complaints are included in the Quantitative Report above; OIPA is also made aware of additional complaints about the BART Police Department by the Office of the District Secretary or other District departments.

[2] This number includes all Citizen Complaints filed against members of the BART Police Department, as well as Administrative Investigations generated internally by BART Police Department members (as opposed to being filed by a citizen). This number also includes previously completed cases that have been re-opened during the current reporting period.

[3] This number indicates all investigations that are open as of the end of the reporting period. It includes Citizen Complaints (regardless of whether the investigation is being conducted by OIPA, the BART Police Department, or both) and Administrative Investigations.

[4] This number includes all cases completed by OIPA during the reporting period for which OIPA's findings are required by the BART Citizen Oversight Model to be submitted to the BART Police Citizen Review Board. It therefore includes independent investigations, as well as reviews of completed BART Police Department investigations initiated via appeal from a complainant. Unless otherwise noted, it does not include reviews of BART Police Department investigations initiated at the discretion of OIPA, which happen commonly and do not always generate a formal report; it also does not include reviews conducted by OIPA of complaint investigations where the complaint was filed with OIPA but did not fall under OIPA's investigative jurisdiction.

[5] This number refers to appeals filed with OIPA by complainants who have been issued the findings of the BART Police Department's internal investigation into their complaint regarding on-duty incidents. OIPA has a responsibility to review such appeals pursuant to the BART Citizen Oversight Model, Chapter 1-04 (E).

[6] This number refers to all appeals initiated by the BART Police Citizen Review Board after receiving and reviewing the findings issued by OIPA in a given case. The routes of all such appeals are described in detail in the BART Citizen Oversight Model, Chapter 1-04 (B) (iv-v).

[7] The BART Police Department defines an Informal Complaint as, "A comment on the actions of a Department employee, where the reporting party expressly states that he or she does not feel that the matter should be formally investigated with the understanding that an Informal Complaint does not hold the potential to result in disciplinary action against the employee." (BART Police Department Policy Manual, Policy 1020.1.1(d)).

[8] BPD policy provides that if a person alleges or raises an issue that does not constitute a violation of Department policy, procedure, rules, regulations, or the law, the Department will classify the issue as an inquiry.

[9] It is important to note that OIPA does not separate citizen complaints it receives into "Formal" and "Informal" classifications. This chart reflects all citizen complaints received by OIPA and all Formal Complaints received by the BART Police Department.

[10] Written Reprimand (first level of formal discipline): If there have been no re-occurrences at the end of the time frames as determined by the collective bargaining agreement (up to 3 years), the immediate supervisor shall meet with the employee and advise him/her that the progressive discipline has become inactive and has been removed from the employee's personnel files.

[11] Letter of Discussion (second level of pre-discipline): A letter of discussion may be the next step of the process of the informal process. It is a written memorandum to the employee making the employee aware of the unacceptable behavior. A letter of discussion is pre-disciplinary, however, if the employee fails to correct the behavior, there will be cause to move to the next level of the process or to move to formal progressive discipline. An employee who may be issued a letter of discussion is entitled to appropriate representation. (BPD Policy Manual)

[12] OIPA may submit recommendations to IA regarding minor clerical or record-keeping adjustments which are intended to maintain the integrity of the data collection and record-keeping processes at BPD. These are not considered by OIPA to be substantive recommendations requiring reporting herein.

Use of the Term of "Excited Delirium" and its Relationship to Racial Equity

BART Citizen Police Review Board Meeting

May 9, 2022

Office of the
INDEPENDENT
POLICE AUDITOR

BAY AREA RAPID TRANSIT DISTRICT

Understanding Racially Disparate Outcomes

- 2020 Center for Policing Equity Report
  - Overall, 63% of persons who experienced force were Black (compared to their 8.7% share of the population served by BART).
  - Black persons were 13 times more likely to experience BART PD use of force than their white counterparts were.
- Recommendation 6 was that BPD work in collaboration with OIPA and the BPCRB to implement the recommendations made in the report.

Since then…

- G.A.R.E. Training for the District
- May 2020 George Floyd Protests
- Board of Directors participation in racial equity training
- August 2020 Progressive Policing and Community Engagement Bureau established

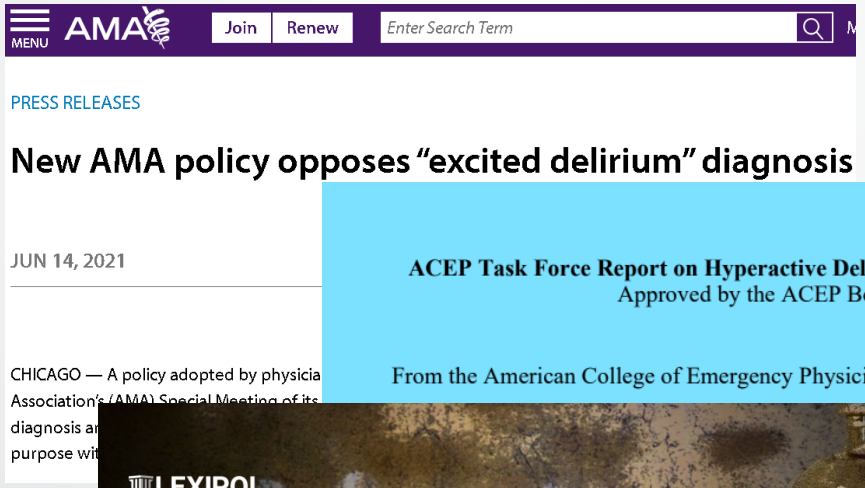
BART Affirms Commitment to Progressive Policing and Fighting Racism

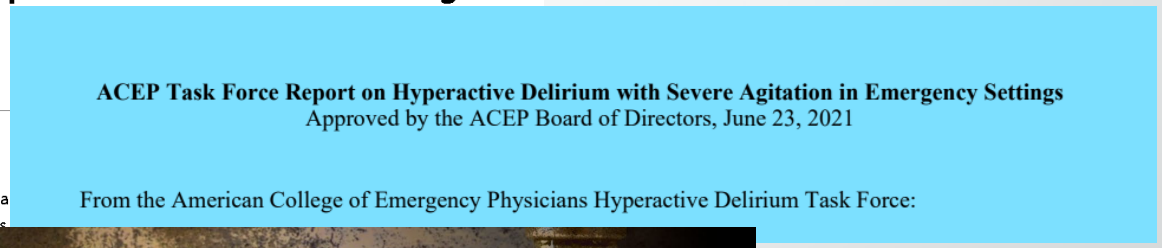
BART leadership is taking steps to build upon more than a decade of reforms and continuous improvements to advance progressive and equitable policing and the commitment to fight racism. Download a 2020 PDF factsheet of BART's commitment to progressive policing that highlights policy updates, reforms, expanded training, and new initiatives to bolster oversight and increase the number of unarmed civilian employees providing presence in the system. View our section on Reforms to Date.

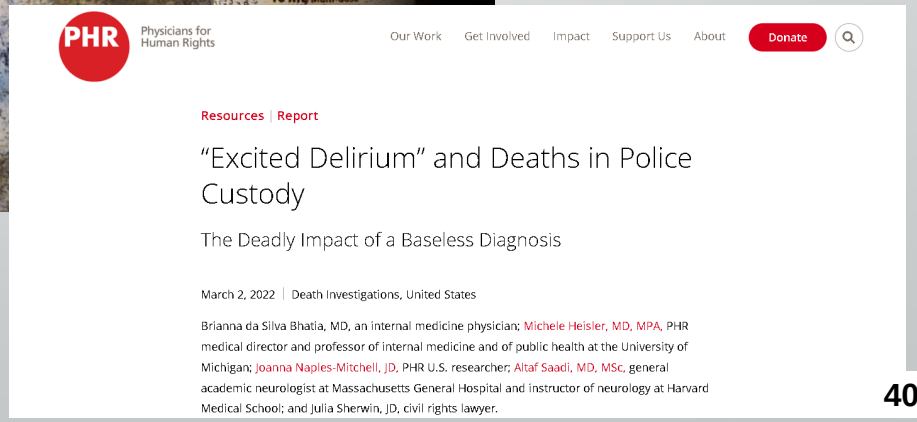
Unpacking "Excited Delirium"

"Excited delirium" is broadly defined as being in a highly agitated and combative state

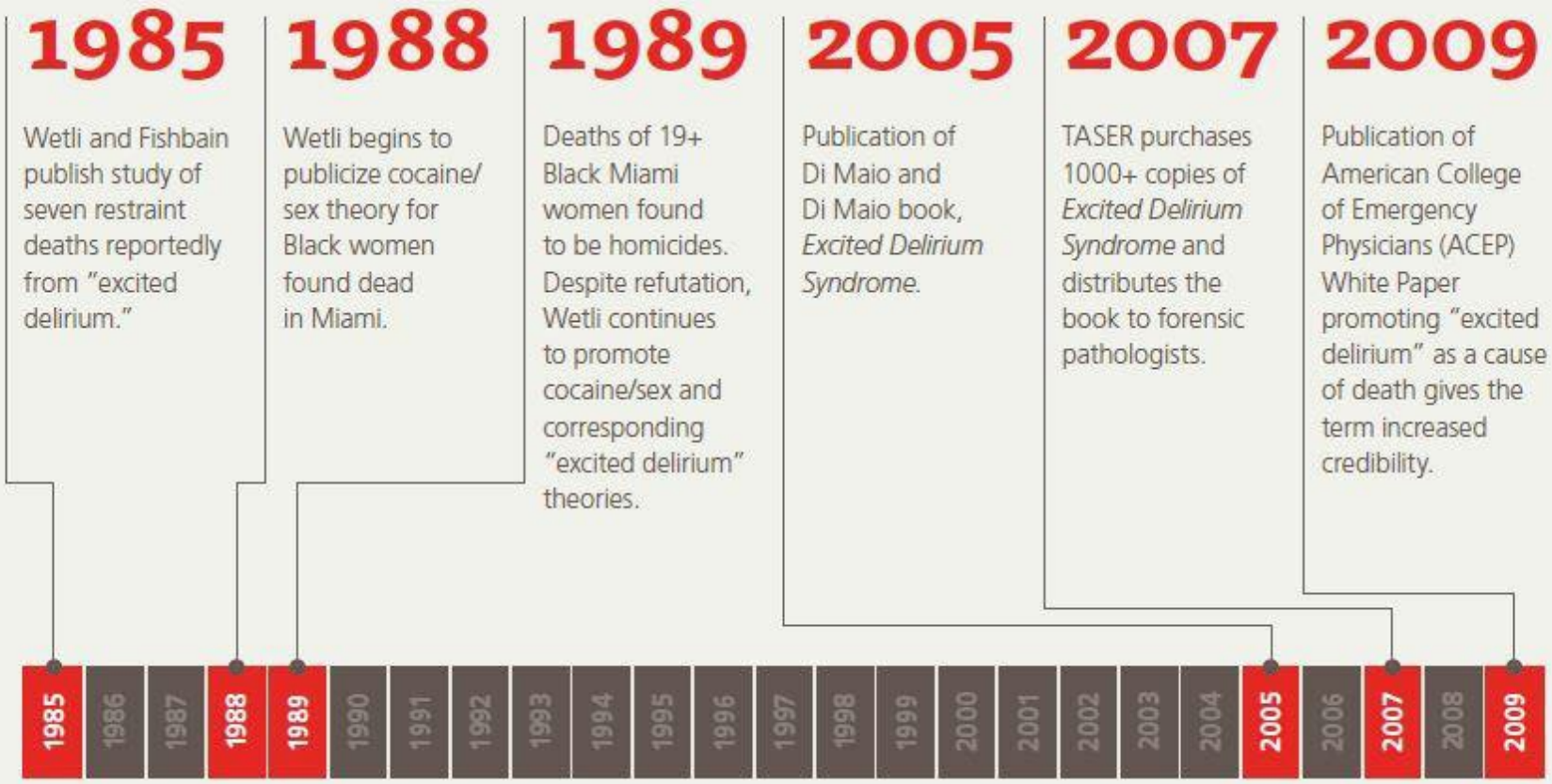
- American Medical Association (AMA) Press Release
- American College of Emergency Physicians (ACEP)
- Lexipol policy change and training
- Physicians for Human Rights Report (PHR)







Origins of "Excited Delirium"



Source: PHR Report pg.28

Since the AMA Press Release

- OIPA identified BPD Policy Manual references
- OIPA met with Deputy Chief, Progressive Policing and Community Engagement Bureau
- OIPA researched 2021 BPD's use of the term in reports
- OIPA met with BPD Subject Matter Experts on Training
- BPD Training Bulletin 22-103 Use of the Recovery Position

The Work of Racial Equity

- Knowing the history
- Using data as a measure
- Receiving authentic community feedback
- Seeking collaboration toward institutional change

BART and BPD not only stand against discrimination but are also fighting racism. As a District we have prioritized advancing systemic racial equity by participating in the Government Alliance on Race and Equity (GARE) training series. BART's Office of Civil Rights oversees a host of equity programs that cover workforce, contract, and economic opportunity policies.

Fighting Racism

Key Takeaways

- "Excited delirium" is not a medical term or diagnosis.
- The behavior and physical symptoms of a person experiencing hyperactive delirium with severe agitation makes them a danger to themselves and others.
- De-escalation strategies, physical restraint techniques, and chemical sedation options have to be trained and further studied by Crisis Intervention Specialists, Law Enforcement and Medical Personnel when interacting with a person experiencing hyperactive delirium with severe agitation to help avoid racial bias.

Recommendations for Discussion

- Remove the term "excited delirium" from the BPD Policy Manual and related training materials.
- Create a BPD Training Bulletin for Officers and Crisis Intervention Specialists about the changes to the Manual as an effort to prevent in-custody deaths and to promote the use of de-escalation techniques.
- District provide additional funding for more opportunities and future trainings for the police department (e.g., provided by Lexipol on the topic).
- Organize a future "Policy Forum" with OIPA, BPD and BPCRB to further discuss this and similar topics to work together on addressing racially disparate outcomes in policing.

PRESS RELEASES

New AMA policy opposes “excited delirium” diagnosis

JUN 14, 2021

CHICAGO — A policy adopted by physicians, residents, and medical students at the American Medical Association’s (AMA) Special Meeting of its House of Delegates (HOD) opposes “excited delirium” as a medical diagnosis and warns against the use of certain pharmacological interventions solely for a law enforcement purpose without a legitimate medical reason.

The new policy addresses reports that show a pattern of using the term “excited delirium” and pharmacological interventions such as ketamine as justification for excessive police force, disproportionately cited in cases where Black men die in law enforcement custody. Specifically, the policy:

- Confirms the AMA’s stance that current evidence does not support “excited delirium” as an official diagnosis, and opposes its use until a clear set of diagnostic criteria has been established
- Denounces “excited delirium” as a sole justification for law enforcement use of excessive force
- Underscores the importance of emergency physician-led oversight of medical emergencies in the field
- Opposes the use of sedative/hypnotic and dissociative drugs—including ketamine—as an intervention for an agitated individual in a law enforcement setting, without a legitimate medical reason
- Recognizes the risk that sedative/hypnotic and dissociative drugs have in relation to an individual’s age, underlying medical conditions, and potential drug interactions when used outside of a hospital setting by a non-physician

Research supporting the new policy echoes current AMA policy recognizing police brutality as a product of structural racism, indicating that racially marginalized and minoritized communities are disproportionately subjected to police force and racial profiling and underscoring the correlation between violent policing and adverse health outcomes. Broadly defined as being in a highly agitated and combative state, studies show that the term “excited delirium” has been misapplied and diagnosed disproportionately in law enforcement-related deaths of Black and Brown individuals, who are also more likely to experience excessive

sedative intervention instead of behavioral de-escalation.

“For far too long, sedatives like ketamine and misapplied diagnoses like ‘excited delirium’ have been misused during law enforcement interactions and outside of medical settings – a manifestation of systemic racism that has unnecessarily dangerous and deadly consequences for our Black and Brown patients,” said AMA President-elect Gerald E. Harmon, M.D. “As physicians and leaders in medicine, it is our duty to define the medical terms that are being used to justify inappropriate and discriminatory actions by non-health care professionals. The adoption of this policy represents an urgent step forward in our efforts to remove obstacles that interfere with safe, high quality medical care – and makes clear that the AMA will continue to aggressively confront all forms of racism or police violence against our patients in marginalized and minoritized communities.”

In addition, the new policy urges law enforcement and frontline emergency medical service (EMS) personnel, who are a part of the “dual response” in emergency situations, to participate in training overseen by EMS medical directors that minimally includes de-escalation techniques and the appropriate use of pharmacological intervention for agitated individuals in the out-of-hospital setting. The policy also urges medical and behavioral health specialists – instead of law enforcement – to serve as first responders and decision-makers in medical and mental health emergencies. It calls for the administration of any pharmacological treatments in an out-of-hospital setting to be done equitably, in an evidence-based, anti-racist, and stigma-free way.

The adoption of the new policy stems from a report on the use of ketamine and pharmacological intervention in the context of “excited delirium,” requested by the AMA BOT, and follows AMA’s advocacy urging lawmakers to act on policing reform to protect public health. It supports existing policy on recognizing racism as a public health threat and acknowledging race as a social, not biological, construct, and is in accordance with AMA’s three-year strategic plan to advance health equity and embed racial justice, released in May 2021. Through the work of its Center for Health Equity, the AMA has remained committed to dismantling structural racism across all of health care and society – starting from within the organization – rooted in scientific evidence showing the harmful effects of racism, discrimination, and other forms of exclusion have on the health of individuals and our nation.

our nation.

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About the American Medical Association

The American Medical Association is the physicians’ powerful ally in patient care. As the only medical association that convenes 190+ state and specialty medical societies and other critical stakeholders, the AMA represents physicians with a unified voice to all key players in health care. The AMA leverages its strength by removing the obstacles that interfere with patient care, leading the charge to prevent chronic disease and confront public health crises and, driving the future of medicine to tackle the biggest challenges in health care.

More about: AMA Policies | Special Meeting of the AMA House of Delegates | Police Brutality | African-American Population Care | AMA Center for Health Equity | Health Care Equity

FEATURED STORIES

PUBLIC HEALTH

Doctors get chance to report late on use of COVID-19 relief funds

SUSTAINABILITY

Doctor shortages are here—and they’ll get worse if we don’t act fast

RESIDENT/STUDENT HEALTH

First came burnout, next suicidal thoughts—then this resident got help

ACEP Task Force Report on Hyperactive Delirium with Severe Agitation in Emergency Settings
Approved by the ACEP Board of Directors, June 23, 2021

From the American College of Emergency Physicians Hyperactive Delirium Task Force:

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Representatives from the American Society of Health-System Pharmacists

Acknowledgement: We gratefully thank Travis Schulz, MLS, AHIP for his tremendous support in this project.

KEY POINTS:

- Hyperactive delirium with severe agitation, a presentation marked by disorientation and aggressive words and/or actions, is an acute life-threatening medical condition that demands emergency medical treatment.

- Patient safety is and must be the primary focus of emergency medical treatment of hyperactive delirium with severe agitation.

- Rapidly restoring normal body physiology, facilitating a safe environment for the patient and medical professionals treating the patient, and providing the opportunity to differentiate and treat life-threatening causes of hyperactive delirium are patient-centered goals of emergency medical treatment of hyperactive delirium with severe agitation.

- De-escalation techniques may be effective and should be attempted when possible.

- Parenteral medications are often required to treat severe agitation. Multiple pharmacologic options exist for effective treatment of hyperactive delirium with severe agitation. There is no consensus on a single "optimal" medication at this time, but ketamine, droperidol, olanzapine, and midazolam delivered via intramuscular injection are the options best supported by current literature.

- Medical treatment of hyperactive delirium with severe agitation – whether prehospital (EMS) or in-hospital (Emergency Department) should be led by a physician board certified in EMS Medicine and/or Emergency Medicine, respectively. All medical treatments must be at the decision of appropriately trained medical professionals on the physician-led care team.

- Additional research is needed to more fully understand inciting pathways and distinct pathophysiology of individual causes of hyperactive delirium with severe agitation. Further research is also warranted to identify optimal medication choices, doses for those medication choices, and additional medical treatments that improve patient-centered outcomes.

CONTEXT FOR THIS PAPER

This document focuses on the emergent patient in the prehospital or emergency department (ED) setting presenting with hyperactive delirium accompanied by severe agitation. In patients with severe agitation, the use of de-escalation techniques is oriented towards preventing disability or death. This clinical scenario requires immediate medical evaluation by clinicians trained in the stabilization, diagnostic evaluation, and initial treatment of the various etiologies associated with hyperactive delirium and may necessitate the use of parenteral sedating medications. However, optimum strategies for preventing morbidity and mortality in patients with hyperactive delirium remain uncertain given the paucity of high-quality research in existence. This paper intends to synthesize the most current

information available regarding recognition, evaluation, and management of patients presenting with hyperactive delirium accompanied by severe agitation when encountered in the prehospital or ED setting. It is not directed towards patients solely demonstrating agitation without signs of delirium or individuals not engaged as patients. The relevant audience is emergency medical services (EMS) professionals, emergency physicians, and ED medical staff (e.g. nurses, technicians). Patient encounters in the field presenting with delirium and severe agitation often involve the interface of law enforcement and EMS. However, all prehospital treatment decisions for patient care fall solely within the domain of physician-led EMS professionals. The expectation is that every patient encounter will involve evaluation and management by appropriately educated and trained EMS professionals in the field and emergency physicians in the ED.

By their nature, syndromes represent a constellation of signs and symptoms without a clearly elucidated singular cause or pathophysiologic definition. This diagnostic uncertainty, along with the dual use of the nomenclature both to describe the initial patient presentation and to provide a causative etiology on post-mortem examination, has led to controversy over use of the term, “Excited Delirium Syndrome,” within medicine and the lay press. Critics of this terminology have raised concern that it has been employed to explain away preventable in-custody deaths as inevitable outcomes, without proper consideration of other contributing factors and alternative management strategies that might have resulted in survival. Supporters of the use of “Excited Delirium Syndrome” have observed patients with agitated or combative behavior that is associated with a delirious state where the individual is not capable of interacting with other individuals or the environment. They recognize such behavior is frequently associated with physiologic abnormalities and high rates of death, warranting immediate treatment to improve patient outcomes. Moreover, the term is only definitively applied as a postmortem cause of death, rather than prospectively at presentation. Given the increasingly charged nature of the term, ACEP is concerned that its use in this document may distract from the intended delivery of critical information surrounding therapeutic options and best practices focused on the patient’s care and survival. Consequently, explicit discussion of “Excited Delirium Syndrome” will only occur in the context of

evidence surroundings its existence as a distinct pathophysiologic phenomenon. Rather, in this paper, we use the term "hyperactive delirium with severe agitation" to describe presentations of interest.

Of note, concerns have been raised about potential bias in a prior publication, the 2009 American College of Emergency Physicians (ACEP) white paper on Excited Delirium Syndrome. Since its publication, ACEP enacted a robust global conflict of interest policy, though notably not in direct response to critics of the 2009 white paper nor with specific concerns regarding the content of that paper or others generated before such a policy was in force. While the authors of this paper were informed by the 2009 paper, this work is *de novo* and not to be construed as an update or refutation of the 2009 paper.

Rather, ACEP has heard urgent questions surrounding initial management of hyperactive delirium presenting with severe agitation raised by its membership, the scientific community at large, community leaders, media, and governmental agencies.[1-5] These questions frequently center on the evidence surrounding the safety of and medical justification for treatment with parenteral sedating medications. Such concerns are addressed within this information paper. In an attempt to involve relevant parties from inception, multiple outside medical organizations, including a patient representative, participated in the drafting of this document.

History and Controversies

Emergency health care professionals are faced with the challenges of treating patients agitated or combative to the point where they cannot be safely or reliably evaluated. For more than a century, medical publications have described dangerous agitation accompanying hyperactive delirium. This phenomenon was recognized as early as 1849 when reports of "Bell's mania" described poor outcomes among psychiatric patients experiencing delirium accompanied by severe agitation prior to the advent of psychotropic medications. The high rate of fatalities in patients suffering from hyperactive delirium due to psychiatric illness prior to the availability of effective treatment underscores the challenge of safely managing this presentation. Although patient demographics, associated medical conditions, and toxic exposures have changed, managing these patients remains challenging.[6-8]

Delirium, or acute cortical-subcortical neuronal encephalopathy, is a form of altered mental status involving a fluctuating disorder of attention and arousal that develops acutely and is characterized by restlessness and illusions, and incoherence of thought and speech.[9] The initial published discussion of excited delirium in the medical literature appeared in a 1981 *Annals of Emergency Medicine* case report describing cocaine intoxication in a "body packer," an individual who attempts to smuggle cocaine by intracorporeal means.[10] This report reviewed subtypes of delirium, stating, "There are two major types of delirium: stuporous (dull, lethargic, hypoactive, mute, somnolent, and apathetic) and excited (thrashing, shouting, hyperactive, fearful, panicky, agitated, hypervigilant, and violent). Patients with excited delirium are more common than the stuporous and, because they present a management problem, are often labeled as suffering from a functional psychiatric illness."

More recent research tends to use the descriptive terminology "hyperactive delirium" rather than "excited delirium" or "agitated delirium" for delirium associated with increased neuromuscular activity, often accompanied by agitation, whereas "hypoactive delirium" occupies the opposite extreme.[11] For consistency, we have chosen to employ the descriptive terminology, hyperactive delirium with severe agitation, as the most accurate language identifying the mental status and the level of activity exhibited by patients of interest. Given that many causes of hyperactive delirium with severe agitation, as well as the presentation itself, are associated with increased mortality, the importance of utilizing a structured diagnostic approach that promotes identification of the correct underlying etiology among a lengthy differential of possible causes is underscored.[12,13]

Frequently overlooked, yet essential to dealing with the challenges inherent in such patient encounters, is the inability to reliably determine on initial assessment the cause(s) of severe agitation in the setting of hyperactive delirium. Such a patient needs rapid de-escalation and calming to allow for definitive medical evaluation and ongoing treatment, in order to avoid preventable fatality due to failure to manage the potential causative life threats, and to treat the danger inherent to the presenting condition. In a delirious patient, severe agitation is an emergency ideally managed using multiple calming measures, often delivered in parallel, to facilitate the safety of all involved, to complete the necessary medical

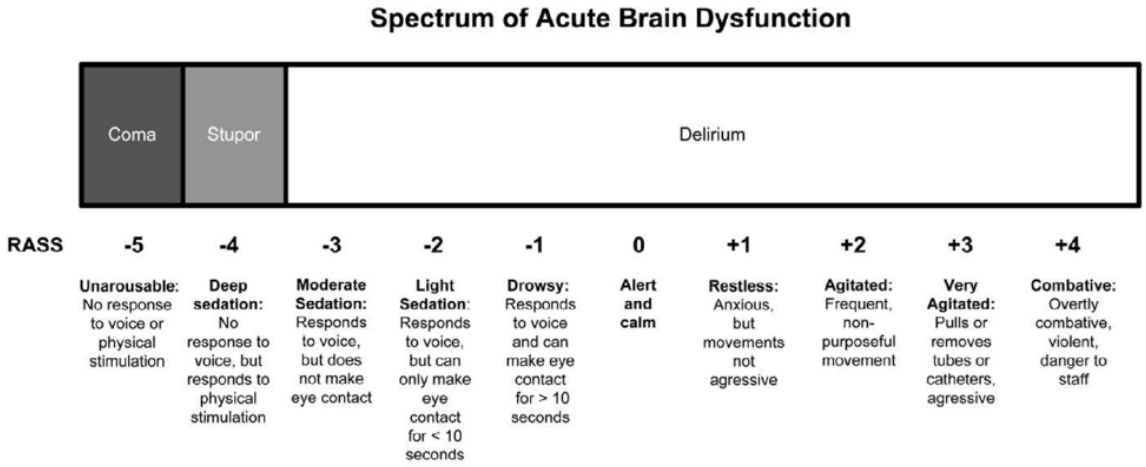
evaluation, and to effectively treat ongoing physiologic derangements that may lead to further decompensation, including fatal outcomes. This critical care should occur while working towards minimization of physical patient restraint and maintenance of patient dignity.[12,14]

CLINICAL PRESENTATION

Description

Hyperactive delirium describes a condition of altered mental status distinguished by disordered thinking and psychomotor agitation, often accompanied by a hyperadrenergic state. Altered mental status in the setting of delirium represents brain function changes such as disorientation, defects in judgment or thought, and disruptions in perception, psychomotor skills, and behavior. It occurs on a continuum, ranging from a hypoactive state (coma) to hyperactive (severe agitation and combativeness), representing extremes of presentation. This spectrum of disease is recognized in multiple scoring systems of acute brain dysfunction, such as the Richmond Agitation-Sedation Scale (RASS) (Figure 1)for critical care patients and the Altered Mental Status Score (AMSS).[15-17] Although not specifically developed in the population of interest, severe agitation in the patient presenting with hyperactive delirium corresponds with RASS of +4 (overly combative, violent, immediate danger to staff) and AMSS of 4 (combative, violent, out of control; loud outbursts of speech; agitated facial expression), though patients with lesser degrees of agitation may require intervention to prevent inadvertent self-harm or injury to caregivers, and to make it possible for medical personnel to identify and treat any dangerous underlying cause of the delirium.

Figure 1. Spectrum of acute brain dysfunction based upon the Richmond Agitation Sedation Scale (RASS).[17] (Used with permission).



Delirium is associated with disordered neurotransmission involving acetylcholine, dopamine, gamma-aminobutyric acid (GABA), and serotonin in the cortical and subcortical regions of the brain.[9] The presentation may result from underlying medical conditions or exposure to toxicants. The condition may be hypoactive, with inattention and decreased activity, or hyperactive, characterized by agitation and combativeness. This paper is limited to consideration of hyperactive delirium demonstrating severe agitation, often involving combative behavior and a hyperadrenergic physiological state.

Differential Diagnosis

Hyperactive delirium with severe agitation, as well as hyperadrenergic physiological states, commonly results from stimulant intoxication and may be caused solely by exposure to this class of drugs. Sympathomimetic toxicity manifests as a broad constellation of signs and symptoms reflecting activation of the autonomic sympathetic nervous system, most commonly due to abuse of cocaine, methamphetamine, or other stimulants. The classic findings of sympathomimetic toxicity are tachycardia, tachypnea, hyperthermia, hypertension, psychomotor agitation, and mydriasis. Patients may also exhibit indefatigability (commonly misinterpreted as “superhuman strength”), confusion, and hyper-attentiveness.[11,18] Distinct exam findings often include tremor, myoclonus, lower extremity rigidity, and repetitive or compulsive behaviors. Features of altered mental status may include aggression, hallucinations and psychosis. Endogenous stress-related catecholamines and exogenous

catecholaminergic drugs likely produce a synergistic effect. Of note, similar presentations of delirium are associated with abrupt cessation of sedative-hypnotic agents. Withdrawal from alcohol, barbiturates, gamma-hydroxybutyrate, or benzodiazepines produces similar clinical features due to release of large amounts of catecholamines, creating an endogenous sympathomimetic syndrome.[18]

Not all cases of hyperactive delirium occur in patients with a history of sympathomimetic use or sedative-hypnotic withdrawal. Alternate etiologies include psychiatric disease and metabolic derangements. As described previously, cases of "Bell's mania" occurred in a psychiatric population prior to the advent of antipsychotic medications and was associated with a diagnosis of schizophrenia.

After initial agitation is treated sufficiently to allow for immediate evaluation, diagnostic testing may identify many causes of altered mental status and agitation. For example, hypoglycemia has been associated with outbursts of violent behavior and/or an appearance of intoxication. However, this diagnosis may be rapidly and conclusively made by determining blood glucose and response to glucose administration. Similarly, stroke, intracranial hemorrhages, and space-occupying CNS lesions causing altered mental status can be discovered with brain imaging. Consequently, awareness of alternative diagnoses along with employment of appropriate diagnostic testing is essential to properly evaluating a patient presenting with hyperactive delirium. In cases where patients rapidly recover as well as in fatal cases without postmortem analysis, underlying medical causes of delirium, such as hypoglycemia, may go undetected. Indeed, hypoglycemia cannot be diagnosed at autopsy due to the natural decrease of glucose concentrations after death. Immediate management of agitation to facilitate a rapid assessment of treatable causes is a fundamental tenet of care of these patients.

It is beyond this paper's scope to exhaustively review all causes of altered mental status and/or delirium. However, it is essential to consider clinical syndromes that may be responsible for hyperactive delirium with severe agitation but do not have immediately available diagnostic testing to confirm the suspected diagnosis. Individuals whose cause of death is listed as "excited delirium" are typically hyperthermic prior to cardiac arrest, suggesting severe physiologic disruption frequently accompanied by extreme psychomotor agitation.[19] Hyperthermia has been described as a "harbinger of death" in the

setting of hyperactive delirium associated with sympathomimetic toxicity.[20] Therefore, hyperthermic conditions have been selected for further discussion. Note that these causative etiologies typically exist along a spectrum of severity based on clinical features and are not diagnosed by rapidly available laboratory or imaging tests. Rather, the clinician relies upon the history, which is often limited, clinical exam findings, and response to treatment. Furthermore, multiple causes may be involved, such as stimulant use exacerbating heat related illness or underlying psychiatric disorder.

Sympathomimetic Toxidrome. The sympathomimetic toxidrome includes hypertension, tachycardia, mydriasis, diaphoresis, hyperreflexia, anxiety, paranoia, agitation, and seizures. It occurs following exposure to excessive doses of stimulant drugs, most often cocaine, methamphetamines, or synthetic cathinones. Depending on the route of administration, sympathomimetic toxicity occurs minutes to hours following exposure. Death is typically due to hyperthermia, dysrhythmia, or hypertensive crisis. These patients often exhibit agitation, aggressiveness, drug induced psychosis, and violent behavior.[21,22]

Alcohol or Sedative-Hypnotic Withdrawal Syndrome/Delirium Tremens. Alcohol withdrawal syndrome (AWS) occurs after cessation of or a reduction in alcohol consumption after a prolonged period of excessive use. Signs and symptoms include anxiety, shakiness/tremor, diaphoresis, vomiting, mild hyperthermia, and tachycardia. A similar syndrome occurs after cessation of sedative-hypnotic agents such as benzodiazepines, barbiturates, or gamma-hydroxybutyrate. Delirium tremens (DTs) falls at the severe end of the spectrum of alcohol withdrawal. DTs typically occurs three days into withdrawal symptoms and lasts for two to three days. It is characterized by a rapid onset of confusion, hallucination, shivering, shaking/tremor, tachycardia, irregular heart rhythm and diaphoresis.[23] Although patients may exhibit dangerous agitation, they are rarely aggressive or violent.

Delirious Mania/Malignant Catatonia

Bell was the first to observe a form of disease resembling some advanced states of mania and fever.[24] There is no clear consensus on the clinical characteristics associated with delirious mania.[25] It is not recognized as a stand-alone diagnosis because many terms have been used over the years to describe patients presenting with mania including excitement, delirium, lethal catatonia, malignant catatonia, and Bell's mania.[25] Delirious mania arises from both psychotic and affective psychiatric diseases and is used to describe manic patients who have delirious symptoms that occur and remit without other evident medical reasons.[25] Delirious mania is a potentially life-threatening but under-recognized neuropsychiatric syndrome.[25] It is characterized by the acute onset of excitement, grandiosity, emotional lability, delusions, and insomnia characteristic of mania and the disorientation and altered consciousness characteristic of delirium.[24] The syndrome may also be accompanied by posturing, stereotypy, mutism, negativism and echo-phenomena suggesting catatonia.[26] The concurrence of delirium and mania is unusual.[25] Catatonia frequently accompanies this syndrome. The distinction between delirious mania and the excited or malignant forms of catatonia is challenging in psychiatry due to diagnostic ambiguity.[24] For example, Fink describes 4 cases of delirious mania. In these cases, delirious mania lasted days to weeks[24] and hospitalization tended to last longer than for manic patients without delirium.[25] These patients may exhibit both agitation and violent behavior.

Serotonin Syndrome. Serotonin syndrome is caused by medications that result in decreased serotonin reuptake, decreased breakdown of serotonin, increased serotonin release, or are serotonin agonists or precursors. Most often, serotonin syndrome is the result of drug-drug interactions but may also result from intentional self-poisoning. Serotonin syndrome is characterized by altered mental status, neuromuscular hyperactivity, and autonomic instability. Typical signs include spontaneous clonus, inducible clonus, ocular clonus, agitation, diaphoresis, tremor and hyperreflexia and muscle rigidity especially in the lower extremities. The Hunter Serotonin Diagnostic Criteria is one set of criteria used to diagnose serotonin syndrome. Note that not all findings need to be present to diagnose serotonin syndrome.[22,27,28]

Figure 2. Decision rules for predicting serotonin toxicity.[27] (Used with permission).

Hunter Serotonin Toxicity Criteria: Decision Rules
In the presence of a serotonergic agent:
1. IF (spontaneous clonus = yes) THEN serotonin toxicity = YES
2. ELSE IF (inducible clonus = yes) AND [(agitation = yes) OR (diaphoresis = yes)] THEN serotonin toxicity = YES
3. ELSE IF (ocular clonus = yes) AND [(agitation = yes) OR (diaphoresis = yes)] THEN serotonin toxicity = YES
4. ELSE IF (tremor = yes) AND (hyperreflexia = yes) THEN serotonin toxicity = YES
5. ELSE IF (hypertonic = yes) AND (temperature > 38°C) AND [(ocular clonus = yes) OR (inducible clonus = yes)] then serotonin toxicity = YES
6. ELSE serotonin toxicity = NO

Patients with serotonin syndrome typically lack violent behavior although agitation may be present.[28]

Neuroleptic Malignant Syndrome (NMS). Neuroleptic malignant syndrome results from repeated exposure to first and second-generation antipsychotics, or abrupt discontinuation of dopaminergic agents. Neuroleptic malignant syndrome typically occurs within the first 2 weeks of antipsychotic medication use. It is defined by unresponsiveness to anticholinergic medications, hyperthermia, increased muscle tone, diaphoresis, dysphagia, fluctuating level of consciousness from stupor and confusion to coma, and an elevated creatine phosphokinase (CPK). Signs include hyperthermia, autonomic instability, severe muscle rigidity, mental status changes, tachycardia, and fluctuating blood pressure. NMS develops over a period of days to weeks and resolves in approximately 7 to 10 days with supportive care and directed treatment.[22,23,28] Patients with NMS are typically not agitated or violent.

Anticholinergic Toxidrome. The anticholinergic toxidrome occurs following exposure to antimuscarinic agents. The presentation includes delirium, dry mucus membranes, dilated pupils, flushed and dry skin, urinary retention, decreased bowel sounds, hyperthermia, and tachycardia. Anticholinergic delirium may cause agitation but rarely purposeful violent behavior. Stereotypical "picking in the air" ("carphologia") and incoherent mumbling are prominent feature of anticholinergic delirium and often distinguishes it from other causes of delirium. The antidote physostigmine often quickly improves the delirium and other symptoms of anticholinergic toxidrome.[28] Patients with anticholinergic delirium typically lack violent behavior although agitation may be present.

Heat-Related Illness. Heat-related illness ranges from heat cramps to heat exhaustion to heat stroke. Heat stroke is an environmental condition resulting from prolonged exposure to or physical exertion in high temperatures and/or high humidity**.** It manifests as tactile hyperthermia, rhabdomyolysis, and delirium. Mental illness and neuroleptic use may exacerbate hyperthermia.

Body temperature rises rapidly to greater than 40ºC (104ºF), and the sweating mechanism fails, so the body is unable to cool. Presentation includes nausea, seizures, altered mental status and sometimes coma. These conditions can most often be distinguished due to history of exertion in high temperatures and lack of violent behavior.[22,23,28]

Thyrotoxicosis. Thyrotoxicosis is the clinical syndrome caused by excess thyroid hormone action at the tissue level due to inappropriately high circulating thyroid hormone concentrations. Findings include heat intolerance, palpitations, anxiety, fatigue, weight loss, and muscle weakness. Clinical findings may include tremor, tachycardia, lid lag, and warm moist skin. Thyroid storm is a life-threatening emergency associated with untreated hyperthyroidism. The likelihood that thyrotoxicosis has progressed to thyroid storm is determined by the Burch-Wartofsky Point Scale (BWPS) which assigns a point value to temperature, central nervous system effects, gastrointestinal-hepatic dysfunction, heart rate, congestive heart failure, presence or absence of atrial fibrillation, and if there was a precipitating event.[23] Thyroid hormone testing is abnormal in such cases, although results may not be available in a timely fashion. Patients with thyrotoxicosis typically lack violent behavior although agitation may be present.

Excited Delirium Syndromes

"Excited delirium" has been listed in cause of death determinations by medical examiners in fatalities thought to result from presentations of hyperactive delirium. However, the descriptive terminology "excited delirium syndrome" (ExDS) has also been used in the EMS and emergency medicine literature to indicate various processes with the common feature of severe agitation in the setting of hyperactive delirium. Controversy has arisen regarding the ability to differentiate ExDS as a distinct entity from causes discussed above. Excited delirium syndrome is not a currently recognized medical or psychiatric diagnosis in either the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-V) of the *American Psychiatric Association,* or the *International Classification of Diseases* (ICD-10) of the World Health Organization. A semantic discussion of the merits of embracing this term was felt to detract from the primary intent of this document, which is to provide updated recommendations for initial management of patients presenting with hyperactive delirium with severe agitation. These patients are at high risk of a fatal outcome likely caused by various treatable etiologies, and from the metabolic consequences of severe exertion itself. Thus, the most important aspect of this document is the discussion of proper evaluation and treatment in such cases. Nevertheless, for contextual completeness and towards transitioning to more precise terminology, a brief discussion of the conclusions reached and limitations of

the evidence surrounding ExDS as a distinct pathophysiologic process follows. Furthermore, the reader is reminded we use the term ExDS in the context of many published references employing this language.

Many of the initial studies of this syndrome relied on forensic data from deaths attributed to ExDS. Because no ICD-10 code for ExDS exists to date, data extraction from medical records is challenging. Case selection often depends on *a priori* definitions of ExDS, which typically include subjective features. Some such descriptors include severe agitation, violence, thrashing, bizarre behavior, inappropriate nudity, extreme paranoia, hypervigilance, lack of tiring, constant physical activity, unusual or unexpected strength, pain tolerance or imperviousness to pain, noncompliance with police directives, combativeness, attraction to reflective surfaces, stupor, fear, and panic.[29] Objective clinical signs associated with ExDS include hyperthermia, tachycardia, tachypnea, increased tidal volume, diaphoresis, and mydriasis. Laboratory data, when available, may reveal hyperkalemia, acidosis, rhabdomyolysis, acute kidney injury, or disseminated intravascular coagulation. Many of these findings are common in hyperactive delirium no matter what the cause. Robust, reproducible data on vital sign abnormalities and laboratory findings are frequently lacking. It is rarely possible to get accurate vital signs in the acute phase of severe agitation. Seizures can occur in fatal ExDS cases, but tend to be more common in patients with known sympathomimetic toxicity and alcohol/sedative withdrawal syndromes.[30]

The incidence of presentations of possible ExDS is difficult to determine because a number of potential cases have historically been handled solely by law enforcement, and an unknown proportion of these have not resulted in a medical system encounter unless an untoward event was recognized. Studies of ExDS derive data from ED encounters,[31,32] EMS encounters,[33-35] encounters with law enforcement officers,[20,36-40] and forensic data.[30,41-50] A presentation with potential ExDS is estimated to occur in 0.02% to 1.5% of EMS encounters.[19,34] Although many case series in the literature rely on law enforcement reports categorizing encounters as ExDS, delirium is a medical emergency that cannot be safely differentiated from purely behavioral concerns by law enforcement personnel. Consequently, there are concerns regarding potential for biased reporting of ExDS in law enforcement literature as justification for in-custody deaths. However, reports of fatal outcomes underscore the emergent nature of the medical

condition at hand. The current literature describes a young (mean age of 33.3 years, range 14 to 71 years)[29] and predominantly male (83% to 95% of ExDS cases) population.[29] Among studies that report patient demographics, Black or African-American race was reported in 33% to 63% of fatal cases[34,37] and 56% of non-fatal cases of severe agitation.[36] Concerns have been raised that differential assessment occurs because persons of color more frequently have dangerous encounters with law enforcement, who may frequently be the source of case finding in the literature.[51] Estimated mortality in presentations with severe agitation where ExDS is suspected ranges from 11.1% to 16.5% depending on the population and case definition,[30,39,40] an exceedingly high proportion of fatal outcomes. However, given that attribution of ExDS is only accurate based on postmortem assessment, prospective study of potential cases is difficult from the point of initial patient contact. Furthermore, less serious cases of severe agitation are also less likely to be captured by the review mechanisms described above.

ExDS presentations are commonly associated with chronic stimulant use disorder, usually cocaine or methamphetamine.[37] A psychiatric diagnosis of schizophrenia accompanied by inconsistent use of psychiatric medications is also frequently seen.[43,52] While most cases of ExDS are associated with sympathomimetic drug use, postmortem analysis shows that not all deaths attributed to ExDS correlate with the detection of drug metabolites; it should also be noted that drug detection capability is limited in any given case by the samples collected and analysis performed.[29]

The proposed pathophysiologic mechanism of chronic stimulant-associated ExDS distinct from other causes are not well studied. As currently theorized, chronic use of cocaine and/or methamphetamine causes increases in extracellular dopamine in crucial areas of the brain with associated alterations in central dopamine transport (DAT) or loss of DAT regulation. Chronic cocaine use impacts hypothalamic D1 and D2 dopamine receptors differentially, such that pathways for generating hyperthermia are not counter-regulated, and severe hyperthermia is allowed to develop.[53] Hyperthermia and hyperactivity may also result from increased thermogenesis due to dopamine alterations in the brain's mesolimbic pathways.[50,54] The disruption of DAT homeostasis by chronic stimulant use creates a hyperdopaminergic state that sets the stage for ExDS in those who are genetically predisposed or situationally “primed” for

ExDS to occur.[43,55] All psychostimulants (e.g., cocaine, methamphetamine, and MDMA) increase the synaptic levels of dopamine,[56,57] which may explain why chronic psychostimulant users are at greater risk for exhibiting the behavioral symptoms associated with ExDS. In people with cocaine use disorder, there is a compensatory upregulation in DAT function, which is an adaptive increase to offset dopamine overflow in the synapse. When this homeostatic control of synaptic dopamine fails, it leads to a functional hyperdopaminergic state, which triggers the acute onset of delirium and marked agitation in ExDS patients.[43,49,50,55]

Oxidative stress has been proposed as a pathogenic mechanism in which cocaine-induced neurotoxicity is induced via production of reactive oxygen species.[58-62] Similarly, reactive oxygen species formed by nicotinamide adenine dinucleotide phosphate (NADPH) oxidase enzymes may be responsible for methamphetamine-induced dopamine-releasing and locomotor-activating properties, based upon a study showing that an antagonist/antioxidant significantly decreases methamphetamine's ability to evoke the dopaminergic response.[63] There is an association between ExDS and gene expression of heat shock proteins 70[64-66] and 90[67] associated with cocaine-induced neurotoxicity. Heat shock protein is thought to be a potential marker for ExDS as it is reported at increased levels in autopsy studies. Oxidative stress is also implicated in decreases of GABAergic neurotransmission due to increased dopamine release in the nigrostriatal nerve terminals[68] with increases in extracellular GABA in the nucleus accumbens.[69] Wetli et al state "the diagnosis of 'agitated delirium' can be made by postmortem measurement of DA synaptic markers in the striatum and the hypothalamus. The distribution found on autopsy is markedly different from both simple cocaine overdose and mechanical or positional asphyxia."[55] Even if a distinct pathophysiologic mechanism identifiable on post mortem examination is ultimately confirmed, EMS professionals and emergency physicians caring for patients will be unable to distinguish ExDS as a distinct etiology from other causes of hyperactive delirium on initial presentation as much of the distinguishing evidence is derived from post-mortem analysis.

There is a paucity of clinical studies on suspected ExDS with a notable exception being the EXCITATION study.[70] This was a prospective multicenter trial that enrolled a convenience sample of

patients who presented to participating EDs with either ExDS (defined as 6 or more of: pain tolerance, tachypnea, sweating, agitation, tactile or measured hyperthermia, non-compliance with police or medical personnel directions, lack of tiring, unusual strength, inappropriately clothed, and mirror or glass attraction) or agitation requiring sedation that did not meet ExDS criteria. A third group of healthy volunteers were exercised and emotionally stressed to serve as a control. Blood stress markers were collected in an attempt to distinguish between patients with ExDS, agitated patients not meeting ExDS criteria, and the control group. Researchers assessed norepinephrine, cortisol, copeptin, orexin A, and dynorphin from the test subjects. Cortisol was more elevated in the ExDS group compared with the other two groups. Orexin was elevated in the ExDS versus the control group but not the non-ExDS agitation group. The trial was not able to identify a single reliable blood marker to differentiate ExDS in living patients.[70]

Neurocardiac dysregulation has also been proposed as a potential contributor to ExDS-associated mortality. There is good evidence that there are cardiovascular afferents to cortical structures and cortical innervation to the cardiovascular system. These neurocardiac pathways can be dysrhythmogenic and can induce ischemia in times of great stress. Examples include myocardial necrosis associated with stroke as well as subarachnoid hemorrhage with myocardial injury noted to be adjacent to cardiac neural tissue as opposed to vascular structures in patients without preexisting coronary atherosclerosis.[71] Additionally, Takotsubo cardiomyopathy is a well-established stress-induced cardiomyopathy.[52,72,73] It has been posited that the hyperadrenergic state associated with chronic substance use, along with stress-induced cortical cardiovascular activity, could contribute to sudden death in agitated patients.[52,72,73]

The importance of a skilled investigation of the scene and circumstances of death cannot be overestimated to fully explore ExDS as a distinct entity. Crucial information such as patient behavior, drug use history, a history or presence of psychosis, or the presence of hyperthermia, can facilitate medical examiner determinations. To improve the precision of death certificate data available for public health surveillance, evidence-based recommendations for the practice of death investigation and autopsy, toxicological analysis, interpretation of toxicology findings, and death certification are necessary.

Certifying a death as "excited delirium due to acute cocaine intoxication" versus simply "acute cocaine intoxication" allows these deaths to be identified, tracked, and studied to better identify unique features of the condition and improve patient care. Without the "excited delirium" component, these deaths are lost as routine acute drug intoxication deaths. Robust documentation may assist future efforts to further our understanding of this presentation.

MANAGEMENT OF HYPERACTIVE DELIRIUM WITH SEVERE AGITATION

There are risks associated with empiric treatment for a presumptive diagnosis in all aspects of medicine; nevertheless, such an approach is required when the patient's clinical condition necessitates the need for resuscitative interventions prior to a definitive diagnosis. The window of evaluation for making a definitive diagnosis is often constrained in the setting of hyperactive delirium with severe agitation due to hemodynamic and respiratory instability and because agitation prevents a more robust initial investigation of causative etiologies. This is particularly true when the differential is broad and the need for intervention is emergent, such as a patient exhibiting agitation sufficiently severe to represent an immediate danger to the patient and to those attempting to care for the patient. Without the ability to immediately determine the cause of severe agitation, and due to the danger to the patient associated with causes of such a state, rapid and effective reduction of severe agitation is essential. The rationale for aggressive treatment of severe agitation is summarized below.

Dangers To:

Patients

Hyperactive delirium with severe agitation is a life-threatening constellation of signs and symptoms with numerous causes discussed above. The combination of vital sign abnormalities, metabolic derangements, altered mental status/agitation, and potential physical trauma raises serious concerns for rapid physiologic deterioration and death[19] particularly in patients with underlying comorbidities (e.g., coronary artery disease, obesity, asthma). Patients presenting in this manner are at high risk of direct physical trauma, not only from unintentional injuries such as falls, but also the secondary physical injuries

that may result from physical restraint. In the setting of severe agitation, restraint without sedation results in a higher injury and fatality rate (Odds Ratio 7.4 for fatality with restraints).[5]

Medical complications due to hyperactive delirium are numerous. Hyperthermia can quickly develop, leading to multiorgan injury. Rhabdomyolysis may be seen not only due to increased metabolic drive, but also in association with physical restraints.[74] Intravascular volume depletion, kidney injury, electrolyte abnormalities and acidemia are all adverse effects potentially exacerbated by physical struggle and restraint. Underlying conditions, such as hypoglycemia, acidosis, life-threatening dysrhythmias and toxic exposure, go untreated until the patient can be safely evaluated by emergency personnel. Additionally, agitation and continued struggling decreases the rapidity of obtaining diagnostic studies such as blood glucose levels and decreases the quality of some diagnostic studies such as CT scans that require the patient remain immobile.

EMS Professionals, Other First Responders, and Hospital -Based Professionals

Beyond the primary concern of harm in the patient, the degree of severe agitation seen with hyperactive delirium presents a physical threat to those in proximity in the field: EMS professionals, police/law enforcement officers, rescue crews, and public bystanders. Unfortunately, physical trauma experienced by EMS professionals, other first responders, and public bystanders occurs frequently in cases of severe agitation.[75] Furthermore, these patients place others in danger of bloodborne and oral pathogen exposure from scratching, biting, and spitting. After transport to the hospital, medical staff, nearby patients, and visitors/family are at risk for the same dangers, including physical trauma, potential for bloodborne and oral pathogen exposure, and psychological injury. Delirious patients are very resource and time-intensive for medical staff, requiring numerous staff and intensive monitoring to ensure safety and appropriate treatment, potentially diverting resources from other critical patients requiring simultaneous care.[76]

De-escalation Techniques

There is broad agreement that patients who present with agitation should initially be provided verbal and non-verbal de-escalation.[77-85] Ideally, verbal techniques for de-escalation are used first. If they fail, more intensive maneuvers can be attempted. Unfortunately, there is a lack of consensus regarding appropriate "verbal" and "non-verbal" de-escalation techniques in severe agitation. In the medical literature, these concepts generally refer to removing the patient from noisy/stimulating environments, offering basic needs such as restroom, food and water, and attempting respectful, verbal interaction.[79,80] At least three papers do offer specifics regarding components of these techniques. While helpful in their relative specificity, the first two reports are focused on inpatients and poorly apply to the prehospital and/or ED settings. A 1991 publication with a focus on mental health nursing included the concepts of personal space for the patient, appropriate open-ended phrases from the clinician, clinician posture and body language, setting an appropriate time limit for the de-escalation attempt, and considerations of environment and personal safety.[85] A more recent publication focused on five types of non-pharmacologic interventions that should be offered before medications for inpatients: description of skills/coping strategies, one-on-one verbal support, distraction with food/water/etc, practical assistance, and relaxation.[78] A third publication provides ten domains of de-escalation related to the emergency environment: respect personal space, do not be provocative, establish verbal contact, be concise, identify wants and feelings, listen closely to what the patient is saying, agree or agree to disagree, set clear limits with clearly verbalized consequences for violations, offer choices and optimism, and debrief the patient and staff.[86,87]

In addition to establishing the components of de-escalation techniques, emerging evidence suggests that effectiveness may also be impacted by the level of specialized training that health care clinicians have received. In the out-of-hospital environment, crisis intervention team (CIT) training that was originally designed for law enforcement officers has been implemented by some EMS agencies to establish formalized de-escalation techniques.[86-88] Specific training for ED personnel regarding these techniques has been associated with decreased use of physical restraints, although evidence surrounding outcomes of interest is limited.[89] Specialized response teams with uniquely trained personnel have been

implemented for response to agitation/behavioral emergencies in both the EMS and ED environments, although conclusive effectiveness studies have yet to be completed.[13,90]

We strongly recommend that the urgency of intervention not inadvertently exclude simple, effective therapies. In a recent large, preliminary analysis of patients in law enforcement custody who were documented as combative and required an EMS response, non-pharmacologic intervention was all that was required in over 80% of cases.[91,92] In nearly all cases, non-pharmacologic interventions may be attempted, even if in parallel with preparations for pharmaceutical administration. As stated above, the circumstances in which severely agitated patients are encountered may require immediate utilization of pharmacologic and physical interventions, but in many scenarios, it is still feasible to attempt verbal and non-verbal de-escalation initially. It appears these techniques may be most effective when provided within a structured format, likely enhanced by assignment of specialized teams. The failure of these de-escalation techniques may indicate a much more severe form of agitation only amenable to treatment with sedating medications.

Pharmacologic Options for Agitation

As opposed to strictly psychiatric or behavioral emergencies with an intact sensorium, patients exhibiting severe agitation due to hyperactive delirium are unlikely to respond to non-pharmaceutical de-escalation techniques due to the degree of brain dysfunction. Such techniques should be attempted at the outset of the patient encounter but if the degree of agitation does not improve or concern for safety requires more rapid control, the timely use of medications to treat severe agitation becomes essential. A sedating medication in a chaotic environment creates a very real risk for respiratory depression and/or airway obstruction, which are well-documented causes of death during prehospital and in-hospital sedation. Proper monitoring of the patient once treatment of agitation allows for close contact is incumbent to minimize these risks.

The two most commonly administered classes of sedating medications in the prehospital environment are benzodiazepines and antipsychotics. In recent years, ketamine has been increasingly used

for these patients.[93] In nearly all cases, initial sedating medications for patients presenting with severe agitation in the setting of hyperactive delirium will be administered parenterally via an intramuscular injection as other administration routes are not practical due to the lack of IV access on initial contact and the degree of agitation present. Oral medications do not provide rapid enough treatment of agitation to be a viable option in the population considered.

A detailed abstraction of studies of medications used by EMS professionals and emergency physicians to treat severe agitation segregated by drug in each study arm is contained in the evidentiary table in the appendix. The evidence surrounding benzodiazepines, antipsychotics, ketamine, and combinations thereof is summarized by class of medication in the discussion below. Additionally, direct comparisons between classes are described when available. Of note, the body of evidence is generally low quality with few direct comparisons between preferred agents, making determination of a clearly superior regimen difficult.

Benzodiazepines

Benzodiazepines bind the gamma aminobutyric acid A ($GABA_A$) receptors in the CNS chloride ion channels. This binding increases inhibitory neurotransmission in the brain causing decreased psychomotor activity, generalized muscle relaxation, and inhibition of catecholamine release. Excessive benzodiazepine dosing can lead to sedation, transient hypotension, and respiratory depression, most often when combined with other sedating agents or in patients with anatomic airway abnormalities. A large amount of published research is available regarding the use of benzodiazepines either as a sole agent or in combination with another agent (most commonly an antipsychotic) for treating severe agitation. Unfortunately, much of this research is limited to case series or is retrospective in nature. Furthermore, the population in the majority of these studies is psychiatric patients rather than undifferentiated emergent patients, although indirect evidence may be of assistance from some of the psychiatric literature. Of the trials available for review, benzodiazepines are typically compared to other sedating agents, and the trials tend to lack placebo or non-pharmacologic arms.[94-97]

The benzodiazepines that have been studied to treat acute, severe agitation via the IM route are midazolam and lorazepam. Direct comparison between these agents occurred in 2 studies. In an RCT, Nobay et al reported on a prospectively randomized group of undifferentiated ED patients who were violent and severely agitated. These patients were randomized to receive midazolam (5 mg IM) or lorazepam (2 mg IM). Both regimens appeared to work effectively to achieve sedation. However, midazolam had mean time to sedation of 18.3 minutes, 13.9 minutes faster than lorazepam. In addition, midazolam demonstrated a more rapid time to re-arousal than lorazepam.[98] Another prospective observational study examined time to sedation in patients receiving midazolam IV (mean dose 3.08 mg), IM (mean dose 2.25 mg), or IN (mean dose 2 mg) compared to lorazepam IV (mean dose 1.9 mg) or IM (mean dose 2.4 mg). The majority of patients received medications via the IV route and the dose of midazolam was lower than typically studied, thus it is unlikely that time to sedation documented in this study is representative of IM administration. Nevertheless, in this study, mean time to control of severe agitation was similar at 14.95 minutes for midazolam versus 17.73 minutes for lorazepam.[99] Multiple additional studies of benzodiazepines compared their use to antipsychotics, ketamine, or a combination of medications but do not directly compare agents. However, it is evident that time to sedation for midazolam IM 5 mg to 10 mg is consistently faster than lorazepam, ranging from 8.5 to 30 minutes for midazolam with the majority of studies falling between 10 to 20 minutes.[16,100-106] Additional studies of lorazepam 2 mg IM utilized less precise time endpoints but time to adequate sedation ranged from 30 to 60 minutes.[96,107,108] Both midazolam and lorazepam may cause equivalent levels of respiratory depression, inconsistent and deeper than anticipated degrees of sedation, and unpredictable duration of sedation with no clear disadvantage for midazolam compared to lorazepam from a safety perspective.[16,98-101,103-108]

To summarize, the benzodiazepines studied for initial treatment of severely agitated patients via IM administration are lorazepam and midazolam. All regimens as single agents at typical doses studied appear effective for controlling agitation. Following IM administration, midazolam achieves desired sedation endpoints faster than lorazepam with mean time to sedation being approximately 10 to 20 minutes for midazolam compared to 30 minutes or greater for lorazepam. All benzodiazepines produce

respiratory depression at higher doses—especially when combined with other sedating medications—and any administration should be followed by close patient monitoring with pulse oximetry, observation of respiratory rate, and continuous waveform capnography at the first opportunity patient condition allows. The safety profile for IM administration is not substantially different between lorazepam and midazolam. Consequently, if using a benzodiazepine for initial treatment of severe agitation, midazolam is recommended rather than lorazepam due to appreciably faster time to adequate sedation.

Antipsychotics

Antipsychotics have a long history of use for agitation, including presentations of hyperactive delirium. They are traditionally grouped into two major subgroups, first generation (haloperidol and droperidol have been studied via IM administration to treat acute agitation) and second generation or atypical (olanzapine and ziprasidone have been studied via IM administration to treat acute agitation) agents. Both subgroups exert their sedative and anti-agitation effects via anti-dopaminergic neurotransmitter effects in the midbrain, sub-cortical regions, and the reticular activating system of the brain. Extrapyramidal side effects (dystonia, akathisia) are relatively common when first generation antipsychotics are used to treat other conditions but are rarely described in studies of sedation. More serious complications, such as neuroleptic malignant syndrome and tardive dyskinesia, are rare with acute administration. In addition, all antipsychotics have the potential to cause prolongation of the QT interval.

Various studies directly comparing antipsychotics have been published. The first-generation agents, droperidol 5 mg to10 mg IM and haloperidol 5 mg to10 mg IM, have been studied as separate arms in 5 studies.[109-113] Droperidol was found to be equivalent or superior to haloperidol in all of these studies. The second-generation agents, olanzapine 10 mg IM and ziprasidone 20 mg IM, were studied as separate arms in one study with olanzapine found to be superior to ziprasidone in achieving adequate sedation at 15 minutes.[103] Multiple comparisons between first- and second-generation antipsychotics have also been published. Droperidol 5 mg IM was compared to olanzapine 10 mg IM in 2 studies with both agents found to be equally effective and with similar safety profiles.[113,114] In contrast, droperidol 5mg IM

was superior to ziprasidone 10 mg to 20mg IM at achieving control of agitation at 15 minutes with increased rates of respiratory depression for those receiving ziprasidone.[16,108] Olanzapine 5 mg to10 mg IM provided equivalent or superior control of agitation when compared to haloperidol 5 mg to 10mg IM.[103,106,113] Haloperidol 5 mg to10 mg IM produced similar effects to ziprasidone 20 mg IM in one study, although both were inferior to other agents studied.[103] Based on studies that directly compare agents, droperidol 5 mg to 10 mg IM and olanzapine 10 mg IM are the best initial options when choosing an antipsychotic for initial treatment of severe, acute agitation.

Many studies have reported time to adequate sedation, although quality and methodologies vary greatly. Nevertheless, there is sufficient data available to estimate an expected time to desired treatment effect. For droperidol 5 mg to 10 mg IM, time to adequate sedation using varied endpoints ranged from 10 to 22 minutes.[16,101,104,108,110,114,115] Similarly, olanzapine 5 mg to 10 mg IM demonstrated mean time to adequate sedation of 11.5 to 17.5 minutes.[94,103,106,114,116] Haloperidol 5 mg to 10 mg IM was slower than both droperidol and olanzapine, with adequate control of agitation at 20 to 60 minutes depending on the study endpoint and, when discretely measured, a mean time to sedation of 11.4 to 28.3 minutes.[96,98-100,102,103,106,107,109-111,113,117-119] Likewise, ziprasidone 10 mg to 20 mg IM was slower than both droperidol and olanzapine, with adequate control of agitation at 17 to 30 minutes.[16,103,108] Unlike other antipsychotics, patients receiving ziprasidone experienced substantially higher instances of respiratory depression.[16,108]

Droperidol is likely the optimal antipsychotic when treating agitation in the setting of hyperactive delirium due to its well-studied safety profile, wide dosing range, and rapid onset compared to most other antipsychotics. Olanzapine is not as well studied providing less confidence that it is equivalent to droperidol. However, data available to date is promising, and there is no evidence to the suggest that olanzapine performs inferior to or has a worse safety profile than droperidol. The preponderance of evidence regarding injectable antipsychotics suggests that droperidol and olanzapine provide the most rapid (10 to 20 minutes to adequate sedation) and effective treatment of agitation. They should be considered first-line agents over ziprasidone or haloperidol.

An additional issue to consider with antipsychotics is the possibility of QTc prolongation leading to torsades de pointes, a life-threatening adverse event. In particular, droperidol was issued a black box warning regarding this potential side effect in 2001 by the U.S. Food and Drug Administration (FDA).[120] This black box warning states that droperidol should be reserved for patients who have not responded to other treatments and that an electrocardiogram (ECG) be performed prior to administration with cardiac monitoring for 2 to 3 hours after administration. These recommendations are impractical for using droperidol for acutely agitated patients presenting with hyperactive delirium. Moreover, QTc prolongation related to common uses of droperidol has not been a complication or concern in subsequent investigations. Independent reviews described below have demonstrated that the black-box warning is unwarranted.[113,121,122] Olanzapine blocks potassium channels to a far lesser degree than other antipsychotics considered. Thus, QT prolongation in patients receiving olanzapine is extremely rare.[116,123]

Several studies have examined QTc prolongation and the occurrence of torsade de pointes in patients receiving medications for agitation within the ED. Droperidol and haloperidol block delayed-rectifier potassium (IKr/HERG) channels in the myocardium, prolonging the QT interval and raising concern regarding the development of torsades des pointes. The majority of the literature addresses droperidol specifically. Knott et al compared QTc following administration of midazolam versus droperidol. Median QTc in the midazolam group was 425 ms. The droperidol group was not significantly different at 439 ms.[124] Despite a QTc of >500 ms in some subjects, no dysrhythmias were seen. In a blinded, randomized trial, Isbister looked for abnormal QT-HR pairs and did not find a difference in patients treated with midazolam, droperidol, or the combination, although numbers in each group were small.[101] Taylor et al compared droperidol versus olanzapine versus combination midazolam/droperidol. Median QTc was 442 ms, 445 ms and 450 ms in each group, respectively. No dysrhythmias were observed.[125] Martel randomized patients to droperidol, ziprasidone, and lorazepam with a median QTc in the droperidol group of 413 ms, no difference in median QTc between drugs studied, and no episodes of torsades de pointes.[108] Chan et al randomized midazolam versus combination midazolam/olanzapine versus midazolam/droperidol.[106] Median QTc was 444 ms, 448 ms and 441 ms in each group,

respectively. No dysrhythmias were seen despite a QTc of >500 ms in two patients (one midazolam and one midazolam/olanzapine). In addition to these randomized studies, Calver et al reported a prospective, multi-center observational study of undifferentiated, agitated ED patients requiring parenteral (IM or IV) droperidol for treatment of agitation.[115] Of the 1,009 study patients, the median total dose of droperidol was 10 mg. Thirteen subjects (1.3%) had an abnormal QTc. Seven of the 13 had another potential cause for the prolonged QTc (another medication associated with prolonged QTc). No dysrhythmias were seen in this study. Multiple large retrospective cohort studies of thousands of agitated prehospital or ED patients receiving droperidol revealed no cases of torsades de pointes.[111-113,122] One retrospective study found the incidence of torsades de pointes in ED patients receiving droperidol to be 1 in 16,546, or 0.006% of patients.[121] Based on the lack of dysrhythmias identified following thousands of cases of studied droperidol administrations, we believe that torsades de pointes is unlikely to occur following droperidol administration at typical IM doses used to treat severe agitation, rendering concerns about this adverse event unwarranted. Furthermore, given the need to rapidly treat severe agitation in hyperactive delirium, obtaining a pre-administration ECG is impractical in these situations.

To summarize the available evidence regarding the use of antipsychotics for ED agitated patients, the best studied agents are droperidol, olanzapine, haloperidol, and ziprasidone. All antipsychotics are effective in reducing the degree of agitation in pre-hospital and ED settings. Intramuscular administration appears to reliably treat agitation, with both droperidol and olanzapine providing adequate sedation within 10 to 20 minutes. However, high quality data on the use of antipsychotic agents to treat agitation in hyperactive delirium is still limited. Despite the FDA black box warning for droperidol, at the commonly utilized doses of 5 mg to 10 mg IM to treat agitation in emergent patients presenting with hyperactive delirium, QTc prolongation is uncommon, and torsades de pointes is unlikely to occur.

Benzodiazepine plus antipsychotic

In addition to studies of individual agents, coadministration of a benzodiazepine and antipsychotic has been compared to monotherapy with either class in a small number of papers. One study

did not find the combination of midazolam 5 mg plus droperidol 5 mg IM to be superior to monotherapy with midazolam 10 mg IM or droperidal 10 mg IM.[101] In that study, median time to adequate sedation for combination therapy was 25 minutes. Three additional studies provided data on time to adequate sedation for the combination of lorazepam 2 mg plus haloperidol 5 mg to 10 mg IM.[99,107,126] While neither lorazepam nor haloperidol monotherapy are preferred for initial treatment of agitation, combination therapy was superior to lorazepam 2 mg IM but not haloperidol 5 mg IM for control of agitation at 60 minutes.[107] These studies demonstrated a time to adequate sedation for combination therapy of 23.3 to 36.5 minutes when timing was measured discretely.[99,126] Given this limited data, there is no compelling evidence to support the combination of a benzodiazepine plus antipsychotic rather than monotherapy with a preferred agent from either class.

Ketamine

Ketamine hydrochloride, a non-selective, non-competitive antagonist of the N-methyl-D-aspartate (NMDA) receptor, is used as a sedative causing complete analgesia, increasing duration of coma with increasing doses, and involving appreciable rates of respiratory depression during the initial phase of coma.[127-129] Ketamine was initially studied as the chemical compound CI-581. The first human trials with CI-581 by Domino et al in 1965 and Corssen et al in 1966 demonstrated that "sensory input may reach cortical receiving areas but fail to be perceived." Specifically, the authors demonstrated through electroencephalogram (EEG) and visual evoked potential studies that CI-581 depresses activity in the association areas of the neocortex and the thalamus, while activating the hippocampus in the limbic system. The depression of one area of the brain, while activating another area of the brain led the authors to propose the concept that CI-581 be labeled as a "dissociative anesthetic."[130-132]

With the introduction of ketamine in emergency medicine for the use of procedural sedation, Green and Krauss wrote that ketamine works by "disconnecting the thalamo-neocortical and limbic systems, effectively dissociating the CNS from outside stimuli."[133] However, this conception overly simplifies the mechanism of effect. Ketamine does not "disconnect" an individual from outside stimuli,

but rather interferes with the circuit in the thalamus that appreciates pain and supports the formation of emotional memory related to the experience in the hippocampus.[132,134,135] Thus, it is perhaps more pharmacologically correct to simply identify that ketamine as a centrally acting anesthetic, with effects ranging from focal to general depending on dose.[132] Ketamine acts on specific areas of the brain related to the perception and memory of painful stimuli, with potential for more global depression of consciousness as the dose increases.

There are two primary advantages to ketamine that make it a useful agent for the management of severe agitation in patients presenting with hyperactive delirium. The first attribute is that it can be administered via the IM route with more reliable achievement of effective sedation compared with benzodiazepines and antipsychotics, although the IM route is slightly less predictable than IV.[128,129] Second, it has a consistently faster onset of action compared to other classes of medication.[130,132,136-138] In one of the earliest studies examining ketamine in clinical practice, Corssen et al reported on 630 patients in an operating room environment. Two-hundred and sixteen patients were treated with IV ketamine, and 76 were treated with IM ketamine. All but 7 patients achieved adequate initial sedation, with those in the IV group achieving sedation adequate to perform procedures within 20 seconds, and those in the IM group achieving sedation within 2 to 3 minutes.[136] Rapidity of onset are essential for any medication chosen for initial reduction of severe agitation in patients with hyperactive delirium.

Most relevant to this document, multiple authors have reported successful treatment of severe agitation with ketamine IM injection.[35,99,105,118,119,126,139-147] These papers are of predominantly low methodological quality consisting of case series, retrospective chart reviews, or small prospective studies. In addition, they employ disparate dosing regimens (most often 4 mg/kg based on estimated weight) and utilize variable sedation endpoints. Time to adequate sedation following ketamine IM for the rapid management of acute agitation in the setting of hyperactive delirium was specifically reported in a subset of these publications.[35,99,118,119,126,139-141,144,145] Despite the poor quality of evidence, reported time to sedation was uniformly rapid with the majority between 2 and 10 minutes (range 1.5 to 15 minutes). Of note, Mankowitz et al conducted a systematic review and meta-analysis of 650 patients from 18

publications utilizing ketamine for managing agitated patients in prehospital or ED encounters. The mean time to sedation was 7.21 minutes with 68.5% achieving sedation in under 5 minutes and 75.6% achieving adequate sedation with a single dose of ketamine.[145] Although the lack of high-quality prospective studies limits the degree of certainty, the current literature suggests that adequate treatment of severe agitation occurs predictably in less than 10 minutes following administration of ketamine 4 mg/kg IM.

Given the demonstration of reliable treatment of agitation and more rapid time to adequate sedation than midazolam, droperidol, or olanzapine, there is a strong argument for ketamine as the preferred initial IM therapy in cases of hyperactive delirium exhibiting severe agitation. However, concerns over the safety profile have led to increased scrutiny of ketamine use for treatment of undifferentiated severe agitation. Although emergence phenomenon related to ketamine is frequently discussed as an adverse event, this is of negligible concern when faced with a severely agitated patient.[126,148] Rather potential hemodynamic and airway complications are of greater import and will be dealt with below.

For a patient presenting with hyperactive delirium with severe agitation patient, if a hypertensive effect does occur after IM administration of ketamine, this could theoretically lead to complications in a patient population whose blood pressure may already be elevated due to sympathomimetic exposure and catecholamine overload. Early volunteer studies of ketamine demonstrated that IV administration could result in elevated blood pressure, typically occurring within 3 to 4 minutes.[132,136,141,149-152] Morgan et al showed that the IM administration of ketamine had less of an effect on raising blood pressure compared to IV administration, but their study was performed in a controlled operating room environment. When hypertensive episodes did occur in patients receiving IM ketamine, the authors noted that these effects were delayed compared to IV administration.[152] In addition, a single center retrospective chart review demonstrated a decrease in systolic blood pressure and heart rate following ketamine.[147] Similarly, a prospective trial revealed no change in heart rate or systolic blood pressure in the first hour following ketamine administration.[99] In contrast, a systematic review reported hypertension in 12.4% of patients

receiving ketamine.[145] In an open label, prospective randomized trial, elevated numbers of patients exhibited hypertension and tachycardia after ketamine administration but this resolved in most cases prior to ED discharge.[126] However, screening for hypertension prior to sedative administration is impractical in most cases and such concerns must be balanced with the risks of ongoing agitation frequently accompanied by sympathomimetic toxicity. To date, there is no evidence to suggest hypertensive complications occur following ketamine administration to treat severe agitation and such concerns should not limit appropriate therapy when indicated.

Second, multiple studies have demonstrated that ketamine administration can result in hypersalivation and laryngospasm. These adverse effects may compromise a patient's respiratory status, although both effects can be managed with definitive airway control in the form of intubation.[136,139,151,153,154] In a prospective study of 64 patients receiving ketamine for prehospital severe agitation, the need for intubation to manage the airway after ketamine administration arose in 2 of 3 patients experiencing laryngospasm and in 4 of 21 patients experiencing hypersalivation.[118] In a subsequent descriptive cohort study performed by the same author, 5 of 49 (10%) patients experienced hypersalivation requiring intubation.[144] Another prospective trial with 45 patients in the ketamine arm described laryngospasm in 2 patients (4.4%) and in hypersalivation in 5 patients (11.1%), with 2 requiring intubation for hypersalivation.[119] A systematic review described laryngospasm in 1.3% of patients and hypersalivation in 19% following ketamine.[145] Additional reports describe hypersalivation and laryngospasm in a minority of patients receiving ketamine for agitation. The majority of these adverse effects are managed without intubation.[139,146,147,155-157] Interestingly, a prospective, randomized open label trial did not demonstrate increased rates of hypersalivation.[126] Both hypersalivation and laryngospasm regularly occur in patients receiving ketamine, although the need for intubation due to these adverse effects is infrequent. Nevertheless, patients receiving ketamine must be monitored for these complications by medical professionals capable of managing the airway.

Studies evaluating respiratory depression separately from hypersalivation and laryngospasm have occurred. Multiple authors have demonstrated cases of decreased ventilatory drive and drops in oxygen

saturation following ketamine administration, although these were not conducted in the prehospital or ED environment.[158-162] Seven studies are available that specifically assess respiratory depression following IM ketamine use for managing agitated patients administered by EMS or ED personnel. In a retrospective chart review of 52 cases, Scheppke et al reported that 5.8% of patients treated with 4 mg/kg of IM ketamine developed significant respiratory depression.[141] In contrast, Hopper et al reported no patients developing hypoxia in their retrospective review of 32 cases.[163] A prehospital retrospective chart review documented 2 intubations for hypoxia/respiratory distress out of 95 patients receiving ketamine for agitation.[146] Another prehospital chart review of patients receiving ketamine for agitation reported 8 of 86 patients intubated for respiratory distress and 3 for apnea. An additional retrospective dose comparison study described 16 intubations for hypoxia/respiratory distress out of 292 subjects receiving ketamine.[157] These studies are all retrospective reviews, making it difficult to interpret their varied results as they are likely dependent on the quality of chart abstraction. A single, prospective randomized open-label trial demonstrated hypoxia (21%) in the group receiving ketamine.[126] In a recently published retrospective review of a large prospectively collected EMS database, out of 3,795 patients who received ketamine for altered mental status/behavioral indications – 10.2% had measured hypoxia and 23% had measured hypercapnia.[93] Finally, a systematic review noted that 1.8% of patients receiving ketamine for agitation experienced transient hypoxia.[145] Although rates of respiratory depression vary between studies, significant respiratory depression occurs regularly. Patients receiving ketamine should be monitored for this complication, ideally with continuous pulse oximetry and $EtCO_2$ monitoring.

Because it is easier to determine through chart review if a patient required mechanical ventilation compared to the development of respiratory depression, other authors have examined intubation rates after ketamine administration to manage agitation in the setting of hyperactive delirium, with results ranging from 0 to 62%.[99,118,142-146,156,157,163,164] The true reason for intubation is not always clear in these studies, and at least in some part reflects variation in practice patterns. For example, Olives et al calculated an Odds Ratio for intubation of 2.57 (95% CI 1.05 to 6.27) for patients managed during the overnight shift compared to patients presenting during the day shift.[143] The authors postulate that perhaps

there is a greater inclination to perform intubation in a patient after arrival to the ED when there are fewer resources, and that the treating emergency physician may find it beneficial to control the airway through intubation compared to dedicating resources toward continual monitoring of a patient's airway. It is also possible that people who develop severe agitation with hyperactive delirium at night do so from different causes than those who develop the syndrome during other times of day. These same authors noted that among the group of ED physicians they studied, individual physician intubation rates varied from 0 to 100%. Other studies have demonstrated individual physicians to more frequently intubate patients who receive prehospital ketamine for agitation.[144,146] In contrast, four publications examining the use of ketamine describe no change[105] or even a decrease,[155,165] in intubation rates when compared to historical controls such as midazolam.[105,148,155,165] Most dramatically, Lebin et al found the introduction of ketamine to treat prehospital agitation was associated with a drop in intubation rates from 63% (historical control of patients treated with benzodiazepines, mostly midazolam) to 3.8% with ketamine.[148] Regardless, the multiple factors contributing to the decision to intubate make this a poor surrogate marker to understand the effect of various doses of ketamine on respiratory depression.

Noting that the literature demonstrates the potential for respiratory depression when ketamine is used for the management of hyperactive delirium with severe agitation, EMS professionals and emergency physicians need to evaluate the proper dose that is effective without causing unwarranted respiratory depression. In terms of context, it is helpful to understand that the current dosing model for treatment of agitation of 4 mg/kg IM that is often used in prehospital protocols was originally extrapolated from a dosing scheme that was developed for pediatric procedural sedation rather than developed prospectively.[35,141,163] Consequently, it is unclear if this is the optimal dose, although such a regimen is widely employed. Specific to the prehospital environment, studies examining different dosing schemes for IM ketamine in managing hyperactive delirium with severe agitation have shown no significant difference in intubation rates between various dose regimens.[143,156,157] However, it is difficult to determine from these studies if there were clinically significant differences in respiratory depression. For example, in the retrospective cohort study of 86 patients given ketamine for agitation by Parks et al,

there was a non-significant difference in dose between patients intubated and those who were not intubated. However, the authors additionally reported on 21 patients who were not intubated yet required supplemental oxygen and did not report on the difference in dosing for those requiring any type of respiratory support versus those who did not require respiratory support[156] At this point, there is no compelling evidence to recommend modifying the typical ketamine dose of 4 mg/kg IM to treat severe agitation.

Despite recent widely publicized events having sparked increased scrutiny, death due to prehospital ketamine administration is exceedingly rare. In a large prospectively collected registry study of 11,291 patients receiving ketamine, including 3,795 receiving ketamine IM/IV with a median dose of 3.7 mg/kg for altered mental status (AMS)/behavioral reasons, ketamine could not be excluded as the cause in only 8 deaths out of the entire cohort. Of these, only 4 received ketamine for AMS/behavioral reasons and only 1 was definitively administered via the IM route. Given the large number of administrations at doses commonly used to treat severe agitation and lack of fatalities documented, this data suggests that ketamine use is unlikely to cause appreciable rates of death in the patient population of interest.[93]

It is clear that ketamine, like other sedating agents, risks respiratory compromise requiring a spectrum of support ranging from supplemental oxygen to intubation. There are insufficient data to date to conclusively determine the proper dose of ketamine IM most appropriate to safely and effectively manage severe agitation. No prospective studies have been performed to examine appropriate dosing in this specific patient population. It is therefore possible that a dose lower than 4 mg/kg IM would be effective with fewer respiratory events. However, an improved safety profile with lower dosing must be balanced with the risk of inadequate severe agitation management leading to prolonged time to effective treatment due to the need for redosing or adjunctive agents. This question warrants further study and emergency physicians should consider this void in the literature when making current decisions in EMS protocols specifying treatment regimens and/or in the ED on the IM ketamine dose when managing patients with hyperactive delirium with severe agitation. Furthermore, the existing dose comparison

studies do not suggest a benefit to lowering the dose from 4 mg/kg. It is essential that treating paramedics and emergency physicians are equipped and prepared to manage ventilatory depression and airway compromise when using ketamine to treat hyperactive delirium with severe agitation.

Comparison Studies

Benzodiazepines versus Antipsychotics

Various investigators have examined benzodiazepine monotherapy alongside antipsychotic monotherapy to treat acute agitation. Midazolam 5 mg to 10 mg IM has been compared to droperidol 5 mg to 10 mg IM in 3 studies.[16,101,104] Time to adequate sedation was similar, although midazolam tended to require additional sedating medications whereas the initial dose of droperidol was more frequently sufficient. In addition, midazolam treated patients demonstrated increased rates of respiratory depression in 2 of the 3 studies.[101,104] Midazolam has also been directly compared to olanzapine in 2 studies.[103,106] Midazalom 5 mg IM was equivalent to olanzapine 10 mg IM in one study with no differential rate of adverse events.[103] However, midazolam 5 mg IM was superior to olanzapine 5 mg IM in the second study with similar rates of adverse events, although the lower dose of olanzapine may have limited the relative effectiveness of the antipsychotic.[106] Thus, when considering the most effective agents from each class, droperidol and midazolam are similar with respect to control of agitation, although midazolam may have increased rates of respiratory depression. Midazolam has also been shown to be equivalent to (and possibly superior to) olanzapine for treatment of severe agitation.

Additional studies have compared various other antipsychotics to midazolam and lorazepam. Haloperidol is well studied for treatment of agitation, although it is consistently inferior to midazolam with respect to time to adequate sedation.[98,100,102,103,106] Likewise, ziprasidone is less well studied but is also inferior to midazolam.[16,103] Lorazepam has been shown to be similar to both haloperidol and ziprasidone but inferior to droperidol.[96,107,108] None of these alternative medications perform as well as droperidol, midazolam, or olanzapine.

Ketamine versus Other Agents

Studies directly comparing ketamine to other agents to treat acute severe agitation are limited. Three studies have examined ketamine alongside midazolam. Riddell demonstrated superiority of ketamine IV/IM compared to midazolam IV/IM/IN.[99] Holland found that ketamine at a mean dose of 3.75 mg/kg IM performed similarly to midazolam 5 mg IM with no appreciable difference in rates of adverse events.[105] A third study of prehospital ketamine and midazolam found that rates of intubation were dramatically lower at the receiving hospital in the group receiving ketamine compared to midazolam (3.8% versus 63%).[148] Unfortunately, there are not additional studies that compare ketamine to the first line antipsychotics: droperidol or olanzapine. Rather, ketamine has been compared to haloperidol in three studies.[99,118,119] All found ketamine to be superior in achieving rapid, adequate sedation. However, intubation occurred more frequently in the ketamine treated subjects in 2 of the 3 studies.[118,119] Two studies found ketamine to be superior to the combination of lorazepam plus haloperidol with similar rates of adverse events.[99,126] Although the body of evidence is small, the information published to date suggests that ketamine is at least as effective as the other first line agents: droperidol, olanzapine, and midazolam, with an adverse event profile similar to midazolam.

Summary of pharmacologic options

For EMS personnel or emergency physicians faced with the need to treat a patient presenting with hyperactive delirium with severe agitation, multiple pharmacologic options are available. Ketamine likely provides the fastest time to adequate sedation, though there may be an increased rate of respiratory related adverse events compared to droperidol and olanzapine. Midazolam, droperidol, and olanzapine all demonstrate similar times to adequate sedation. All three are slightly slower compared to ketamine. The adverse event profile for midazolam is similar to ketamine, with increased rates of respiratory depression and intubation along with variable depth of sedation when compared to droperidol and olanzapine. Droperidol, and to a lesser extent olanzapine, has been widely studied with safe use documented thousands of times. No appreciable risk of torsades de pointes with use of droperidol to treat agitation has

been identified, and its use should not be limited by this concern. Either of these antipsychotics are less likely to result in serious drug-related adverse events when compared to ketamine and midazolam. However, the overall body of evidence is generally low quality making it difficult to determine a clearly superior regimen with certainty. Nevertheless, there is abundant experience in the expert panel along with sufficient differentiation within each class of medication in the literature to provide multiple reasonable options for initial treatment of agitation (Table 1).

Table 1. Pharmacologic options to treat hyperactive delirium with severe agitation.

Drug	Dose	Time to adequate sedation
Ketamine	4 mg/kg IM	2 to 15 minutes
Droperidol	5 mg to 10 mg IM	10 to 20 minutes
Olanzapine	10 mg IM	10 to 20 minutes
Midazolam	5 mg to 10 mg IM	10 to 20 minutes

Future Research

While notable research endeavors since 2009 have enabled a stronger evidenced-based review of both hyperactive delirium with severe agitation and specific therapies, many areas for scientific investigation remain. In light of these knowledge gaps, and acknowledging the challenges inherent to research in a population presenting with hyperactive delirium and severe agitation due to a wide range of potential causes, we offer the following topical list in support of emerging research. Specific needs are those related to finding additional approaches towards patient safety, stabilization, and promotion of optimal health outcomes.

Education and training:

- Impact of coordinated training across the continuum of professionals interfacing with hyperactive delirium with severe agitation patients, including law enforcement, EMS, nursing, nurse practitioners, physician assistants, and physicians across the spectrum of medical specialties.

- Identifying "core content" curricula for hyperactive delirium with severe agitation to standardize care.

- Identifying optimal platforms, delivery techniques, and timing of professional development education on hyperactive delirium with severe agitation.

- Identification and impact of de-escalation techniques that protect patient safety and reduce risk of injury to public safety and medical professionals.

- Identifying knowledge and knowledge gaps about hyperactive delirium with severe agitation in law enforcement, EMS, nursing, nurse practitioners, physician assistants, and physicians across the spectrum of medical specialties.

Inciting events:

- Identifying underlying co-morbidities that predispose to hyperactive delirium with severe agitation and may represent modifiable risk factors.

- Identifying precipitating factors that allow for early intervention to prevent progression to hyperactive delirium.

Pathophysiologies:

- Impact of severe agitation on oxygenation and ventilation, including airway protection and risk of airway obstruction.

- Role of electrophysiologic abnormalities and dysrhythmias, possibly related to metabolic derangements, that increase risk of sudden death in the setting of hyperactive delirium with severe agitation.

Assessment:

- Standardized and validated instrument to be uniformly used for research on treatment of hyperactive delirium with severe agitation in the ED

- Validated assessment tools for use in the clinical environment to direct pharmacologic and non-pharmacologic treatment

Therapies:

- Development of comprehensive strategies for de-escalation.

- Identification of optimal medication regimen for treatment of hyperactive delirium with severe agitation by EMS and ED professionals.

- Examining methods of minimizing adverse events when patients are treated for acute agitation in hyperactive delirium with severe agitation.

Conclusions

Over the past decade, progress has been made in identifying distinguishing features, causative etiologies, and effective therapies to treat hyperactive delirium presenting with severe agitation. When faced with a patient presentation concerning for hyperactive delirium, rapid management of severe agitation is necessary to prevent injury to the patient and others as well as to permit clinicians to identify and treat dangerous underlying causes. While it is often impossible to accurately differentiate causes of hyperactive delirium with severe agitation early in the patient encounter, best practice is to initially attempt de-escalation techniques. Due to dangers to the patient, restraints should be utilized as a temporizing measure and are not a substitute for adequate treatment of severe agitation. Pharmacologic management is often necessary. Based on available data, ketamine dosed at approximately 4 mg/kg IM appears to provide the most rapid and reliable results, although regimens from 2 mg to 5 mg/kg have been reported. Alternative IM medications with best evidence for treatment of agitation include droperidol 5 mg to 10 mg, olanzapine 10 mg, or midazolam 5 mg to 10 mg. Of note, it remains unclear whether benzodiazepine-based regimens are less likely to result in respiratory compromise than ketamine, although the recommended antipsychotics demonstrate only rare instances of respiratory adverse events. This uncertainty is due to the heterogeneity of studies available, high rates of intubation necessitated by critical illness and life-threatening causative etiologies, and difficulties studying a population that presents at an extreme of severe agitation. Even though ketamine demonstrates more rapid management of agitation, it is also not clear whether the difference in time to effect improves clinical outcomes in all cases. Thus, appropriately dosed ketamine, droperidol, olanzapine and midazolam administered via IM injection are all reasonable initial options to treat agitation in the setting of hyperactive delirium with severe agitation. No matter the choice of therapy, a minority of these patients will subsequently require intubation due to critical illness, progression of disease, or failure to adequately treat severe agitation with initial intervention. This outcome should not necessarily be considered as an adverse event given that the population being treated is critically ill at presentation.

As soon as it is safe, patients presenting with hyperactive delirium with severe agitation should be placed on ECG monitoring, pulse oximetry, and continuous waveform capnography. Complete vital signs and point-of-care blood glucose should be obtained. Imaging and laboratory studies as indicated within the ED should accompany treating the patient for any time-dependent emergency. No patient with hyperactive delirium with severe agitation should be released from the field into a non-medical setting following sedative treatment as many causes of hyperactive delirium with severe agitation, along with the condition itself, are life-threatening conditions when not properly recognized and treated.

REFERENCES

1. Excited Delirium: The Controversial Syndrome That Can Be Used to Protect Police from Misconduct Charges. *CBS News*, CBS Interactive. 12 December 2020. Available at: www.cbsnews.com/news/excited-delirium-police-custody-george-floyd-60-minutes-2020-12-13/. Accessed March 13, 2021.

2. De Yoanna M, Solomon R. Medics in Colorado dosed 902 people with ketamine for "excited delirium" in 2.5 years, including Elijah McClain. *The Colorado Sun* [Denver]. 23 July 2020. Available at: https://coloradosun.com/2020/07/23/ketamine-use-paramedics-elijah-mclain/. Accessed March 13, 2021.

3. O'Hare M, Budhu J, Saadi A. 'Excited delirium or whatever'? It's not a real thing. *Star Tribune* [Minneapolis]. 20 July 2020. Available at: https://www.startribune.com/excited-delirium-or-whatever-it-s-not-a-real-thing/571839801/. Accessed April 29, 2021.

4. Anesthesiologists Group Says Ketamine Protocols Should Be Reviewed. Journal of Emergency Medical Services. 23 July 2020. Available at: https://www.jems.com/news/anesthesiologist-group-says-ketamine-protocols-should-be-reviewed/. Accessed April 28, 2021.

5. Strömmer EMF, Leith W, Zeegers MP, et al. The role of restraint in fatal excited delirium: a research synthesis and pooled analysis. *Forensic Sci Med Pathol*. 2020;16:680-692.

6. Kraines SH. Bell's Mania (Acute Delirium). *Am J Psychiatry*. 1934;91:29-40.

7. Wozniak JSG. Missing the moral: excited delirium as a negative case study of a moral panic. *Punishment & Society*. 2016;18:198-219.

8. DiMaio TG, DiMaio VJM. *Excited Delirium Syndrome: Cause of Death Prevention*. Boca Raton, FL: CRC; 2006.

9. Brent DA, Max J. Psychiatric sequelae of concussions. *Curr Psychiatry Rep*. 2017;19: 108. doi: 10.1007/s11920-017-0862-y.

10. Fishbain DA, Wetli CV. Cocaine intoxication, delirium, and death in a body packer. *Ann Emerg Med*. 1981;10:531-532.

11. Wilson JE, Mart MF, Cunningham C, et al. Delirium. *Nat Rev Dis Primers*. 2020;6.

12. Roppolo LP, Morris DW, Khan F, et al. Improving the management of acutely agitated patients in the emergency department through implementation of Project BETA (Best Practices in the Evaluation and Treatment of Agitation). *J Am Coll Emerg Physicians Open*. 2020;1:898-907.

13. Wong AH, Ray JM, Auerbach MA, et al. Study protocol for the ACT response pilot intervention: development, implementation and evaluation of a systems-based Agitation Code Team (ACT) in the emergency department. *BMJ Open*. 2020;10: e036982. doi: 10.1136/bmjopen-2020-036982.

14. Wilson MP, Pepper D, Currier GW, et al. The Psychopharmacology of Agitation: Consensus Statement of the American Association for Emergency Psychiatry Project BETA Psychopharmacology Workgroup. *West J Emerg Med*. 2012;13:26-34.

15. Sessler CN, Grap MJ, Brophy GM. Multidisciplinary management of sedation and analgesia in critical care. *Semin Respir Crit Care Med*. 2001;22(2):211-216.

16. Martel M, Sterzinger A, Miner J, et al. Management of acute undifferentiated agitation in the emergency department: a randomized double-blind trial of droperidol, ziprasidone, and midazolam. *Acad Emerg Med*. 2005:12(12):1167-1172.

17. Han JH, Wilber ST. Altered mental status in older patients in the emergency department. *Clin Geriat Med*. 2013;29:101-136.

18. King AM, Malone ER, Aaron CK. "Sympathomimetic syndrome." Critical Care Toxicology. Springer; 2017; pp. 549-567.

19. Baldwin S, Hall C, Bennell C, et al. Distinguishing features of Excited Delirium Syndrome in non-fatal use of force encounters. *J Forensic Leg Med*. 2016;41:21-27.

20. Hall CA, Kader AS, Danielle McHale AM, et al. Frequency of signs of excited delirium syndrome in subjects undergoing police use of force: descriptive evaluation of a prospective, consecutive cohort. *J Forensic Leg Med*. 2013;20:102-107.

21. *Special Panel Review of Excited Delirium*. Weapons & Protective Systems Technologies Center. December 2011. Available at: https://www.prisonlegalnews.org/media/publications/wpstc_special_panel_review_excited_delirium_dec_2011.pdf. Accessed April 28, 2021.

22. Rusyniak D, Sprague JE. Hyperthermic syndromes induced by toxins. *Clin Lab Med*. 2006;26:165-184.

23. Simon HB. Hyperthermia. *N Engl J Med*. 1993;329:483-487.

24. Fink M. Delirious mania. *Bipolar Disord*. 1999;1:54-60.

25. Lee BS, Huang SS, Hsu WY, et al. Clinical features of delirious mania: a series of five cases and a brief literature review. *BMC Psychiatry*. 2012;12:65.

26. Detweiler MB, Mehra A, Rowell T, et al. Delirious mania and malignant catatonia: a report of 3 cases and review. *Psychiatr Q*. 2009;80:23-40.

27. Dunkley EJ, Isbister GK, Sibbritt D, et al. The Hunter Serotonin Toxicity Criteria: simple and accurate diagnostic decision rules for serotonin toxicity. *QJM*. 2003;96:635-642.

28. Caroff SN, Watson CB, Rosenberg H. Drug-induced hyperthermic syndromes in psychiatry. *Clin Psychopharmacol Neurosci*. 2021;19:1-11.

29. Gonin P, Beysard N, Yersin B, et al. Excited delirium: A systematic review. *Acad Emerg Med*. 2017;25:552-565.

30. Ruttenber AJ, Lawler-Heavner J, Yin M, et al. Fatal excited delirium following cocaine use: epidemiologic findings provide new evidence for mechanisms of cocaine toxicity. *J Forensic Sci*. 1997;42:25-31.

31. Wetli CV, Fishbain DA. Cocaine-induced psychosis and sudden death in recreational cocaine users. *J Forensic Sci*. 1985;30:873-880.

32. Blaho K, Winbery S, Park L, et al. Cocaine metabolism in hyperthermic patients with excited delirium. *J Clin Forensic Med*. 2000;7:71-76.

33. Iwanicki JL, Barrett W, Saghafi O, et al. Prehospital ketamine for excited delirium in the setting of acute drug intoxication. *Clinical Toxicology*. 2014;52:685-686.

34. Stratton SJ, Rogers C, Brickett K, et al. Factors associated with sudden death of individuals requiring restraint for excited delirium. *Am J Emerg Med*. 2001;19:187-191.

35. Scaggs TR, Glass DM, Hutchcraft MG, et al. Prehospital ketamine is a safe and effective treatment for excited delirium in a community hospital based EMS system. *Prehospital Disaster Med*. 2016;31:563-569.

36. Strote J, Wash M, Auerbach D, et al. Medical conditions and restraint in patients experiencing excited delirium. *Am J Emerg Med*. 2014;32:1093-1096.

37. Grant JR, Southall PE, Mealey J, et al. Excited delirium deaths in custody: past and present. *Am J Forensic Med Pathol*. 2009;30:1-5.

38. Ross DL. Factors associated with excited delirium deaths in police custody. *Mod Pathol*. 1998;11:1127-1137.

39. Southall P, Grant J, Fowler D, et al. Police custody deaths in Maryland, USA: an examination of 45 cases. *J Forensic Leg Med*. 2008;15:227-230.

40. Best D, Havis S, Gossop M, et al. The risk of drug swallowing at the point of arrest: an analysis of 24 cocaine-related deaths following police care or custody in England and Wales. *Polic Soc*. 2004;14:380-391.

41. O'Halloran RL, Lewman LV. Restraint asphyxiation in excited delirium. *Am J Forensic Med Pathol.* 1993;14:289-295.

42. Martín Cazorla F, Amaya S, Miguel I, et al. Muerte por síndrome de delirium agitado en Andalucía. *Rev Esp Med Leg*. 2010;36:62-67.

43. Mash DC, Duque L, Pablo J, et al. Brain biomarkers for identifying excited delirium as a cause of sudden death. *Forensic Sci Int*. 2009;190:e13-19.

44. Strote J, Range Hutson H. Taser use in restraint-related deaths. *Prehosp Emerg Care*. 2006;10:447-450.

45. Pollanen MS, Chiasson DA, Cairns JT, et al. Unexpected death related to restraint for excited delirium: a retrospective study of deaths in police custody and in the community. *CMAJ*. 1998;158:1603-1607.

46. Michaud A. Restraint related deaths and excited delirium syndrome in Ontario (2004-2011). *J Forensic Leg Med*. 2016;41:30-35.

47. Segal DM, Moraes CT, Mash DC. Up-regulation of D3 dopamine receptor mRNA in the nucleus accumbens of human cocaine fatalities. *Brain Res Mol Brain Res*.1997;45:335-339.

48. Mash DC, Ouyang Q, Pablo J, et al. Cocaine abusers have an overexpression of alpha-synuclein in dopamine neurons. *J Neurosci*. 2003;23:2564-2571.

49. Mash DC, Pablo J, Ouyang Q, et al. Dopamine transport function is elevated in cocaine users. *J Neurochem*. 2002;81:292-300.

50. Staley JK, Rothman RB, Rice KC, et al. Kappa2 opioid receptors in limbic areas of the human brain are upregulated by cocaine in fatal overdose victims. *J Neurosci*. 1997;17:8225-8233.

51. Edwards F, Lee H, Esposito M. Risk of being killed by police use of force in the United States by age, race-ethnicity, and sex. *Proc Natl Acad Sci U S A*. 2019;116:16793-16798.

52. Baldwin S, Hall C, Blaskovits B, et al. Excited delirium syndrome (ExDS): Situational factors and risks to officer safety in on-fatal use of force encounters. *Int J Law Psychiatry*. 2018;60:26-34.

53. Mash D. Excited delirium and sudden death: a syndromal disorder at the extreme end of the neuropsychiatric continuum. *Front Physiol*. 2016;7: doi: 10.3389/fphys.2016.00435.

54. Ruttenber JA, McAnally HB, Wetli CV. Cocaine-Associated Rhabdomyolysis and Excited Delirium: DifferentStages of the Same Syndrome. *Am J Forensic Med Pathol*. 1999;20:120-127.

55. Wetli CV, Mash D, Karch SB. Cocaine-associated agitated delirium and the neuroleptic malignant syndrome. *Am J Emerg Med*. 1996;14:425-428.

56. Amara SG, Kuhar MJ. Neurotransmitter transporters: recent progress. *Annu Rev Neurosci*. 1993;16:73-93.

57. Giros B, Caron MG. Molecular characterization of the dopamine transporter. *Trends Pharmacol Sci*. 1993;14:43-49.

58. Gill JR. The syndrome of excited delirium. *Forensic Sci Med Pathol*. 2014;10:223-228.

59. Pedrajas JR, McDonagh B, Hernandez-Torres, et al. Glutathione is the resolving thiol for thioredoxin peroxidase activity of 1-Cys Peroxiredoxin without being consumed during the catalytic cycle. *Antioxid Redox Signal*. 2016;24:115-128.

60. Uys JD, Knackstedt L, Hurt P, et al. Cocaine-induced adaptations in cellular redox balance contributes to enduring behavioral plasticity. *Neuropsychopharmacology*. 2011;36:2551-2560.

61. Sordi AO, Pechansky F, Kessler FHP, et al. Oxidative stress and BDNF as possible markers for the severity of crack cocaine use in early withdrawal. *Psychopharmacology*. 2014;231:4031-4039.

62. Muriach M, López-Pedrajas R, Barcia JM, et al. Cocaine causes memory and learning impairments in rats: involvement of nuclear factor kappa B and oxidative stress, and prevention by topiramate. *J Neurochem*. 2010;114:675-684.

63. Miller DK, Oelrichs CE, Sun GY, et al. Subchronic apocynin treatment attenuates methamphetamine-induced dopamine release and hyperactivity in rats. *Life Sci*. 2014;98:6-11.

64. Mas VR, Fisher RA, Archer KJ, et al. Proteomics and liver fibrosis: identifying markers of fibrogenesis. *Expert Rev Proteomics*. 2009;6:421-431.

65. Riezzo I, Cerretani D, Fiore C, et al. Enzymatic-nonenzymatic cellular antioxidant defense systems response and immunohistochemical detection of MDMA, VMAT2, HSP70, and apoptosis as biomarkers for MDMA (Ecstasy) neurotoxicity. *J Neurosci Res*. 2010;88:905-916.

66. Lind D, Franken S, Kappler J, et al. Characterization of the neuronal marker NeuN as a multiply phosphorylated antigen with discrete subcellular localization. *J Neurosci Res*. 2005;79:295-302.

67. Madrigal-Matute J, Fernandez-Garcia CE, Gomez-Guerrero C, et al. HSP90 inhibition by 17-DMAG attenuates oxidative stress in experimental atherosclerosis. *Cardiovasc Res*. 2012;95:116-123.

68. Centonze D, Picconi, Baunez C, et al. Cocaine and amphetamine depress striatal GABAergic synaptic transmission through D2 dopamine receptors. *Neuropsychopharmacology*. 2002;26:164-175.

69. Xi ZX, Ramamoorthy S, Shen H, et al. GABA transmission in the nucleus accumbens is altered after withdrawal from repeated cocaine. *J Neurosci*. 2003;23:3498-3505.

70. Vilke GM, Mash DC, Pardo M, et al. EXCITATION study: Unexplained in-custody deaths: Evaluating biomarkers of stress and agitation. *J Forensic Leg Med*. 2019;66:100-106.

71. Taggart P, Critchly H, Lambiase PD. Heart-brain interactions in cardiac arrhythmia. *Heart*. 2011;97:698-708.

72. Lindsay J, Paixao A, Chao T, et al. Pathogenesis of the Takotsubo syndrome: a unifying hypothesis. *Am J Cardiol*. 2010;106:1360-1363.

73. Ceblin MS, Hirsch SC. Human stress cardiomyopathy. Myocardial lesions in victims of homicidal assaults without internal injuries. *Hum Pathol*. 1980;11:123-132.

74. Hick JL, Smith SW, Lynch MT. Metabolic acidosis in restraint-associated cardiac arrest: a case series. *Acad Emerg Med*. 1999;6:239-243.

75. Maguire BJ, Smith S. Injuries and fatalities among emergency medical technicians and paramedics in the United States. *Prehosp Disaster Med*. 2013;28:376–382.

76. Kostas TR, Zimmerman KM, Rudolph JL. Improving delirium care: prevention, monitoring, and assessment. *Neurohospitalist*. 2013;3:194-202.

77. Baldacara L, Ismael F, Leite V, et al. Brazilian guidelines for the management of psychomotor agitation. Part 1. Non-pharmacological approach. *Braz J Psychiatry*. 2019;41:153-167.

78. Martin K, Arora V, Fischler I, et al. Analysis of non-pharmacological interventions attempted prior to pro re nata medication use. *Int J Ment Health Nurs*. 2018;27:296-302.

79. Aubanel S, Bruiset F, Chapuis C, et al. Therapeutic options for agitation in the intensive care unit. *Anaesth Crit Care Pain Med*. 2020;39:639-646.

80. Gottlieb M, Long B, Koyfman A. Approach to the agitated emergency department patient. *J Emerg Med*. 2018;54:447-457.

81. Harwood RH. How to deal with violent and aggressive patients in acute medical settings. *J R Coll Physicians Edinb*. 2017;47:176-182.

82. Marder SR. A review of agitation in mental illness: treatment guidelines and current therapies. *J Clin Psychiatry*. 2006;67 Suppl 10:13-21.

83. Patel MX, Sethi FN, Barnes TR, et al. Joint BAP NAPICU evidence-based consensus guidelines for the clinical management of acute disturbance: De-escalation and rapid tranquillisation. *J Psychopharmacol*. 2018;32:601-640.

84. Vieta E, Garriga M, Cardete L, et al. Protocol for the management of psychiatric patients with psychomotor agitation. *BMC Pscyhiatry*. 2017;17:328.

85. Stevenson S. Heading off violence with verbal de-escalation. *J Psychosoc Nurs Ment Health Serv*. 1991;29:6-10.

86. Richmond JS, Berlin JS, Fishkind AB, et al. Verbal De-escalation of the Agitated Patient: Consensus Statement of the American Association for Emergency Psychiatry Project BETA De-escalation Workgroup. *West J Emerg Med*. 2012;13:17-25.

87. Fishkind A. Calming agitation with words, not drugs: 10 commandments for safety. *Curr Psyciatry*. 2002;1:32-39.

88. Watson AC, Compton MT, Draine JN. The crisis intervention team (CIT) model: An evidence-based policing practice? *Behav Sci Law*. 2017;35:431-441.

89. Cole R. Reducing restrain use in a trauma center emergency room. *Nurs Clin North Am*. 2014;49:371-381.

90. Creed JO, Cyr JM, Owino H, et al. Acute Crisis Care for Patients with Mental Health Crises: Initial Assessment of an Innovative Prehospital Alternative Destination Program in North Carolina. *Preshosp Emerg Care*. 2018;22:555-564.

91. Crowe R, Pepe P, Fernandez A, et al. "Comparison of benzodiazepines, ketamine, and antipsychotics for prehospital sedation of patients experiencing behavioral health emergencies with combativeness." Abstracts for the 2021 NAEMSP Scientific Assembly. *Prehospital Emergency Care*. 2021;25:125-170.

92. Miller M, Watanabe B, Brown L. "Are there gender or racial disparities in EMS-administered sedation among patients in police custody?" Abstracts for the 2021 NAEMSP Scientific Assembly. *Prehospital Emergency Care*. 2021;25:125-170.

93. Fernandez AR, Bourn SS, Crowe RP, et al. Out-of-Hospital Ketamine: Indications for Use, Patient Outcomes, and Associated Mortality [published online ahead of print, 2021 Jun 7]. *Ann Emerg Med*. 2021;S0196-0644(21)00152-9.

94. Centorrino F, Meyers AL, Ahl J, et al. An observational study of the effectiveness and safety of intramuscular olanzapine in the treatment of acute agitation in patients with bipolar mania or schizophrenia/schizoaffective disorder. *Hum Psychopharmacol*. 2007;22:455-462.

95. Currier GW, Simpson GM. Risperidone liquid concentrate and oral lorazepam versus intramuscular haloperidol and intramuscular lorazepam for treatment of psychotic agitation. *J Clin Psychiatry*. 2001;62:153-157.

96. Foster, S, Kessel J, Berman ME, et al. Efficacy of lorazepam and haloperidol for rapid tranquilization in a psychiatric emergency room setting. *Int Clin Psychopharmacol*. 1997;12:175-179.

97. Hovens JE, Dries PJT, Melman CTM, et al. Oral risperidone with lorazepam versus oral zuclopenthixol with lorazepam in the treatment of acute psychosis in emergency psychiatry: a prospective, comparative, open-label study. *J Psychopharmacol*. 2005;19:51-57.

98. Nobay F, Simon BC, Levitt A, et al. A prospective, double-blind, randomized trial of midazolam versus haloperidol versus lorazepam in the chemical restraint of violent and severely agitated patients. *Acad Emerg Med*. 2004;11:744-749.

99. Riddell J, Tran A, Bengiamin R, et al. Ketamine as a first-line treatment for severely agitated emergency department patients. *Am J Emerg Med*. 2017;35:1000-1004.

100. TREC Collaborative Group. Rapid tranquillisation for agitation patients in emergency psychiatric rooms: a randomized trial of midazolam versus haloperidol plus promethazine. *BMJ*. 2003;327:708-713.

101. Isbister GK, Calver LA, Page CB, et al. Randomized controlled trial of intramuscular droperidol versus midazolam for violence and acute behavioral disturbance: the DORM study. *Ann Emerg Med.* 2010;56:392-401.

102. Isenberg DL, Jacobs D. Prehospital Agitation and Sedation Trial (PhAST): A randomized control trial of intramuscular haloperidol versus intramuscular midazolam for the sedation of the agitated or violent patient in the prehospital environment. *Prehosp Disaster Med*. 2015;30:491-495.

103. Klein LR, Driver BE, Miner JR, et al. Intramuscular midazolam, olanzapine, ziprasidone, or haloperidol for treating acute agitation in the emergency department. *Ann Emerg Med*. 2018;72:374-385.

104. Page CB, Parker LE, Rashford SJ, et al. A Prospective Before and After Study of Droperidol for Prehospital Acute Behavioral Disturbance. *Prehosp Emerg Care*. 2018;22(6):713-721

105. Holland, D, Glober N, Christopher S, et al. Prehospital sedation with ketamine vs. midazolam: Repeat sedation, intubation, and hospital outcomes. *Am J Emerg Med*. 2020;38:1748-1753.

106. Chan, EW, Lao KS, Lam L, et al. Intramuscular midazolam, olanzapine, or haloperidol for the management of acute agitation: A multi-centre, double-blind, randomised clinical trial. EClinicalMedicine. 2021;32:100751. doi: 10.1016/j.eclinm.2021.100751.

107. Battaglia J, Moss S, Rush J, et al. Haloperidol, lorazepam, or both for psychotic agitation? A multicenter, prospective, double-blind, emergency department study. *Am J Emerg Med*. 1997;15:335-340.

108. Martel ML, Driver BE, Miner JR, et al. Randomized double-blind trial of intramuscular droperidol, ziprasidone, and lorazepam for acute undifferentiated agitation in the emergency department. *Acad Emerg Med*. 2021;28:421-434.

109. Resnick M, Burton BT. Droperidol vs. haloperidol in the initial management of acutely agitated patients. *J Clin Psychiatry*. 1984;45:298-299.

110. Thomas Jr H, Schwartz E, Petrilli R. Droperidol versus haloperidol for chemical restraint of agitated and combative patients. *Ann Emerg Med*. 1992;21:407-413.

111. Macht M, Mull AC, McVaney KE, et al. Comparison of droperidol and haloperidol for use by paramedics: assessment of safety and effectiveness. *Prehosp Emerg Care*. 2014;18(3):375-380.

112. Cole JB, Klein LR, Martel ML. Parenteral antipsychotic choice and its association with emergency department length of stay for acute agitation secondary to alcohol intoxication. *Acad Emerg Med*. 2019;26:79-84.

113. Klein LR, Driver BE, Horton G, et al. Rescue sedation when treating acute agitation in the emergency department with intramuscular antipsychotics. *J Emerg Med*. 2019;56:484-490.

114. Cole JB, Stang JL, DeVries PA, et al. A prospective study of intramuscular droperidol or olanzapine for acute agitation in the emergency department: a natural experiment owing to drug shortages. *Ann Emerg Med*. 2021; Article in press.

115. Calver L, Page CB, Downes MA, et al. The Safety and Effectiveness of Droperidol for Sedation of Acute Behavioral Disturbance in the Emergency Department. *Ann Emerg Med*. 2015;66:230-238.

116. Cole JB, Moore JC, Dolan B, et al. A prospective observational study of patients receiving intravenous and intramuscular olanzapine in the emergency department. *Ann Emerg Med*. 2017;69:327-336.

117. Asadollahi S, Heidari K, Hatamadadi H, et al. Efficacy and safety of valproic acid versus haloperidol in patients with acute agitation: results of a randomized, double-blind, parallel-group trial. *Int Clin Psychopharmacol*. 2015;30:142-150.

118. Cole JB, Moore JC, Nystrom PC, et al. A prospective study of ketamine versus haloperidol for severe prehospital agitation. *Clin Toxicol*. 2016;54:556-562.

119. Heydari F, Gholamian A, Zamani M, et al. Effect of intramuscular ketamine versus haloperidol on short-term control of severe agitated patients in emergency department; a randomized clinical trial. *Bull Emerg Trauma*. 2018;6:292-299.

120. Inapsine (droperidol) injection [package insert]. Decatur, IL. Taylor Pharmaceuticals.; 2001.

121. Cole JB, Lee SC, Martel ML, et al. The incidence of QT prolongation and Torsades des Pointes in patients receiving droperidol in an urban emergency department. *West J Emerg Med*. 2020;21:728-736.

122. Gaw CM, Cabrera D, Bellolio F, et al. Effectiveness and safety of droperidol in a United States emergency department. *Am J Emerg Med*. 2020;38:1310-1314.

123. Martel ML, Klein LR, Rivard RL, et al. A large retrospective cohort of patients receiving intravenous olanzapine in the emergency department. *Acad Emerg Med*. 2016;23:29-35.

124. Knott JC, Taylor D McD, Castle DJ. Randomized clinical trial comparing intravenous midazolam and droperidol for sedation of the acutely agitated patient in the emergency department. *Ann Emerg Med*. 2006;47:61-67.

125. Taylor D McD, Yap CYL, Knott JC, et al. Midazolam-Droperidol, Droperidol, or Olanzapine for Acute Agitation: A Randomized Clinical Trial. *Ann Emerg Med*. 2017;69:318-326.

126. Lin J, Figuerado Y, Montgomery A, et al. Efficacy of ketamine for initial control of acute agitation in the emergency department: A randomized study. *Am J Emerg Med*. 2020;S0735-6757:30241-30242.

127. McCarthy DA, Chen G, Kaump DH, et al. General anesthetic and other pharmacological properties of 2-(O-chlorophenyl)-2-methylaminocyclohexanone HCL (CI-58L). *J New Drugs*. 1965;5:21-23.

128. Ketamine Hydrochloride. In: MICROMEDEX [database on the Internet]. Greenwood Village (CO): Thompson Micromedex; 1974-2020 [cited 2020 Dec 18].

129. Ketamine Hydrochloride. In: Lexi-Comp [database on the Internet]. Hudson (OH): Lexicomp, Inc.; 2020 [cited 2020 Dec 18].

130. Domino EF, Chadoff P, Corssen G. Pharmacologic effects of CI-581, a new dissociative anesthetic, in man. *Clin Pharmacol Ther*. 1965;6:279-291.

131. Corssen G, Domino EF. Dissociative anesthesia: further pharmacologic studies and first clinical experience with the phencyclidine derivative CI-581. *Anesth Analg*. 1966;45:29-40.

132. Corssen G, Miyasaka M, Domino EF. Changing concepts in pain control during surgery: dissociative anesthesia with CI-581. A progress report. *Anesth Analg*. 1968;47:746-59.

133. Green SM, Krauss B. The semantics of ketamine. *Ann Emerg Med*. 2000;36:480-2.

134. Dillingham CM, Milczarek MM, Perry JC, Vann SD. Time to put the mammillothalamic pathway into context. *Neurosci Biobehav Rev*. 2020;121:60-74.

135. Baumann O, McFadyen J, Humphreys MS. Behavioral and Neural Effects of Familiarization on Object-Background Associations. *Front Psychol*. 2020;11:591231. doi: 10.3389/fpsyg.2020.591231.

136. Corssen G, Bjarnesen W. Recent advances in intravenous anesthesia. *J Am Assoc Nurse Anesth*. 1966;34:416-427.

137. Corssen G, Hayward JR, Gunter JW, et al A new parenteral anesthesia for oral surgery. *J Oral Surg*. 1969;27:627-632.

138. Green SM, Nakamura R, Johnson NE. Ketamine sedation for pediatric procedures: Part 1, A prospective series. *Ann Emerg Med*. 1990;19:1024-1032.

139. Burnett AM, Salzman JG, Griffith KR, et al. The emergency department experience with prehospital ketamine: a case series of 13 patients. *Prehosp Emerg Care*. 2012;16:553-559.

140. Ho JD, Smith SW, Nystrom PC, et al. Successful management of excited delirium syndrome with prehospital ketamine: two case examples. *Prehosp Emerg Care*. 2013;17:274-279.

141. Scheppke KA, Braghiroli J, Shalaby M, et al. Prehospital use of i.m. ketamine for sedation of violent and agitated patients. *West J Emerg Med*. 2014;15:736-741.

142. Keseg D, Cortez E, Rund D, et al. The Use of Prehospital Ketamine for Control of Agitation in a Metropolitan Firefighter-based EMS System. *Prehosp Emerg Care*. 2015;19:110-115.

143. Olives TD, Nystrom PC, Cole JB, et al. Intubation of Profoundly Agitated Patients Treated with Prehospital Ketamine. *Prehosp Disaster Med*. 2016;31:593-602.

144. Cole JB, Klein LR, Nystrom PC, et al. A prospective study of ketamine as primary therapy for prehospital profound agitation. *Am J Emerg Med*. 2018;36:789-796.

145. Mankowitz SL, Regenberg P, Kaldan J, et al. Ketamine for Rapid Sedation of Agitated Patients in the Prehospital and Emergency Department Settings: A Systematic Review and Proportional Meta-Analysis. *J Emerg Med*. 2018;55:670-681.

146. O'Connor L, Rebesco M, Robinson C, et al. Outcomes of Prehospital Chemical Sedation With Ketamine Versus Haloperidol and Benzodiazepine or Physical Restraint Only. *Prehosp Emerg Care*. 2019;23:201-209.

147. Li M, Martinelli AN, Oliver WD, et al. Evaluation of Ketamine for Excited Delirium Syndrome in the Adult Emergency Department. *J Emerg Med*. 2019; S0736-4679(19)30802-9. doi: 10.1016/j.jemermed.2019.09.019.

148. Lebin JA, Akhavan A, Hippe DS, et al. Psychiatric outcomes of patients with severe agitation following administration of prehospital ketamine. *Acad Emerg Med*. 2019;26:889-896.

149. Stanley V, Hunt J, Willis KW, Stephen CR. Cardiovascular and respiratory function with CI-581. *Anesth Analg*. 1968;47:760-768.

150. Morgan M, Loh L, Singer L, Moore PH. Ketamine as the sole anaesthetic agent for minor surgical procedures. *Anaesthesia*. 1971;26:158-159.

151. Sussman DR. A comparative evaluation of ketamine anesthesia in children and adults. *Anesthesiology*. 1974;40:459-464.

152. Lohit K, Srinvas V, Kulkarni C, Shaheen. A clinical evaluation of the effects of administration of midazolam on ketamine-induced emergence phenomenon. *J Clin Diag Res*. 2011;5:320-323.

153. Green SM, Rothrock SG, Lynch EL, et al. Intramuscular ketamine for pediatric sedation in the emergency department: safety profile in 1,022 cases. *Ann Emerg Med*. 1998;31:688-697.

154. Cole JB, Driver BE, Klein LR, et al. In reply: Ketamine is an important therapy for prehospital agitation - Its exact role and side effect profile are still undefined. *Am J Emerg Med*. 2018;36:502-503.

155. Parsch C, Boonstra A, Teubner D, et al. Ketamine reduces the need for intubation in patients with acute severe mental illness and agitation requiring transport to definitive care: An observational study. *Emerg Med Australas*. 2017;29:291-296.

156. Parks DJ, Alter SM, Shih RD, Solano JJ, Hughes PG, Clayton LM. Rescue Intubation in the Emergency Department After Prehospital Ketamine Administration for Agitation. *Prehosp Disaster Med*. 2020;35:651-655.

157. Cunningham C, Gross K, Broach JP, et al. Patient outcomes following ketamine administration for acute agitation with a decreased dosing protocol in the preshospital setting. Prehosp Disaster Med. 2021;36:276-282.

158. Zsigmond EK, Matsuki A, Kothary SP, et al. Arterial hypoxemia caused by intravenous ketamine. *Anesth Analg*. 1976;55:311-314.

159. Pederson L, Benumof J. Incidence and magnitude of hypoxaemia with ketamine in a rural African hospital. *Anaesthesia*. 1993;48:67-69.

160. Streatfeild KA, Gebremeskel A. Arterial oxygen saturation in Addis Ababa during diazepam-ketamine anaesthesia. *Ethiop Med J*. 1999;37:255-261.

161. Bredmose PP, Lockey DJ, Grier G, et al. Pre-hospital use of ketamine for analgesia and procedural sedation. *Emerg Med J*. 2009;26:62-64.

162. Bredmose PP, Grier G, Davies GE, et al. Pre-hospital use of ketamine in paediatric trauma. *Acta Anaesthesiol Scand*. 2009;53:543-545.

163. Hopper AB, Vilke GM, Castillo EM, et al. Ketamine use for acute agitation in the emergency department. *J Emerg Med*. 2015;48:712-9.

164. Burnett AM, Peterson BK, Stellpflug SJ, et al. The association between ketamine given for prehospital chemical restraint with intubation and hospital admission. *Am J Emerg Med*. 2015;33:76-79.

165. Le Cong M, Humble I. A ketamine protocol and intubation rates for psychiatric air medical retrieval. *Air Med J*. 2015;34:357-359.

Appendix A. Conflict of interest disclosures for Hyperactive Delirium Task Force

Questions asked of participants:

- Employment: Please indicate the name of your employer and describe your position of employment, including the nature of the business of your employer, the position you hold and a description of your daily employment responsibilities.
- Leadership: Do you hold any positions of leadership in other organizations, chapters, commissions, groups, coalitions, agencies, and/or entities (e.g. board of director positions, committees and/or spokesperson roles)? If yes, please describe the position you hold, including a brief description of the nature and purposes of the organization or entity.
- Relationships: To the best of your knowledge, do you have any outside relationships with any person or entity from which ACEP obtains goods and services, or which provides services that compete with ACEP where such relationship involves: a) holding a position of responsibility; b) an equity interest (other than a less than 1% interest in a publicly traded company); c) any gift, gratuities, lodging, dining, or entertainment valued at more than $100? If yes, please explain:
- Financial interests: Do you have any financial interests or positions of responsibility in entities providing goods or services in support of the practice of emergency medicine (e.g. physician practice management company, billing company, physician placement company, book publisher, medical supply company, and/or a malpractice insurance company), other than owning less than a 1% interest in a publicly traded company? If yes, please explain.
- Other potential conflict: Do you have any other interest that may create a conflict with your fiduciary duty to ACEP or that may create the appearance of a conflict of interest?
- Health administration: Do you have any outside relationships with any health plan, health insurance company, delegated payer, health insurance company administrative service organization, or health insurance company related philanthropic organization or entity where such relationship involves: a) holding any position of responsibility; b) an equity interest (other than a less than 1% interest in a publicly traded company); c) any stipend, contribution, gift, gratuities, lodging, dining, or entertainment valued at more than $100?

Benjamin W. Hatten, MD, MPH

- Employment: Assistant Professor, University of Colorado School of Medicine.
- Leadership: None.
- Relationships: None.
- Financial interests: None.
- Other potential conflict: None.
- Health administration: None.

Caitlin Bonney, MD

- Employment: Attending Emergency Physician, Maine Medical Center; Attending Medical Toxicologist, Northern New England Poison Center.
- Leadership: None.
- Relationships: None.
- Financial interests: None.

- Other potential conflict: None.
- Health administration: None.

Robert B. Dunne, MD
- Employment: Professor, EMS Fellowship Director, Team Health; Emergency Medicine Physician, St. John Hospital, Detroit, Michigan.
- Leadership: None.
- Relationships: None.
- Financial interests: None.
- Other potential conflict: None.
- Health administration: None.

Stacey L. Hail, MD
- Employment: Associate Professor of Emergency Medicine and Medical Toxicology, University of Texas Southwestern Medical Center; Attending Physician, Parkland Hospital, Dallas, Texas; Attending Physician, Medical Toxicology, North Texas Poison Control Center, Dallas, Texas.
- Leadership: None.
- Relationships: None.
- Financial interests: None.
- Other potential conflict: None.
- Health administration: None.

Graham S. Ingalsbe, MD
- Employment: Assistant Professor of Clinical Emergency Medicine, University of Nevada-Reno School of Medicine.
- Leadership: Treasurer, Nevada Chapter of the American College of Emergency Medicine.
- Relationships: None.
- Financial interests: None.
- Other potential conflict: None.
- Health administration: None.

Michael K. Levy, MD
- Employment: Vice President, Staff Emergency Physician, Denali Emergency Medicine Physicians, Anchorage, Alaska; EMS Medical Director, State of Alaska; EMS Medical Director, Anchorage Fire Department; EMS Medical Director, Kenai Peninsula.
- Leadership: President-Elect, National Association of EMS Physicians.
- Relationships: None.
- Financial interests: Chief Medical Advisor for Stryker Emergency Care; paid for consultations services but does not have stock or other financial interests.
- Other potential conflict: None.
- Health administration: None.

Michael Millin, MD, MPH

- Employment: Faculty, Department of Emergency Medicine, Johns Hopkins University School of Medicine; Medical Director, Prince George Fire/EMS Department, District Heights, Maryland; Medical Director, Maryland and Mid-Atlantic Wilderness Rescue Squad/Austere Medical Professionals.
- Leadership: Resuscitation Sub-Council Vice-Chair, American Red Cross Scientific Advisory Council.
- Relationships: None.
- Financial interests: None.
- Other potential conflict: None.
- Health administration: None.

Brent J. Myers, MD

- Employment: Chief Medical Officer, ESO solutions Inc., a data and software company serving EMS, hospitals, and fire departments.
- Leadership: Chair, Advocacy Committee, National Association of EMS Physicians.
- Relationships: None.
- Financial interests: None.
- Other potential conflict: None.
- Health administration: None.

Richard D. Shih, MD

- Employment: Professor of Integrated Medical Science, Division Director for the Emergency Medicine Residency Program, Florida Atlantic University College of Medicine.
- Leadership: None.
- Relationships: None.
- Financial interests: None.
- Other potential conflict: None.
- Health administration: None.

Jeffrey M. Goodloe, MD

- Employment: Professor of Emergency Medicine, University of Oklahoma School of Community Medicine.
- Leadership: None.
- Relationships: None.
- Financial interests: None.
- Other potential conflict: None.
- Health administration: None.

Appendix B. (Studies examining IM treatment of acute agitation with sedating medications in EMS or ED patients with sedation outcomes recorded by individual study arm)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Midazolam					

TREC Collaborative Group[100] (2003)	Midazolam 7.5 mg to 15 mg IM
	• prospective, pseudo-randomized open label
	• dose at treating physician discretion
	
	Compared to:
	• combination of haloperidol 5 mg to 10 mg plus promethazine 25 mg to 50 mg IM
		Adults presenting to psychiatric EDs with agitation or dangerous behavior
		
		150 patients in the Midazolam arm
		• 48% male/52% female; mean age: 38 years;
		• dose: 15 mg (124 patients)/7.5 mg (26 patients):
		• presumed etiology:
		  • psychosis 71%
		  • substance abuse 20%, other 9%
			Primary endpoint was "tranquil or asleep" at 20 minutes, with tranquil defined as peaceful and without restlessness or threatening behavior; secondary endpoints included tranquil or asleep at 40, 60, and 120 minutes; need for physical restraints; recurrent episode of agitation; major adverse events; midazolam superior for primary endpoint at 20 minutes as well as secondary endpoint at 40 minutes; no difference at 60 minutes or greater; no difference in need for restraints; no difference in additional tranquilizing drugs	At 20 minutes, 89% in the midazolam arm versus 67% in the haloperidol/promethazine arm reached study endpoint
				• relative risk 1.32 (95% CI 1.16 to 1.49)
				• 22% (95% CI 12% to 30%) more in midazolam arm w/ adequate sedation at 20 minutes
					1 patient in midazolam group experienced respiratory depression that resolved with flumazenil

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Midazolam					
Nobay et al[98] (2004)	Midazolam 5 mg IM • randomized and double blind • if a patient continued to be disruptive 20 minutes after the study drug was administered, a ‘‘rescue drug’’ could be given at the discretion of the treating attending physician. Patient enrollment in the study was terminated if a rescue medication was given; these patients were considered sedation failures, and their data were not included in the analysis Compared to: • lorazepam 2 mg IM • haloperidol 5 mg IM	ED patients who required emergency sedation for the control of violent behavior or severe agitation; all patients were initially physically restrained; 42 patients in the midazolam group; • mean age 39.8 • 23 African American, 1 Asian, 2 Hispanic, and 16 White • 8 with recreational drug use, 6 without, and 28 unknown • 13 with alcohol use, 2 without, and 27 unknown • 20 with prior psychiatric history, 3 without, and 19 unknown	Level of sedation was continuously observed with data collected every 15 minutes; adequacy of sedation was assessed using the Modified Thomas Combativeness Scale with the goal endpoint a score of 3 (No agitation, no supervision required, maybe asleep); 7 midazolam patients (17%) needed rescue drugs; midazolam reached adequate sedation 13.9 minutes faster than lorazepam (95% CI 5.1 to 22.8; p=0.0026); midazolam reached adequate sedation 9.9 minutes faster than haloperidol (95% CI 0.5 to 19.3; p=0.0388)	The mean time to sedation • midazolam 5 mg IM: 18.3 minutes	There were no statistically significant differences over time in regard to change in systolic and diastolic blood pressure (p=0.8965, p=0.9581), heart rate (p=0.5517), respiratory rate (p=0.8191), and oxygen saturation (p=0.8991) among patients receiving each of the medications; there were no adverse events in the midazolam group

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Midazolam					
Martel et al[16] (2005)	Midazolam 5 mg IM; prospective, randomized, double-blind trial; rescue sedation at treating physician discretion permitted 30 minutes after study drug administration for AMS >0 Compared to: • ziprasidone 20 mg IM • droperidol 5 mg IM	ED patients with acute undifferentiated agitation requiring emergent sedation as determined by the treating physician; 48 patients in midazolam group; mean age 36.9; 33 male/15 female; initial mean AMS scale score of 3.10; initial assessment of reason for agitation: alcohol intoxication (46), illicit substance intoxication (8), head injury (14), psychiatric etiology (4), and seizure (1); discharge diagnoses: acute alcohol intoxication (46), acute drug intoxication (4), and closed head injury (18)	AMS scale score was obtained every 15 minutes from time 0 to 120 minutes following study medication administration with effective sedation defined as an AMS of 0 or less Mean AMS scale scores in the midazolam group: • at 15 minutes -0.81 (95% CI -1.54 to -0.08), • at 30 minutes -1.46 (95% CI -2.19 to -0.73), • at 45 minutes -1.31 (95% CI -2.02 to -0.60), • at 60 minutes -1.13 (-1.86 to -0.38) More patients receiving midazolam or ziprasidone required rescue medications at 30 minutes compared to droperidol ($p<0.05$) • droperidol: 5 patients required 6 doses • ziprasidone: 9 patients requiring 11 doses • midazolam: 24 patients requiring 30 doses	Less patients remained agitated at 15 minutes in the droperidol and midazolam groups compared to the ziprasidone group ($p=0.01$) • droperidol: 20/50 • midazolam: 15/48 • ziprasidone: 28/46 There was no difference between groups at 30 minutes ($p=0.08$). • droperidol: 6/50 • midazolam: 11/48 • ziprasidone: 14/46 More patients were agitated at 45 minutes in the midazolam group compared to the droperidol and ziprasidone groups ($p=0.03$) • droperidol: 9/50 • midazolam: 14/48 • ziprasidone: 9/46	Respiratory depression: • 24/48 patients who received midazolam • 10 required supplemental oxygen • no difference in proportion with respiratory depression ($p=0.26$) or supplemental oxygen ($p=0.20$) when compared to ziprasidone and droperidol • no patients required intubation for respiratory depression Akathisia: • 1/48 patients who received ziprasidone Cardiac dysrhythmias: • none

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Midazolam					
Isbister et al[101] (2010)	Midazolam 10 mg IM • blinded, randomized controlled trial • further sedation allowed at discretion of attending physician Compared to: • droperidol 10 mg IM • midazolam 5 mg plus droperidol 5 mg IM	ED patients requiring physical restraint and parenteral sedation 29 patients in midazolam group • median age: 35 • 18 male/11 female • initial assessment of agitation due to: alcohol intoxication (22), self-harm (12), drug-induced delirium (3), and acute psychosis (1)	Primary sedation outcome was time security staff were required according to a security log from the time of initial call to the "all clear" • duration was not different between groups (p=0.66) with median for: midazolam (20 minutes), droperidol (24 minutes), and midazolam plus droperidol (25 minutes) Secondary sedation outcomes were: • time additional sedation was administered: the hazard ratio for additional sedation medications for midazolam versus droperidol was 2.31 (95% CI 1.01 to 4.71; post prob 0.98 for HR>1.0) indicating that midazolam was more likely to require additional sedation compared to droperidol	Secondary outcome of reduction in AMSS by 3 points or to a score of <1 20 minutes after drug administration: • midazolam: 15/29	Respiratory events occurred in: • midazolam: 8/29 patients involving desaturation events (7) and airway obstruction (2) Hypotension occurred in: • midazolam: 1/29 Abnormal QT-HR pairs occurred in: • midazolam: 2/29 No dystonic reactions were identified Although oversedation was not a secondary endpoint, AMSS scores revealed that both midazolam and midazolam plus droperidol resulted in unpredictable and oftentimes deep sedation while droperidol resulted in consistent moderate sedation

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Midazolam					
Isenberg et al[102] (2015)	Midazolam 2.5 mg to 5 mg IM (5 mg if younger than 65 years and 2.5 mg if 65 years or older) • redosing available every 10 minutes if sedation endpoint not met but maximum dose received was 5 mg. • randomized, non-blinded Compared to haloperidol 2.5 mg to 5 mg IM	EMS patients with either: • a psychiatric or behavioral disorder who is at imminent risk of self-injury or is a threat to others • patient with a medical condition causing agitation and possibly violent behavior 5 patients in midazolam group • age: 26 to 90 years • all with initial RASS +4 • patient diagnosis: sepsis, urinary tract infection, alcohol intoxication, hypoglycemia, and acute renal failure	Sedation evaluated using RASS with goal of less than +1. 4/5 patients in midazolam group with RASS<1 on arrival to ED	Mean time to achieve a RASS of less than +1 • midazolam 2.5 mg to 5 mg IM: 13.5 minutes	No patients in the midazolam group had any adverse effects

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Midazolam					
Riddell et al[99] (2017)	Midazolam (mean dose 3.08 mg) IV/(mean dose 2.25 mg) IM/(mean dose 2 mg) IN • prospective, observational Compared to: • lorazepam (mean dose 1.9 mg) IV/ (mean dose 2.4 mg) IM • haloperidol (mean dose 5.71 mg) IM • combination of lorazepam (mean dose 2 mg) IV/(mean dose 2 mg) IM plus haloperidol (mean dose 5 mg) IM • ketamine (mean dose 0.87 mg/kg) IV/(mean dose 2.97 mg/kg) IM	Acutely agitated patients requiring chemical sedation in the ED 19 patients in the midazolam group • median age: 43 years • 18 male/1 female • race: African American (1)/Asian (0)/Hispanic (10)/White (7) • drug use: 63.2% • alcohol use: yes (42.1%)/no (36.8%)/unknown (21.1%) • prior psychiatric visits (36.8%) • route of administration: IV(12)/IM(4)/IN(3)	Primary outcome: agitation score less than or equal to 2 on a six-point agitation scale • recorded prior to medication administration then at 5, 10, and 15 minutes • midazolam (and other arms) inferior to ketamine at: 5 minutes (p=0.001), 10 minutes (p<0.001), and 15 minutes (p=0.032) Secondary outcomes of: • provider assessment of time to adequate sedation: No difference between groups (p=0.107) • need for redosing of sedative medications (p=0.199) • HR/SBP change: HR reduction seen with midazolam (p=0.026) but no other significant HR/SBP changes in any other study arms	Mean time to adequate sedation: • midazolam: 14.95 minutes	Intubation: • midazolam: 1/19 • lorazapam: 1/33 • haloperidol: 1/14 • combination lorazepam plus haloperidol: 1/10 • ketamine: 2/24

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Midazolam					
Klein et al[103] (2018)	Midazolam 5 mg IM • prospective, observational Compared to: • olanzapine 10 mg IM • haloperidol 5 mg IM • haloperidol 10 mg IM • ziprasidone 20 mg IM	ED patients receiving medication to treat acute agitation 127 patients in midazolam arm • median age: 40 • 97 male/30 female • cause of agitation: alcohol (82%)/illicit substance (17%)/psychiatric illness (17%)/medical (1%)	Primary endpoint was adequate sedation, defined as Altered Mental Status Score <1 15 minutes after medication administration • midazolam 5 mg IM not superior to olanzapine 10 mg IM (9% greater for midazolam: 95% CI 1% lesser to 20% greater) • midazolam 5 mg IM superior to haloperidol 5 mg IM (30% greater for midazolam: 95% CI 19% to 41%) • midazolam 5 mg IM superior to haloperidol 10 mg IM (28% greater for midazolam: 95% CI 17% to 39%) • midazolam 5 mg IM superior to ziprasidone 20 mg IM (18% greater for midazolam: 95% CI 6 to 29%) Median difference in AMSS score compared to baseline at 15 minutes: • midazolam 5 mg IM not superior to olanzapine 10mg IM (1 point greater decrease for midazolam: 95% CI 1 to 0 point greater decrease) • midazolam 5 mg IM superior to haloperidol 5mg IM (2 point greater decrease for midazolam: 95% CI 2.5 to 1.5 point greater decrease) • midazolam 5 mg IM superior to haloperidol 10mg IM (2 point greater decrease for	Median time to adequate sedation: • midazolam 5 mg IM: 12 minutes	No difference in adverse events between groups Respiratory distress: • 1 to 3 patients in each arm with hypoxemia • 1 patient intubated in each arm except haloperidol 10 mg with no intubations Cardiovascular: • 1 to 2 patients in each group with hypotension except ziprasidone with no episodes of hypotension • 1 patient in each arm with bradycardia except midazolam with no episodes of bradycardia • no patients in any arm with torsades de pointes or other dysrhythmias Extrapyrimadal symptoms: • 2 patients in haloperidol 10 mg arm with dystonia. No other dystonic reactions in any arm • no episodes of akathisia in entire study

			midazolam: 95% CI 2.5 to 1.5 point greater decrease) • midazolam 5 mg IM superior to ziprasidone 20 mg IM (1 point greater decrease for midazolam: 95% CI 1.5 greater decrease to 0.5 lesser decrease) Time to adequate sedation (compared to midazolam 5 mg IM): • olanzapine 10 mg IM no different (HR 0.97 95% CI 0.76 to 1.22) • haloperidol 5 mg IM inferior (HR 0.73 95% CI 0.58 to 0.90) • haloperidol 10 mg IM inferior (HR 0.72 95% CI 0.57 to 0.88) • ziprasidone 20 mg IM inferior (HR 0.78 95% CI 0.61 to 0.93) Time to adequate sedation for subset receiving monotherapy and no rescue sedation medications (compared to midazolam 5 mg IM): • olanzapine 10 mg IM no different (HR 0.84 95% CI 0.65 to 1.07) • haloperidol 5 mg IM inferior (HR 0.63 95% CI 0.48 to 0.81) • haloperidol 10 mg IM inferior (HR 0.59 95% CI 0.46 to 0.78) • ziprasidone 20 mg IM inferior (HR 0.64 95% CI 0.48 to 0.82)		

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Midazolam					
Page et al[104] (2018)	Midazolam IM (38%), IM/IV (29%), and IV (33%) • per protocol: 5 mg initial IM dose with repeat doses of 5 mg to10 mg every 10 minutes or 2.5 mg to 5 mg IV with repeat doses of 2.5 mg to 5 mg every 10 minutes • median dose received in study: 7 mg Prospective before/after protocol change observational study with primary endpoint to compare adverse events and secondary endpoints of sedation outcomes Compared to: • droperidol 10 mg IM • optional redosing of 10 mg at 15 minutes	141 EMS patients with acute behavioral disturbance and SAT score of +2 (34 patients) to +3 (103 patients). • 86 male/55 female • reason for agitation: alcohol (55), amphetamines (39), medical (23), mental illness (9), other stimulants (11), self-harm (21), and marijuana (3) • police were on scene for 110 encounters • median prehospital time of 47 minutes	Sedation was defined as a decrease in SAT score by at least 2 points or score of 0; successful sedation was defined as sedated, no adverse effects, and no requirement for additional sedation • 20/141 required additional EMS sedation • 59/141 required additional ED sedation • median number of drug administrations was 2 • 50/141 were successfully sedated • 91 with unsuccessful sedation due to: failed to sedate prehospital (17), adverse effects (33), EMS additional sedation (20), and ED additional sedation (59) 123/149 were successfully sedated in droperidol group	Median time to sedation: • midazolam 5 mg IM: 30 minutes	33/141 patients exhibited 49 adverse events in the midazolam group. • airway obstruction requiring airway maneuver (24: 19 chin lift/jaw thrust, 3 oropharyngeal airway (OPA)/ nasopharyngeal airway (NPA) placement, and 2 intubation), hypotension (9), and SAT score of -3 (7) • compared to those receiving droperidol, a 16% greater proportion in the midazolam group exhibited adverse events (p=0.0001, 95% CI 8% to 24%)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Midazolam					
Lebin et al[148] (2019)	Midazolam 1 mg to 10 mg IV, 5 mg to 10 mg IM, or 2.5 mg to 10 mg IN • alternative benzodiazepine: diazepam 2.5 mg to 10 mg IV (3 patients) • retrospective cohort study Compared to: • ketamine 1 mg to 2 mg/kg IV or 3 mg to 5 mg/kg IM	Patients with severe agitation requiring prehospital sedation with ketamine or benzodiazepine 82 patients in benzodiazepine group • age: 32 years • male (92.7%) • Caucasian (54.9%)/Black or African American (0%)/Asian (6.1%)/other or not reported (39.0%) • 16 patients received midazolam IM	Sedation endpoint was not studied	Not reported	Intubation • benzodiazepine (63.0%) • ketamine (3.8%) • 59.1% (95% CI 37.9% to 79.35%) more likely to be intubated after benzodiazepine administration than ketamine administration

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Midazolam					
Holland et al[105] (2020)	Midazolam 5 mg IV/IM/IN • dose per protocol: 2.5 mg to 5 mg (61/66 patients received 5 mg) • route: IM (32/66), IV (24/66), and IN (10/66) • retrospective chart review Compared to: • ketamine (mean dose 3.75 mg/kg) IM	Patients with acute agitation requiring sedation by paramedics 66 patients in midazolam treated group • mean age of 36.1 years • 41 male/25 female • race: white (32), African-American (29), and other (5) • mean weight: 79.1 kg • suspicion of illicit drugs: 71.2% • IM dosing: 32/66 (48.5%)	Primary endpoint was need for repeat sedative dose. • 7/66 required repeat sedation at 20 minutes. No difference compared to ketamine (p=0.306) • 18/66 required repeat sedation at 90 minutes. Significantly less than ketamine group (p=0.01) • when limiting the analysis to only sedation given via IM route, there was no difference in need for repeat sedation between midazolam and ketamine groups at 20 minutes (p=0.212) or 90 minutes (p=0.503) Secondary endpoints • time to repeat sedation of 77.2 minutes. No difference compared to ketamine group (p=0.658) • total number of sedation doses did not differ between ketamine and midazolam (p=0.084)	Need for repeat sedative dose at 20 minutes used as proxy for adequate control of agitation • 7/66 in midazolam group required repeat sedation	5 patients in the midazolam group were intubated. • 1 patient was found to have a traumatic intracranial hemorrhage • 1 received repeat sedation (midazolam) before intubation • 3 (4.6%) were intubated within an hour of ED arrival for altered mental status without further complicating factors or further sedative administration For patients administered midazolam, median GCS was 14 (IQR 13 to 15) prior to administration and 12 (IQR 6.5 to 15) after administration (p<0.0001) with a mean difference of 4.5, 95% CI 3.4 to 5.6). There was no significant difference compared to the change in GCS achieved with ketamine, p=0.4116). There were no significant differences in use of bag valve mask or intubation, use of physical restraints, admission location/level of care, or length of stay in the ED, hospital, or ICU

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Midazolam					
Chan et al[106] (2021)	Midazolam 5 mg IM • single optional redose allowed per study protocol • randomized, double-blind Compared to: • olanzapine 5 mg IM • haloperidol 5 mg IM	ED patients requiring parenteral drug sedation for acute agitation 56 patients in midazolam group • mean age 44 • 34 male/22 female • perceived possible causes: drug/substance abuse (16), alcohol intoxication (15), underlying mental illness (47), medication non-compliance (24), suicidal ideation/attempt (18), exposure to tramadol (1), concurrent psychotropic medication (19) • baseline sedation scores: 3 (13 patients), 4 (17 patients), and 5 (26 patients) 18 patients in the midazolam group received a second dose of study drug or alternative sedatives	Agitation/sedation level was measured on a 6-point validated sedation scale: (5=highly aroused, violent; 4=highly aroused, possibly distressed, or fearful; 3=moderately aroused, unreasonable, or hostile; 2=mildly aroused, willing to talk reasonably; 1=minimal agitation; and 0=asleep). Adequate sedation was defined as a score of 2 or less Sedation scores were recorded at baseline, at first observed adequate sedation, and at 10, 20, 30, 45, and 60 minutes after the first dose regardless of observed time to sedation • midazolam was superior with significant differences detected in the Kaplan-Meier curves compared with olanzapine (p=0.03) and haloperidol (p=0.002) At 10 minutes after the initial dose, 52% in the midazolam group were adequately sedated At 60 minutes, the proportion of patients adequately sedated increased to 98% Fully adjusted accelerated factors for olanzapine and haloperidol were compared with midazolam at 1.72 (95% CI 1.16 to 2.55) and 1.89 (95% CI 1.28 to 2.80), respectively, indicating significantly faster sedation for midazolam	Median time to sedation: • midazolam 5 mg IM: 8.5 minutes	2 patients in the midazolam group experienced an adverse event, both with oxygen desaturation 28 patients receiving midazolam fell asleep after treatment

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Lorazepam					
Foster et al[96] (1997)	Lorazepam 2 mg oral concentrate or IM • randomized and double blind • redoses allowed every 30 minutes up to 4 hours until sedated or no longer a danger to self or others Compared to • haloperidol 5 mg oral concentrate or IM	Patients presenting at the psychiatric emergency service of a large urban hospital judged by emergency room staff to be an imminent danger to themselves, they required 4-point physical restraints, they scored a 5 or higher on at least 3 items on the Brief Psychiatric Rating Scale, and they had a score of at least 4 on the GCI Scale 17 patients in the lorazepam group. • mean age 41.35 years • 12 male and 5 female • final diagnoses of schizophrenia (5), bipolar (10), schizoaffective (1), and psychotic disorder not otherwise specified (1) • 6 patients with drug abuse or dependence by history	The primary endpoint was reduction in the Brief Psychiatric Rating Scale with a secondary endpoint of reduction in the GCI Scale The lorazepam group exhibited significant decreases in both rating scales over the course of the study, although no drug by time interactions were found. Analysis of route of administration did not reveal significant effects Brief Psychiatric Rating Scale reductions were not different for lorazepam and haloperidol at 1 hour; the lorazepam group exhibited a significantly greater reduction compared to the haloperidol group on the GCI Scale at 1 hour	Serial hourly evaluations were performed by trained evaluators; only 1-hour outcomes are relevant for this review	There were no group differences in HR/SBP pressure, and diastolic blood pressure and all parameters significantly decreased across time

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Lorazepam					
Battaglia et al[107] (1997)	Lorazepam 2 mg IM • randomized and double blind • repeat doses allowed but not until after the first post-treatment standardized evaluation at 1 hour Compared to • haloperidol 5 mg IM • lorazepam 2 mg plus haloperidol 5 mg IM	ED patients with psychosis and behavioral dyscontrol (agitated, aggressive, destructive, assaultive, or restless behavior) to the extent that they were capable of harming themselves or others 31 patients in lorazepam group: • 23 male/8 female • mean age 33.9 years – mean weight 74.4 kg • final diagnoses were mania, psychoactive substance abuse, psychosis not otherwise specified, schizophrenia, and schizophreniform disorder	Agitation was assessed serially using the Agitated Behavior Scale with a significant reduction in agitation from baseline at 1 hour in the lorazepam arm; however, greater reduction in agitation was seen with combination therapy compared to lorazepam alone (p=0.014); haloperidol alone was not different than lorazepam alone (p=0.426) Approximately 10% of patients in the lorazepam group were asleep at 1 hour, significantly more than the haloperidol alone group and similar to the combination therapy group	Serial evaluations occurred for 12 hours with redosing allowed after reevaluations; only 1-hour endpoints were abstracted as they are most relevant to this review	11 lorazepam-treated patients (35%) reported adverse effects: • ataxia: 2 (6%) • dizziness: 3 (10%) • dry mouth: 5 (16%) • EPS symptoms: 1 (3%) • speech disorder: 2 (6%) "No serious side effects" were reported.

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Lorazepam					
Nobay et al[98] (2004)	Lorazepam 2 mg IM • randomized and double blind • if a patient continued to be disruptive 20 minutes after the study drug was administered, a ‘‘rescue drug’’ could be given at the discretion of the treating attending physician. Patient enrollment in the study was terminated if a rescue medication was given. These patients were considered sedation failures, and their data were not included in the analysis Compared to: • midazolam 5 mg IM • haloperidol 5 mg IM	ED patients who required emergency sedation for the control of violent behavior or severe agitation. All patients were initially physically restrained 27 patients in the lorazepam group • mean age: 39.5 years • 13 African American, 1 Asian, 3 Hispanic, and 10 White • 10 with recreational drug use, 2 without, and 15 unknown • 8 with alcohol use, 3 without, and 16 unknown • 14 with prior psychiatric history, 1 without, and 12 unknown An interim analysis showed that lorazepam demonstrated a statistically significant longer time to sedation and time to awakening than midazolam. Therefore, the lorazepam arm was terminated early	Level of sedation was continuously observed with data collected every 15 minutes; adequacy of sedation was assessed using the Modified Thomas Combativeness Scale with the goal endpoint a score of 3 (No agitation, no supervision required, maybe asleep) Midazolam reached adequate sedation 13.9 minutes faster than lorazepam (95% CI 5.1 to 22.8; p=0.0026) Haloperidol required similar time to adequate sedation: 4.0 minutes faster than lorazepam (95% CI -8.2 to 16.3; p=0.5124) 7 lorazepam patients (26%) needed rescue drugs	The mean time to sedation: • lorazepam 2 mg IM: 32.2 minutes	There were no statistically significant differences over time in regard to change in systolic and diastolic blood pressure (p=0.8965, p=0.9581), heart rate (p=0.5517), respiratory rate (p=0.8191), and oxygen saturation (p=0.8991) among patients receiving each of the medications There were no adverse events in the lorazepam group

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Lorazepam					
Riddell et al[99] (2017)	Lorazepam (mean dose 1.9 mg) IV/ (mean dose 2.4 mg) IM • prospective, observational Compared to: • midazolam (mean dose 3.08 mg) IV/(mean dose 2.25 mg) IM/(mean dose 2 mg) IN • haloperidol (mean dose 5.71 mg) IM • combination of lorazepam (mean dose 2 mg) IV/(mean dose 2 mg) IM plus haloperidol (mean dose 5 mg) IM • ketamine (mean dose 0.87 mg/kg) IV/(mean dose 2.97 mg/kg) IM	Acutely agitated patients requiring chemical sedation in the ED 33 patients in the lorazepam group • median age: 43 years • 19 male/14 female • race: African American (5)/Asian (1)/Hispanic (13)/White (13) • drug use: 78.8% • alcohol use: yes (24.2%)/no (21.9%)/unknown (34.4%) • prior psychiatric visits (53.1%) • route of administration: IV(28)/IM(5)	Primary outcome: agitation score less than or equal to 2 on a six-point agitation scale • recorded prior to medication administration then at 5, 10, and 15 minutes • lorazepam (and other arms) inferior to ketamine at: 5 minutes ($p=0.001$), 10 minutes ($p<0.001$), and 15 minutes ($p=0.032$) Secondary outcomes of: • provider assessment of time to adequate sedation: No difference between groups ($p=0.107$) • need for redosing of sedative medications ($p=0.199$) • HR/SBP change: HR reduction seen with midazolam ($p=0.026$) but no other significant HR/SBP changes in any other study arms	Mean time to adequate sedation: • lorazepam: 17.73 minutes	Intubation: • lorazapam: 1/33

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines					
Lorazepam					
Martel et al[108] (2020)	Lorazepam 2 mg IM • prospective, randomized, double-blind trial • rescue sedation at treating physician discretion permitted 30 minutes after study drug administration for AMSS >0 Compared to: • droperidol 5 mg IM • ziprasidone 10 mg IM • ziprasidone 20 mg IM	ED patients with acute undifferentiated agitation requiring emergent sedation as determined by the treating physician. 31 patients in lorazepam group • median age: 39 years • 23 male/8 female • initial median AMSS scale score of 3 • initial median BARS score of 7 • initial assessment of reason for agitation: alcohol intoxication (25), drug intoxication (3), head injury (8), and primary psychiatric etiology (5). • final diagnoses: acute alcohol intoxication (29), acute drug intoxication (1), head injury (5), psychiatric disease (5), and other (1)	Primary outcome was adequate sedation at 15 minutes • a lesser proportion of lorazepam compared to droperidol treated patients met the primary outcome: 33% lower (95% CI 8% to 58%) while lorazepam did not differ from either dose of ziprasidone • lorazepam: 15/31 • droperidol: 16/25 • ziprasidone 10 mg: 7/28 • ziprasidone 20 mg: 11/31 AMSS scores were obtained every 15 minutes from time 0 to 120 minutes following study medication administration with median AMSS for lorazepam at: • 15 minutes: 2 • 30 minutes: 0 • 45 minutes: 0 • 60 minutes: -1 Additional sedation was required: • 7/31 before adequate sedation achieved • 12/31 in entire encounter • at a median time of 60 minutes following the initial administration	The post-administration assessment of adequate sedation occurred every 15 minutes post administration. The proportion achieving this at each check for lorazepam were: • at 15 minutes: 9/31 • at 30 minutes: 15/31 • at 45 minutes: 18/31 • at 60 minutes: 23/31	Respiratory depression was greater in lorazepam along with both ziprasidone groups compared to droperidol (p=0.04); for lorazepam: • 7/31 with hypoxemia (SpO_2<90%) • 14/31 with change in $ETCO_2$ • 15/31 with respiratory depression No patients in the lorazepam group required intubation. Median QTc: 414 ms • no dysrhythmias in lorazepam group No patients in lorazepam group experienced dystonia

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Droperidol					
Resnick et al[109] (1984)	Droperidol 5 mg IM • repeat dosing at 30-minute intervals up to 4 doses allowed for BPRS>17 • double-blind, prospective study Compared to: • haloperidol 5 mg IM	ED and psychiatric crisis patients with acute agitation and a score of >16 on BPRS. 11 patients in droperidol arm	Need for repeat medication administration used as a surrogate for inadequate control of agitation. Droperidol group with significantly higher proportion requiring only 1 injection (64% versus 19%, p<0.05) • 7/11 with 1 injection • 4/11 with 2 injections	No need for repeat medication injection used as a surrogate for adequate control of agitation at 30 minutes and each reevaluation thereafter	No adverse effects noted in droperidol group. • EPS symptoms were specifically monitored for.
Thomas et al[110] (1992)	Droperidol 5 mg IV/IM • study drug could be repeated or additional agent given at 30 minutes if initial administration ineffective. If additional or alternate drugs were received, only data up to 30 minutes were included for analysis. Compared to • haloperidol 5 mg IV/IM	ED patients who were markedly agitated and required physical restraint and constant attention from medical personnel were considered; those in whom 2 physicians agreed that the patient's agitation was not due to a readily correctible etiology such as hypoglycemia and that chemical restraint was warranted were included in the study 35 patients in the droperidol arm • 26 patients with IM administration (mean age: 34, 31% female, mean blood alcohol: 231 mg%) • 9 patients with IV administration (mean age: 36, 17% female, mean blood alcohol: 240 mg%)	5-point combativeness scale assessed at 5, 10, 15, 30, and 60-minute intervals after the study drug was administered. (1 is violently agitated and 5 is no agitation) • more rapid response to droperidol IM than haloperidol IM (p=0.03) • less agitation in droperidol IM than haloperidol IM at 10 minutes (p=0.004) • less agitation in droperidol IM than haloperidol IM at 15 minutes (p=0.01) • less agitation in droperidol IM than haloperidol IM at 30 minutes (p=0.04)	Combativeness scores for each assessment: • on agitation scale 4=slight agitation; unrestrained. • no definitive endpoint for adequate sedation defined in the study but removal of restraints could be considered a proxy with 4 considered adequate sedation Droperidol 5 mg IM 5 minutes—2.14 10 minutes—3.00 15 minutes—4.00 30 minutes—4.43	Droperidol 5 mg IM • clinically insignificant hypotension (4) No other adverse events observed

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Droperidol					
Martel et al[16] (2005)	Droperidol 5 mg IM • prospective, randomized, double-blind trial • rescue sedation at treating physician discretion permitted 30 minutes after study drug administration for AMS >0 Compared to: • midazolam 5 mg IM • ziprasidone 20 mg IM	ED patients with acute undifferentiated agitation requiring emergent sedation as determined by the treating physician. 50 patients in droperidol group • mean age 36.9 • 33 male/17 female • initial mean AMS scale score of 3.12 • initial assessment of reason for agitation: alcohol intoxication (46), illicit substance intoxication (4), head injury (7), and psychiatric etiology (2). • discharge diagnoses: acute alcohol intoxication (49), acute drug intoxication (1), and closed head injury (11)	AMS scale score was obtained every 15 minutes from time 0 to 120 minutes following study medication administration with effective sedation defined as an AMS of 0 or less Mean AMS scale scores in the droperidol group: • at 15 minutes: 0.28 (95% CI -0.34 to 0.9) • at 30 minutes: -1.3 (95% CI -1.76 to -0.84) • at 45 minutes: -1.56 (95% CI -2.02 to -1.1) • at 60 minutes: -1.56 (-1.99 to -1.13) Less patients receiving droperidol required rescue medications at 30 minutes compared to ziprasidone or midazolam ($p<0.05$) • droperidol: 5 patients required 6 doses • ziprasidone: 9 patients requiring 11 doses • midazolam: 24 patients requiring 30 doses	Less patients remained agitated at 15 minutes in the droperidol and midazolam groups compared to the ziprasidone group ($p=0.01$) • droperidol: 20/50 • midazolam: 15/48 • ziprasidone: 28/46 There was no difference between groups at 30 minutes ($p=0.08$) • droperidol: 6/50 • midazolam: 11/48 • ziprasidone: 14/46 Less patients were agitated at 45 minutes in the droperidol and ziprasidone groups compared to the midazolam group ($p=0.03$) • droperidol: 9/50 • midazolam: 14/48 • ziprasidone: 9/46	Respiratory depression: • 20/50 patients who received droperidol • 4 required supplemental oxygen • no difference in proportion with respiratory depression ($p=0.26$) or supplemental oxygen ($p=0.20$) when compared to midazolam and ziprasidone • no patients required intubation for respiratory depression Akathisia: • 1/50 patients who received droperidol Cardiac dysrhythmias: • none

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Droperidol					
Isbister et al[101] (2010)	Droperidol 10 mg IM • blinded, randomized controlled trial • further sedation allowed at discretion of attending physician Compared to: • midazolam 10 mg IM • midazolam 5 mg plus droperidol 5 mg IM	ED patients requiring physical restraint and parenteral sedation 33 patients in droperidol group • median age: 37 • 12 male/21 female • initial assessment of agitation due to: alcohol intoxication (23), self-harm (16), drug-induced delirium (2), acute psychosis (2), and other (1)	Primary sedation outcome was time security staff were required according to a security log from the time of initial call to the "all clear" • duration was not different between groups (p=0.66) with median for: midazolam (20 minutes), droperidol (24 minutes), and midazolam plus droperidol (25 minutes) Secondary sedation outcomes were: • time additional sedation was administered: the hazard ratio for additional sedation medications for midazolam versus droperidol was 2.31 (95% CI 1.01 to 4.71; post probability 0.98 for HR>1.0) indicating that midazolam was more likely to require additional sedation compared to droperidol	Secondary outcome of reduction in AMSS by 3 points or to a score of <1 20 minutes after drug administration • droperidol: 24/33	Respiratory events occurred in: • droperidol: 2/33 involving desaturation events (2) Hypotension occurred in: • droperidol: 0/33 Abnormal QT-HR pairs occurred in: • droperidol: 2/31 No dystonic reactions were identified Although oversedation was not a secondary endpoint, AMSS scores revealed that both midazolam and midazolam plus droperidol resulted in unpredictable and oftentimes deep sedation while droperidol resulted in consistent moderate sedation

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Droperidol					
Macht et al[111] (2014)	Droperidol IM (61%) and IV (39%) • mean dose 2.9 mg (median 2.5 mg) Compared to haloperidol IM (92%) and IV (8%) • mean dose 7.9 mg (median 10 mg) Retrospective chart review	218 EMS patients receiving droperidol for acute agitation • median age 31 • 75% male	Need for repeat sedating medication within 30 minutes of ED arrival was used as a surrogate endpoint for inadequate sedation • 21/207 (10%) received additional medication: butyrophenone (11) and benzodiazepine (14) There was no difference in need for sedating medications between the droperidol and haloperidol groups	Need for repeat sedation within 30 minutes of ED arrival was used as a surrogate endpoint for inadequate sedation but additional details of time to sedation are not reported	Adverse events reported were: SBP<90 mmHg (6), administration of an anti-arrhythmic medication (1), bag-valve mask (4), intubation (4), and cardiopulmonary arrest (1). No deaths were reported in the droperidol group • The cardiac arrest occurred in the midst of a physical struggle with staff in a combative patient with a history of congenital heart disease; CPR was administered for 1 minute with return of circulation. Post arrest QTc was 481 ms with no abnormal features. The patient was eventually discharged neurologically intact • no difference in proportion of adverse events compared to the haloperidol group QTc recorded in the hospital record for 166 patients; timing of measurement in relation

					to drug administration is not reported • Median QTc 453 ms • QTc 450 to 474 ms (59) • QTc 475 to 499 ms (27) • QTc >500 ms (5) • No difference in median QTc or proportion in any of the prolonged QTc stratifications compared to haloperidol group

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Droperidol					
Calver et al[115] (2015)	Droperidol 10 mg IM • clinician judgement for additional sedation at minutes with agent of clinician's choice although droperidol 10 mg IM recommended for repeat dosing	1,403 ED patients with acute behavioral disturbance, risk to self/others, and SAT score of 2 to 3 • mean age: 34 • 59.9% male • mean blood alcohol: 0.23 mg/dl • baseline SAT scores: 3 (61.9%)/2 (35.4%)/1 (2.6%) • presumed etiology: o alcohol intoxication: 52.6% o self-harm: 24.8% o psychostimulants: 13.8% o mental illness/psycho-sis: 15.7% o medical cause: 2.6% o other: 4.8%	Adequate sedation defined as reduction of SAT score by 2 or more, or reaching a score of 0 69% had adequate sedation after single dose 97% sedated by 120 minutes	Median time to sedation: • droperidol 10 mg IM: 20 minutes	No cases of torsades de pointes in entire cohort 1,009 patients with electrocardiogram recorded within 2 hours of droperidol administration: • median QT: 360 ms (95% CI: 320 to 440 ms) • 13/1,009 (1.3%: 95% CI 0.7% to 2.3%) with abnormal QT: 7 with other reasons for prolonged QT interval • 6/1,009 (0.6%: 95% CI 0.2% to 1.4%) with abnormal QT possibly due to droperidol 109/1,403 with oversedation based on SAT score with no clinical complications 70/1,403 (5.0%; 95% CI 3.9% to 6.3%) patients with total of 71 adverse events: • hypotension: 2.0% • desaturation: 1.6% • airway obstruction: 0.6% • hypoventilation: 0.2% • extrapyramidal side effects: 0.5% • seizure: 0.1% • arrhythmia: 0.1% 34 staff members injured 4 patients injured

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Droperidol					
Page et al[104] (2018)	Droperidol 10 mg IM • optional redosing of 10 mg at 15 minutes • prospective before/after protocol change observational study with primary endpoint to compare adverse events and secondary endpoints of sedation outcomes Compared to Midazolam IM or IV Per protocol: 5 mg initial IM dose with repeat doses of 5 to 10 mg every 10 minutes or 2.5 to 5 mg IV with repeat doses of 2.5 to 5 mg every 10 minutes	149 EMS patients with acute behavioral disturbance and SAT score of +2 (57 patients) to +3 (92 patients) • 81 male/68 female • reason for agitation: alcohol (66), amphetamines (32), medical (19), mental illness (18), other stimulants (8), self harm (20), and marijuana (1) • police were on scene for 123 encounters • median prehospital time of 44 minutes	Sedation was defined as a decrease in SAT score by at least 2 points or score of 0; successful sedation was defined as sedated, no adverse effects, and no requirement for additional sedation • 6/149 required additional EMS sedation • 11/149 required additional ED sedation • median number of drug administrations was 1 • 123/149 were successfully sedated. • 26 with unsuccessful sedation due to: failed to sedate prehospital (4), adverse effects (11), EMS additional sedation (6), and ED additional sedation (11)	Median time to sedation: • droperidol 10 mg IM: 22 minutes	11/149 patients exhibited 15 adverse events in the droperidol group • airway obstruction requiring airway maneuver (3: 2 chin lift/jaw thrust and 1 intubation), desaturation (3), hypotension (2), and SAT score of -3 (4). • compared to those receiving midazolam, a proportion 16% less in the droperidol group exhibited adverse events (p=0.0001, 95% CI 8% to 24%)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Droperidol					
Klein et al[113] (2019)	Droperidol 5 mg IM • retrospective chart review Compared to: • olanzapine 10 mg IM • haloperidol 5 mg IM	ED patients receiving parenteral antipsychotic for agitation 4,947 patients in droperidol arm • median age: 40 • 3,681 male/1,266 female • etiologies: alcohol (4,528), drug intoxication (411), psychiatric (552), and medical (8)	Primary outcome was rescue sedation administered within 1 hour of initial sedative • 547/4,947 (11%) required rescue sedation during initial hour: olanzapine (48), droperidol (478), haloperidol (1), benzodiazepine (18), and ketamine (2) • 832/4,947 (17%) received rescue sedation during ED encounter There was no difference between proportion of rescue sedation at 1 hour when comparing droperidol and olanzapine (0% difference: 95% CI -1% to 1%). Patients receiving droperidol required 7% less instances of rescue medication compared to haloperidol (95% CI 9% to 5% less)	Need for rescue medication at 1 hour documented but no additional details of time to sedation	In group receiving droperidol: Respiratory events • 9/4,947 (0.2%: 95% CI 0.1 to 0.3%) intubated Cardiac events • no cases of torsades de pointes or other cardiac events reported. Extrapyramidal side effects • 5 cases of akathisia • 2 cases of dystonia Allergic reactions • None

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Droperidol					
Gaw et al[122] (2020)	Droperidol • median dose of 0.625 mg • dose for different indications not documented • IM versus IV not documented • retrospective cohort study	ED droperidol administration for any indication 6,353 visits with droperidol administration • median age: 38 • female: 69.9%/male: 30.1% • indications: pain (21%); headache (57%); sedative (8.7%); antiemetic (12.5%)	Adequate sedation achieved in 48.3% of 56 patients receiving droperidol for sedation in a subgroup that underwent chart review	Not reported	QTc prolongation • no fatal arrhythmias • 0.7% with QTc of 500 ms or greater within 24 hours after droperidol • 1.2% with QTc of 500 ms or greater within 6 months prior to droperidol No deaths attributable to droperidol in entire Adverse events: • Akathisia: 2.9%

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Droperidol					
Martel et al[108] (2020)	Droperidol 5 mg IM • prospective, randomized, double-blind trial • rescue sedation at treating physician discretion permitted 30 minutes after study drug administration for AMSS >0 Compared to: • ziprasidone 10 mg IM • ziprasidone 20 mg IM • lorazepam 2 mg IM	ED patients with acute undifferentiated agitation requiring emergent sedation as determined by the treating physician 25 patients in droperidol group • median age: 39 • 21 male/4 female • initial median AMSS scale score: 3 • initial median BARS score: 7 • initial assessment of reason for agitation: alcohol intoxication (19), drug intoxication (1), head injury (3), and primary psychiatric etiology (3). • final diagnoses: acute alcohol intoxication (20), acute drug intoxication (0), head injury (1), psychiatric disease (3), and other (2)	Primary outcome was adequate sedation at 15 minutes • a greater proportion of droperidol treated patients compared to lorazepam 33% greater (95% CI 8% to 58%), ziprasidone 10 mg 39% greater (95% CI 14% to 64%), and ziprasidone 20 mg 29% greater (95% CI 3% to 54%) treated patients met the primary outcome • lorazepam: 15/31 • droperidol: 16/25 • ziprasidone 10 mg: 7/28 • ziprasidone 20 mg: 11/31 AMSS scores were obtained every 15 minutes from time 0 to 120 minutes following study medication administration with median AMSS for droperidol at: • 15 minutes: 0 • 30 minutes: -2 • 45 minutes: -2 • 60 minutes: -1 Additional sedation was required: • 2/25 before adequate sedation achieved • 5/25 in entire encounter • at a median time of 90 minutes following the initial administration	The post-administration assessment of adequate sedation occurred every 15 minutes post administration. The proportion achieving this endpoint at each check for droperidol was: • at 15 minutes: 16/25 • at 30 minutes: 22/25 • at 45 minutes: 21/25 • at 60 minutes: 22/25	Respiratory depression was less in the droperidol group compared to both ziprasidone groups along with lorazepam (p=0.04). For droperidol: • 2/25 with hypoxemia (SpO_2<90%) • 2/25 with change in $ETCO_2$ • 3/25 with respiratory depression No patients in the droperidol group required intubation. Median QTc: 413 ms. • one patient in the droperidol group experienced atrial flutter One patient in droperidol group experienced dystonia

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Droperidol					
Cole et al[114] (2021)	Droperidol 5 mg IM • prospective observational study Compared to: olanzapine 10 mg IM	ED patients with suspected drug or alcohol intoxication who received IM medication to treat acute agitation 538 patients in droperidol group • median age: 40 • male: 70% • 39% White/38% Black/14% Native American or Alaska Native/6% Hispanic/1% Asian/2% other or unknown • 86% with detectable alcohol concentration (median 0.2 % (g/dl)) • presumed cause: alcohol intoxication (86%)/illicit substance (15%)/psychiatric illness (12%)/medical (2%)	Adequate sedation defined as AMSS less than or equal to 0 No difference in the proportion of patients adequately sedated before 15 minutes: (droperidol 38%; olanzapine 42%; absolute difference -4% (95% CI -9% to 2%) • the hazard ratio for adequate sedation for droperidol compared with olanzapine was 1.12 (95% CI 1.00 to 1.25) Nadir AMSS scores tended to be higher (less sedation) for droperidol (median AMSS score -2) compared with olanzapine (median AMSS score -3). Patients who received olanzapine were more likely to receive additional medication for agitation while in the ED (droperidol 17%; olanzapine 24%; absolute difference -8% (95% CI -12% to -3%)	Median time to adequate sedation • droperidol 5 mg IM: 16 minutes	Of 538 patients in droperidol group Respiratory events: • any event: 23 • hypoxemia: 20 • supplemental oxygen: 6 • intubation: 4 • airway maneuver: 2 • aspiration: 1 Cardiovascular events: • hypotension: 13 • bradycardia: 2 Extrapyramidal events: • dystonia: 4 • akathisia: 2

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Olanzapine					
Centorrino et al[94] 2007	Olanzapine 10 mg IM • initial mean olanzapine dose was 9.9 ±2.2 mg • open label mixed retrospective and prospective observational report No comparison medication	Clinically agitated inpatient and emergency psychiatric services patients with bipolar mania or schizophrenia 74 patients receiving olanzapine IM: • 56.8% male • mean age 34.2 • diagnoses: bipolar mania or mixed-episode 29.7%; schizophrenia, schizoaffective disorder or schizophreniform disorder 70.3%	Agitation was assessed using the excitement component of the Positive and Negative Syndrome Scale (PANSS-EC), the changes in GCI Scale and the Agitation Calmness Evaluation Scale (ACES) There was significant improvement from baseline in all patients at 15 minutes ($p<0.001$)	Median time to adequate response: • olanzapine 10 mg IM: 30 minutes	No serious adverse events Treatment related adverse events in at least 4% of patients: • insomnia (9.5%) • arthralgia (7.9%) • headache (6.3%)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Olanzapine					

Cole et al[116] (2017)	Olanzapine 10 mg IM
	• actual dose received: 5 mg (6.4%)/10 mg (93.2%)/20 mg (0.4%)
	• prospective, observational report
	
	No comparison group
		ED patients receiving parenteral olanzapine during the study period
		
		489 in IM administration group:
		• median age: 39.5
		• male sex: 64%
		• White (38.9%)/Black American (32.2%)/American Indian (16.8%)/Hispanic (4.3%)/Somali (1.4%)/Asian (0.6%)/Other or mixed (5.7%)
		• median breath ethanol: 220 mg/dl
		• 430 received olanzapine for agitation
			Observer's Assessment of Alertness/Sedation (OAA/S) scale recorded at 0, 5, 10, 15, 30 and 60 minutes after initial dose
			
			Of those receiving olanzapine IM for agitation:
			84% did not require additional sedating medications within 60 minutes
			• provider satisfaction with improvement in symptoms was:
			  • none (0%)
			  • minimal (7%)
			  • moderate (25%)
			  • significant (49%)
			  • complete (19%)
				Median Observer's Assessment of Alertness/Sedation (OAA/S) score for Olanzapine 10 mg IM at time:
				• baseline: 5
				• 10 minutes: 4
				• 30 minutes: 3
				• 60 minutes: 3
					No patients experienced an allergic reaction, death, or a tachydysrhythmia.
					
					Respiratory depression: 10 patients
					• intubation: 5
					• bilevel positive airway pressure: 1
					• bag-valve-mask ventilation: 3
					• protective airway reflexes lost: 2
					• airway repositioning: 2
					• stimulation to induce respiration: 3
					• supplemental oxygen added: 7
					• airway suctioning 1
					
					Non respiratory adverse events:
					• sinus bradycardia: 1
					• akathisia: 1

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Olanzapine					
Klein et al[103] (2018)	Olanzapine 10 mg IM • prospective, observational Compared to: • midazolam 5 mg IM • haloperidol 5 mg IM • haloperidol 10 mg IM • ziprasidone 20 mg	ED patients receiving medication to treat acute agitation 163 patients in olanzapine arm • median age: 45 • 113 male/50 female • cause of agitation: alcohol (90%)/illicit substance (11%)/psychiatric illness (12%)/medical (1%)	Primary endpoint was adequate sedation, defined as Altered Mental Status Score <1 15 minutes after medication administration. • olanzapine 10 mg IM not inferior to midazolam 5 mg IM (9% lesser for olanzapine: 95% CI 20% lesser to 1% greater) • olanzapine 10 mg IM superior to haloperidol 5 mg IM (20% greater for olanzapine: 95% CI 10% to 31%) • olanzapine 10 mg IM superior to haloperidol 10 mg IM (18% greater for olanzapine: 95% CI 7% to 29%) • olanzapine 10 mg IM not superior to ziprasidone 20 mg IM (8% greater for olanzapine: 95% CI 3% lesser to 19% greater) Median difference in AMSS score compared to baseline at 15 minutes: • olanzapine 10 mg IM not inferior to midazolam 5 mg IM (1 point lesser decrease for olanzapine: 95% CI 0 to 1 point lesser decrease) • olanzapine 10 mg IM superior to haloperidol 5 mg IM (1 point greater	Median time to adequate sedation: • olanzapine 10 mg IM: 14 minutes	No difference in adverse events between groups Respiratory distress: • 1 to 3 patients in each arm with hypoxemia • 1 patient intubated in each arm except haloperidol 10 mg with no intubations Cardiovascular: • 1 to 2 patients in each group with hypotension except ziprasidone with no episodes of hypotension • 1 patient in each arm with bradycardia except midazolam with no episodes of bradycardia • no patients in any arm with torsades de pointes or other dysrhythmias Extrapyrimadal symptoms: • 2 patients in haloperidol 10 mg arm with dystonia; no other dystonic reactions in any arm • no episodes of akathisia in entire study

			decrease for olanzapine: 95% CI 1.5 to 1 point greater decrease) • olanzapine 10 mg IM superior to haloperidol 10 mg IM (1 point greater decrease for olanzapine: 95% CI 1.5 to 0.5 point greater decrease) • olanzapine 10 mg IM not superior to ziprasidone 20 mg IM (0 point difference: 95% CI 0.5 point greater decrease to 0.5 point lesser decrease) Time to adequate sedation (compared to midazolam 5 mg IM): • olanzapine 10 mg IM no different (HR 0.97, 95% CI 0.76 to 1.22) Time to adequate sedation for subset receiving monotherapy and no rescue sedation medications (compared to midazolam 5 mg IM): • olanzapine 10 mg IM no different (HR 0.84, 95% CI 0.65 to 1.07)		

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Olanzapine					
Klein et al[113] (2019)	Olanzapine 10 mg IM • retrospective chart review Compared to: • droperidol 5 mg IM • haloperidol 5 mg IM	ED patients receiving parenteral antipsychotic for agitation 8,825 patients • median age: 35 • 6,658 male/2,167 female • etiologies: alcohol (8,181), drug intoxication (619), psychiatric (891), and medical (25)	Primary outcome was rescue sedation administered within 1 hour of initial sedative • 988/8,825 (11%) required rescue sedation during initial hour: olanzapine (669), droperidol (17), haloperidol (274), benzodiazepine (26), and ketamine (2) • 1,665/8,825 (19%) received rescue sedation during ED encounter There was no difference between proportion of rescue sedation at 1 hour when comparing droperidol and olanzapine (0% difference: 95% CI -1% to 1%) Patients receiving olanzapine required 7% less instances of rescue medication compared to haloperidol (95% CI 9% to 5% less)	Need for rescue medication at 1 hour documented but no additional details of time to sedation	In group receiving olanzapine: Respiratory events: 36/8825 (0.4%: 95% CI 0.2% to 0.6%) intubated Cardiac events: • cardiac arrest occurred in 1 patient • no cases of torsades de pointes Extrapyramidal side effects: 2 cases of akathisia and 2 cases of dystonia Allergic reactions: 2 cases of rash

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Olanzapine					
Chan et al[106] (2021)	Olanzapine 5 mg IM • single optional redose allowed per study protocol • randomized, double-blind Compared to: • haloperidol 5 mg IM • midazolam 5 mg IM	ED patients requiring parenteral drug sedation for acute agitation 54 patients in olanzapine group • mean age 40 • 38 male/16 female • perceived possible causes: drug/substance abuse (14), alcohol intoxication (12), underlying mental illness (45), medication non-compliance (22), suicidal ideation/attempt (17), exposure to haloperidol (1), concurrent psychotropic medication (17) • baseline sedation scores: 3 (16 patients), 4 (21 patients), and 5 (16 patients) 16 patients in the olanzapine group received a second dose of study drug or alternative sedatives.	Agitation/sedation level was measured on a 6-point validated sedation scale: (5=highly aroused, violent; 4=highly aroused, possibly distressed, or fearful; 3=moderately aroused, unreasonable, or hostile; 2=mildly aroused, willing to talk reasonably; 1=minimal agitation; and 0=asleep); adequate sedation was defined as a score of 2 or less Sedation scores were recorded at baseline, at first observed adequate sedation, and at 10, 20, 30, 45, and 60 minutes after the first dose regardless of observed time to sedation • midazolam was superior to olanzapine with significant differences detected in the Kaplan-Meier curves (p=0.03) • no difference for haloperidol compared with olanzapine (p=0.78) At 10 minutes after the initial dose, 34% in the olanzapine group were adequately sedated. At 60 minutes, the proportion of patients adequately sedated increased to 87% Fully adjusted accelerated factor for olanzapine was compared with midazolam at 1.72 (95% CI 1.16 to 2.55), indicating significantly slower sedation for olanzapine	Median time to sedation for olanzapine 5 mg IM: 11.5 minutes	3 patients in the olanzapine group experienced an adverse event; 1 patient experienced oxygen desaturation and 2 patients reported dry mouth 10 patients receiving olanzapine fell asleep after treatment

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Olanzapine					
Cole et al[114] (2021)	Olanzapine 10 mg IM • prospective observational study Compared to: droperidol 5 mg IM	ED patients with suspected drug or alcohol intoxication who received IM medication to treat acute agitation 719 patients in olanzapine group • median age: 43 • male: 75% • 40% White/33% Black/16% Native American or Alaska Native/3% Hispanic/1% Asian/<1% other or unknown • 87% with detectable alcohol concentration (median 0.2 % (g/dl)) • presumed cause: alcohol intoxication (87%)/illicit substance (13%)/psychiatric illness (13%)/medical (1%)	Adequate sedation defined as AMSS less than or equal to 0 No difference in the proportion of patients adequately sedated before 15 minutes: (olanzapine 42%; droperidol 38%; absolute difference -4% [95% CI -9% to 2%]) • the hazard ratio for adequate sedation for droperidol compared with olanzapine was 1.12 (95% CI 1.00 to 1.25) Nadir AMSS scores tended to be higher (less sedation) for droperidol (median AMSS score -2) compared with olanzapine (median AMSS score -3) Patients who received olanzapine were more likely to receive additional medication for agitation while in the ED (olanzapine 24%; droperidol 17%; absolute difference -8% [95% CI -12% to -3%])	Median time to adequate sedation • olanzapine 10 mg IM: 17.5 minutes	Of 719 patients in olanzapine group Respiratory events • any event: 47 • hypoxemia: 42 • supplemental oxygen: 30 • intubation: 7 • airway maneuver: 5 • aspiration: 3 Cardiovascular events • hypotension: 19 • bradycardia: 1 Extrapyramidal events • dystonia: 0 • akathisia: 1

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					
Resnick et al[109] (1984)	Haloperidol 5 mg IM • repeat dosing at 30-minute intervals up to 4 doses allowed for BPRS>17 • double-blind, prospective study Compared to: • droperidol 5 mg IM	ED and psychiatric crisis patients with acute agitation and a score of >16 on BPRS 16 patients in haloperidol arm	Need for repeat medication administration used as surrogate for inadequate control of agitation Haloperidol group with significantly lower proportion requiring only 1 injection (19% versus 64%, p<0.05) • 3/16 with 1 injection • 10/16 with 2 injections • 2/16 with 3 injections • 1/16 with 4 injections	No need for repeat medication injection surrogate for adequate control of agitation at 30 minutes and each reevaluation thereafter	1 dystonic reaction noted in haloperidol group
Thomas et al[110] (1992)	Haloperidol 5 mg IV/IM • study drug could be repeated, or additional agent given at 30 minutes if initial administration ineffective. If additional or alternate drugs were received, only data up to 30 minutes were included for analysis Compared to droperidol 5 mg IV/IM	ED patients who were markedly agitated and required physical restraint and constant attention from medical personnel were considered. Those in whom 2 physicians agreed that the patient's agitation was not due to a readily correctible etiology such as hypoglycemia and that chemical restraint was warranted were included in the study 33 patients in the haloperidol arm • 21 patients with IM administration (mean age: 31, 52% female, mean blood alcohol: 174 mg%) • 12 patients with IV administration (mean age: 31, 0% female, mean blood alcohol: 250 mg%)	5-point combativeness scale assessed at 5, 10, 15, 30, and 60-minute intervals after the study drug was administered. (1 is violently agitated and 5 is no agitation) • less rapid response to haloperidol IM than droperidol IM (p=0.03) • more agitation in haloperidol IM than droperidol IM at 10 minutes (p=0.004) • more agitation in haloperidol IM than droperidol IM at 15 minutes (p=0.01) • more agitation in haloperidol IM than droperidol IM at 30 minutes (p=0.04)	Combativeness scores for each assessment: -on agitation scale 4=slight agitation; unrestrained. • no definitive endpoint for adequate sedation defined in the study but removal of restraints could be considered a proxy with 4 considered adequate sedation Haloperidol 5 mg IM at time: 5 minutes—1.33 10 minutes—2.11 15 minutes—3.11 30 minutes—3.75	Haldol 5 mg IM -Clinically insignificant hypotension (2) -Dystonic reaction 18 hours after drug administration (1) No other adverse events observed

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					
Battaglia et al[107] (1997)	Haloperidol 5 mg IM • randomized and double blind • repeat doses allowed but not until after the first post-treatment standardized evaluation at 1 hour Compared to: • lorazepam 2 mg IM • lorazepam 2 mg plus haloperidol 5 mg IM	ED with psychosis and behavioral dyscontrol (agitated, aggressive, destructive, assaultive, or restless behavior) to the extent that they were capable of harming themselves or others 35 ED patients in the haloperidol group • 25 male/10 female • mean age 34.3 years • mean weight 73.3 kg patients • final diagnoses were mania, psychoactive substance abuse, psychosis not otherwise specified, schizophrenia, and schizophreniform disorder	Agitation was assessed serially using the Agitated Behavior Scale with a significant reduction in agitation from baseline at 1 hour in the haloperidol arm; the reduction in agitation seen with haloperidol was not greater than lorazepam alone (p=0.426) or combination therapy (p=0.064) Approximately 2.5% of patients in the haloperidol group were asleep at 1 hour, significantly less than the lorazepam alone group or the combination therapy group	Serial evaluations occurred for 12 hours with redosing allowed after reevaluations; only 1-hour endpoints were abstracted as they are most relevant to this review	14 lorazepam-treated patients (40%) reported adverse effects: • ataxia: 1 (3%) • dizziness: 3 (9%) • dry mouth: 3 (9%) • EPS symptoms: 7 (20%) • speech disorder: 4 (11%) "No serious side effects" were reported

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					
Foster et al[96] (1997)	Haloperidol 5 mg oral concentrate or IM • redoses allowed every 30 minutes up to 4 hours until sedated or no longer a danger to self or others Compared to: • lorazepam 2 mg oral concentrate or IM	Patients presenting at the psychiatric emergency service of a large urban hospital judged by emergency room staff to be an imminent danger to themselves, they required 4-point physical restraints, they scored a 5 or higher on at least 3 items on the BPRS, and they had a score of at least 4 on the GCI Scale 20 patients in the haloperidol group: • mean age 42.35 years • 14 male and 6 female • final diagnoses of schizophrenia (8), bipolar (3), schizoaffective (3), and psychotic disorder not otherwise specified (6) • 4 patients with drug abuse or dependence by history	The primary endpoint was reduction in the BPRS with a secondary endpoint of reduction in the GCI Scale The haloperidol group exhibited significant decreases in both rating scales over the course of the study, although no drug by time interactions were found; analysis of route of administration did not reveal significant effects BPRS reductions were not different for lorazepam and haloperidol at 1 hour; the lorazepam group exhibited a significantly greater reduction compared to the haloperidol group on the GCI scale at 1 hour	Serial hourly evaluations were performed by trained evaluators. Only 1-hour outcomes are relevant for this review	There were no group differences HR/SBP pressure, and diastolic blood pressure and all parameters significantly decreased across time

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					

TREC Collaborative Group[100] (2003)	Combination of haloperidol 5 mg to 10 mg plus promethazine 25 mg to 50 mg IM
	• prospective, pseudo-randomized open label
	• dose at treating physician discretion
	
	Compared to:
	• midazolam 7.5 mg to 15 mg IM
		Adults presenting to psychiatric Eds with agitation or dangerous behavior
		
		148 patients in the haloperidol/promethazine arm
		• 49% male/51% female
		• mean age: 38
		• dose of haloperidol: 10 mg (71 patients)/5 mg (77)
		• dose of promethazine: 50 mg (147)/25 mg (1)
		• presumed etiology:
		  • Psychosis 75%
		  • Substance abuse 14%
		  • Other 11%
			Primary endpoint was "tranquil or asleep" at 20 minutes, with tranquil defined as peaceful and without restlessness or threatening behavior
			
			Secondary endpoints included tranquil or asleep at 40, 60, and 120 minutes; need for physical restraints; recurrent episode of agitation; major adverse events.
			
			Haloperidol inferior for primary endpoint at 20 minutes as well as secondary endpoint at 40 minutes
			• no difference at 60 minutes or greater
			• no difference in need for restraints
			• no difference in additional tranquilizing drugs
			
			At 20 minutes, 67% in the Haloperidol/promethazine arm versus 89% in the midazolam arm reached primary endpoint
			• RR 1.32 (95% CI 1.16 to 1.49)
			• 22% (95% CI 12% to 30%) less in haloperidol/promethazine group w/ adequate sedation at 20 minutes
				At 20 minutes, 67% in the haloperidol/ promethazine arm reached primary endpoint	1 patient in Haldol/promethazine group with history of epilepsy experienced seizure that resolved with benzodiazepine

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					
Nobay et al[98] (2004)	Haloperidol 5 mg IM • randomized and double blind • if a patient continued to be disruptive 20 minutes after the study drug was administered, a ‘‘rescue drug’’ could be given at the discretion of the treating attending physician; patient enrollment in the study was terminated if a rescue medication was given; these patients were considered sedation failures, and their data were not included in the analysis Compared to: • lorazepam 2 mg IM • midazolam 5 mg IM	ED patients who required emergency sedation for the control of violent behavior or severe agitation; all patients were initially physically restrained 42 patients in the haloperidol group • mean age 42.4 years • 23 African American, 1 Asian, 3 Hispanic, and 15 White • 11 with recreational drug use, 2 without, and 29 unknown • 14 with alcohol use, 1 without, and 27 unknown • 20 with prior psychiatric history, 4 without, and 18 unknown	Level of sedation was continuously observed with data collected every 15 minutes; adequacy of sedation was assessed using the Modified Thomas Combativeness Scale with the goal endpoint a score of 3 (No agitation, no supervision required, maybe asleep) 8 haloperidol patients (19%) needed rescue drugs Lorazepam required similar time to adequate sedation: 4.0 minutes slower than haloperidol (95% CI -8.2 to 16.3; p=0.5124) Midazolam reached adequate sedation 9.9 minutes faster than haloperidol (95% CI 0.5 to 19.3; p=0.0388)	The mean time to sedation • haloperidol 5 mg IM: 28.3 minutes	There were no statistically significant differences over time in regard to change in systolic and diastolic blood pressure (p=0.8965, p=0.9581), heart rate (p=0.5517), respiratory rate (p=0.8191), and oxygen saturation (p=0.8991) among patients receiving each of the medications There were 2 adverse events in the haloperidol group; one patient became hypotensive and another apneic, but both subsequently recovered fully

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					
Macht et al[111] (2014)	Haloperidol IM (92%) and IV (8%) • mean dose 7.9 mg (median 10 mg) • retrospective chart review Compared to: • droperidol IM (61%) and IV (39%) • mean dose 2.9 mg (median 2.5 mg)	314 EMS patients receiving haloperidol for acute agitation • median age 31 • 69% male	Need for repeat sedating medication within 30 minutes of ED arrival was used as a surrogate endpoint for inadequate sedation • 41/314 (13%) received additional medication: butyrophenone (22) and benzodiazepine (20) There was no difference in need for sedating medications between the haloperidol and droperidol groups	Need for repeat sedation within 30 minutes of ED arrival was used as a surrogate endpoint for inadequate sedation but additional details of time to sedation are not reported	Adverse events reported were: SBP<90 mmHg (13), administration of an anti-arrhythmic medication (5), bag-valve mask (12), and intubation (12). No cardiac arrest or death in the haloperidol group • no difference in proportion of adverse events compared to the droperidol group QTc recorded in the hospital record for 78 patients; timing of measurement in relation to drug administration is not reported • median QTc 448 ms • QTc 450 ms to 474 ms (23) • QTc 475 ms to 499 ms (9) • QTc >500ms (3) • no difference in median QTc or proportion in any of the prolonged QTc stratifications compared to droperidol group

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					
Asadohalli et al[117] (2015)	Haloperidol 5 mg IM • randomized, double blind placebo-controlled study • single redose allowed Compared to: • valproic acid	80 ED patients with violent, controlled, or uncontrolled muscular movement that placed both themselves and hospital staff in danger because of severely disruptive behavior • mean age: 44.55 years • 49 male/31 female • 61 physically restrained • etiology: 55-mental disorders, 21-other (infection, substance intoxication, or withdrawal), and 4 unknown • 58 reported prior use of psychiatric medications	The primary outcome measure was the Agitation–Calmness Evaluation Scale (ACES); secondary outcomes were changes in the Positive and Negative Syndrome Scale-Excited Component (PANSS-EC) and the Agitated Behavior Scale (ABS) scores Haloperidol exhibited a greater change in Agitation Calmness Evaluation Scale (ACES) score at 30 minutes compared to valproate (p=0.028). there was no difference in Positive and Negative Syndrome Scale-Excited Component (PANSS-EC) (p=0.649) or Agitated Behavior Scale scores (0.651). Similar numbers of patients required a 2nd dose of medication (haloperidol=17 and valproate=13; p=0.418); the mean duration of physical restraint did not differ significantly between patients receiving valproate and haloperidol (37.4 versus 38.9 minutes, p=0.100)	Outcomes were measured at 30 minutes following medication administration.	There were no statistically significant differences up to 30 minutes after injection with respect to changes in systolic and diastolic blood pressure (P=0.77, P=0.12), heart rate (P=0.64), and respiratory rate (P=0.78) among patients receiving each of the interventions The haloperidol treatment group had a significantly larger proportion (37 patients, p=0.034) who showed at least one adverse event • intense sedation 30 minutes after intervention was the most frequent adverse event in the haloperidol versus valproate group (29 versus 2, p<0.001) • 7 patients (p=0.007) experienced EPS in the haloperidol study arm; these patients • received anticholinergic agents • hypotension occurred in • one patient receiving haloperidol

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					
Isenberg et al[102] (2015)	Haloperidol 2.5 mg to 5 mg IM (5 mg if younger than 65 years and 2.5 mg if 65 years or older) • randomized, non-blinded • re-dosing available every 10 minutes if sedation endpoint not met but maximum dose received was 5 mg Compared to: • midazolam 2.5 mg to 5 mg IM	EMS patients with either: • a psychiatric or behavioral disorder who is at imminent risk of self-injury or is a threat to others • patient with a medical condition causing agitation and possibly violent behavior 5 patients in haloperidol group • age 18 to 89 • all with initial RASS +4 • patient diagnosis: urinary tract infection (1) and alcohol intoxication (4)	Sedation evaluated using RASS with goal of less than +1. 5/5 patients in haloperidol group with RASS less than +1 on arrival to ED	Mean time to achieve a RASS of less than +1: • haloperidol 2.5 mg to 5 mg IM: 24.8 minutes	No patients in the haloperidol group had any adverse effects Mean time to return to baseline mental status was 84 minutes (95% CI 0 to 202 minutes)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					
Cole et al[118] (2016)	Haloperidol 10 mg IM (5 patients received 5 mg rather than 10 mg as initial dose) • prospective open label EMS study Compared to: • ketamine 5 mg/kg IM	82 acute undifferentiated agitation with AMSS +3 (60 patients) to +2 (22 patients); AMSS +4 excluded as "profound agitation" • median age 31 • 44 male/38 female • 33 Caucasian, 25 Black American, 14 American Indian, 2 Somali, 2 Hispanic, 1 Asian, 5 mixed/unknown • 55 (67%) with history of mental illness, 59 (72%) with history of chemical dependency, and 43 (52%) with both • EMS impressions of: agitated combative (21), substance abuse (23), behavioral (8), AMS (10), trauma (7), overdose (4), and seizure (1)	Primary endpoint of AMSS < +1. • 53/82 patients achieved adequate sedation • 16/82 patients required second injection prehospital: midazolam (15) and haloperidol (1) Compared to the group receiving ketamine, 30% less patients in the haloperidol group successfully achieved adequate sedation ($p<0.0001$, 95% CI 18% to 42%) Time to sedation was 12 minutes greater for haloperidol group compared to the ketamine group ($p<0.0001$, 95% CI 9 to 15 minutes)	Median time to adequate sedation: • haloperidol 10 mg IM: 17 minutes	Five complications occurred in 4/82 patients: vomiting (2), dystonia (2), and death (1); per communication with study author, the death was related to polytrauma and the patient died days after receiving haloperidol due to traumatic injuries • complications occurred in 44% less patients in the haloperidol group compared to the ketamine group ($p<0.0001$, 95% CI 30% to 57%) Intubation occurred in 3/82 patients for the following indications: not protecting airway (1) and refractory agitation (2) • intubation occurred in 35% less patients in the haloperidol group compared to the ketamine group ($p<0.0001$, 95% CI 23% to 48%)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					
Riddell et al[99] (2017)	Haloperidol (mean dose 5.71 mg) IM • prospective, observational Compared to: • midazolam (mean dose 3.08 mg) IV/(mean dose 2.25 mg) IM/(mean dose 2 mg) IN • lorazepam (mean dose 1.9 mg) IV/ (mean dose 2.4 mg) IM • combination of lorazepam (mean dose 2 mg) IV/(mean dose 2 mg) IM plus haloperidol (mean dose 5 mg) IM • ketamine (mean dose 0.87 mg/kg) IV/(mean dose 2.97 mg/kg) IM	Acutely agitated patients requiring chemical sedation in the ED 14 patients in the haloperidol group • median age: 44 • 11 male/3 female • race: African American (1)/Asian (1)/Hispanic (8)/White (4) • drug use: 85.7% • alcohol use: yes (35.7%)/no (35.7%)/unknown (28.6%) • prior psychiatric visits (50.0%) • route of administration: IM (14)	Primary outcome: agitation score less than or equal to 2 on a six-point agitation scale • recorded prior to medication administration then at 5, 10, and 15 minutes • haloperidol (and other arms) inferior to ketamine at: 5 minutes ($p=0.001$), 10 minutes ($p<0.001$), and 15 minutes ($p=0.032$) Secondary outcomes of: • provider assessment of time to adequate sedation: No difference between groups ($p=0.107$) • need for redosing of sedative medications ($p=0.199$) • HR/SBP change: HR reduction seen with midazolam ($p=0.026$) but no other significant HR/SBP changes in any other study arms	Mean time to adequate sedation: • haloperidol IM: 13.43 minutes	Intubation: • haloperidol: 1/14 • midazolam: 1/19 • lorazapam: 1/33 • combination lorazepam plus haloperidol: 1/10 • ketamine: 2/24

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					
Heydari et al[119] (2018)	Haloperidol 5 mg IM • if patient was inadequately sedated or additional medication needed per physician discretion then 2.5 mg repeat dose allowed • randomized, double blind prospective trial Compared to: • ketamine 4 mg/kg IM	ED patients with acute agitated and aggressive behavior who required chemical sedation for agitation, according to an emergency medicine resident or attending physician were eligible for enrollment. (AMSS +2 or +3) 45 patients in haloperidol group: • mean age of 29.93 • male 75.6%/female 24.4% • cause of aggressive behavior: psychotropic substances (26.7%)/psychiatric history (33.3%)/alcohol consumption (28.9%)/trauma (11.1%)	The primary outcome was time to adequate sedation (AMSS≤+1) -slower for haloperidol compared to ketamine (p<0.01, difference 3.7 minutes, 95% CI: 2.1 to 5.5) Mean AMSS scores: • 5 minutes: haloperidol (1.70) was not different from ketamine (1.36) (p=0.115) • 10 minutes: haloperidol (1.27) was higher than ketamine (0.67) (p=0.001) • 15 minutes: haloperidol (0.3) was not different from ketamine (0.14) (p=0.167) • proportion not adequately sedated at 15 minutes was higher in haloperidol group (28.9%) than ketamine group (6.7%) • difference of 22%: 95% CI 11% to 33% (p<0.0001) Physician satisfaction was lower in haloperidol group than ketamine group (p=0.011)	Median time to adequate sedation • haloperidol 5 mg IM: 11.4 minutes	Complications: • 17.8% for haloperidol • 35.6% for ketamine • no significant difference between groups (p=0.094, difference 17%, 95% CI 11% to 22%). Haloperidol group: • vomiting (n=1, 2.2%), • dystonia (n=2, 4.4%), • akathisia (n=4, 8.9%), • hypoxia (n=1, 2.2%) • Intubation (n=3, 6.7%) Primary indications for intubation in haloperidol group were refractory agitation (2) and hypoxia (1)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					
Klein et al[103] (2018)	Haloperidol 5 mg IM • prospective, observational Compared to: • midazolam 5 mg IM • olanzapine 10 mg • IM • haloperidol 10 mg IM • ziprasidone 20 mg	ED patients receiving medication to treat acute agitation 151 patients in haloperidol 5 mg arm • median age: 40 • 101 male/50 female • cause of agitation: alcohol (90%)/illicit substance (10%)/psychiatric illness (10%)/medical (1%)	Primary endpoint was adequate sedation, defined as Altered Mental Status Score <1 15 minutes after medication administration • haloperidol 5 mg IM inferior to midazolam 5 mg IM (30% lesser for haloperidol: 95% CI 41% lesser to 19% lesser) • haloperidol 5 mg IM inferior to olanzapine 10 mg IM (20% lesser for haloperidol: 95% CI 31% lesser to 10% lesser) • haloperidol 5 mg IM no different than haloperidol 10 mg IM (2% lesser for haloperidol 5 mg: 95% CI 13% lesser to 9% greater) • haloperidol 5 mg IM inferior to ziprasidone 20 mg IM (12% lesser for haloperidol 5 mg: 95% CI 23% lesser to 1% lesser) Median difference in AMSS score compared to baseline at 15 minutes: • haloperidol 5 mg IM inferior to midazolam 5 mg IM (2 point lesser decrease for haloperidol: 95% CI 2.5 lesser decrease to 1.5 point lesser decrease) • haloperidol 5 mg IM inferior to olanzapine 10	Median time to adequate sedation: • haloperidol 5 mg IM: 20 minutes	No difference in adverse events between groups Respiratory distress: • 1 to 3 patients in each arm with hypoxemia • 1 patient intubated in each arm except haloperidol 10 mg with no intubations Cardiovascular: • 1 to 2 patients in each group with hypotension except ziprasidone with no episodes of hypotension • 1 patient in each arm with bradycardia except midazolam with no episodes of bradycardia • no patients in any arm with torsades de pointes or other dysrhythmias Extrapyrimadal symptoms: • 2 patients in haloperidol 10 mg arm with dystonia. No other dystonic reactions in any arm • no episodes of akathisia in entire study

			mg IM (1 point lesser decrease for haloperidol 5 mg, 95% CI 1.5 lesser decrease to 1 point lesser decrease) • haloperidol 5 mg IM no different than haloperidol 10 mg IM (0 point difference between haloperidol doses, 95% CI 0.5 point lesser decrease to 0.5 point greater decrease) • haloperidol 5 mg IM inferior to ziprasidone 20 mg IM (1 point lesser decrease for haloperidol 5 mg, 95% CI 1.5 point lesser decrease to 0.5 point lesser decrease) Time to adequate sedation (compared to midazolam 5mg IM): • haloperidol 5 mg IM inferior (HR 0.73, 95% CI 0.58 to 0.90) Time to adequate sedation for subset receiving monotherapy and no rescue sedation medications (compared to midazolam 5 mg IM): • haloperidol 5 mg IM inferior HR 0.63 (95% CI 0.48 to 0.81)		

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					
Klein et al[103] (2018)	Haloperidol 10 mg IM • prospective, observational Compared to: • midazolam 5 mg IM • olanzapine 10 mg • IM • haloperidol 5 mg IM • ziprasidone 20 mg	ED patients receiving medication to treat acute agitation 151 patients in haloperidol 10 mg arm • median age: 38 • 107 male/44 female • cause of agitation: alcohol (85%)/illicit substance (15%)/psychiatric illness (9%)/medical (1%)	Primary endpoint was adequate sedation, defined as Altered Mental Status Score <1 15 minutes after medication administration. • haloperidol 10 mg IM inferior to midazolam 5 mg IM (28% lesser for haloperidol: 95% CI 39% lesser to 17% lesser) • haloperidol 10 mg IM inferior to olanzapine 10 mg IM (18% lesser for haloperidol: 95% CI 29% lesser to 7% lesser) • haloperidol 10 mg IM no different than haloperidol 5 mg IM (2% greater for haloperidol 10 mg: 95% CI 9% lesser to 13% greater) • haloperidol 10 mg IM no different than ziprasidone 20 mg IM (10% lesser for haloperidol 10 mg: 95% CI 21% lesser to 0% different) Median difference in AMSS score compared to baseline at 15 minutes: • haloperidol 10 mg IM inferior to midazolam 5 mg IM (2 point lesser decrease for haloperidol: 95% CI 2.5 lesser decrease to 1.5 point lesser decrease)	Median time to adequate sedation: • haloperidol 10 mg IM: 19 minutes	No difference in adverse events between groups Respiratory distress: • 1 to 3 patients in each arm with hypoxemia • 1 patient intubated in each arm except haloperidol 10 mg with no intubations Cardiovascular: • 1 to 2 patients in each group with hypotension except ziprasidone with no episodes of hypotension • 1 patient in each arm with bradycardia except midazolam with no episodes of bradycardia • no patients in any arm with torsades de pointes or other dysrhythmias Extrapyrimadal symptoms: • 2 patients in haloperidol 10 mg arm with dystonia; no other dystonic reactions in any arm • no episodes of akathisia in entire study

			• haloperidol 10 mg IM inferior to olanzapine 10 mg IM (1 point lesser decrease for haloperidol 5 mg: 95% CI 1.5 lesser decrease to 0.5 point lesser decrease) • haloperidol 10 mg IM no different than haloperidol 5 mg IM (0 point difference between haloperidol doses: 95% CI 0.5 point lesser decrease to 0.5 point greater decrease) • haloperidol 5 mg IM inferior to ziprasidone 20 mg IM (1 point lesser decrease for haloperidol 5 mg: 95% CI 1.5 point lesser decrease to 0.5 point lesser decrease) Time to adequate sedation (compared to midazolam 5 mg IM): • haloperidol 10 mg IM inferior (HR 0.72: 95% CI 0.57 to 0.88) Time to adequate sedation for subset receiving monotherapy and no rescue sedation medications (compared to midazolam 5 mg IM): • haloperidol 10 mg IM inferior (HR 0.59: 95% CI 0.46 to 0.78)		

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					
Klein et al[113] (2019)	Haloperidol 5 mg IM • retrospective chart review Compared to: • droperidol 5 mg IM • olanzapine 10 mg IM	ED patients receiving parenteral antipsychotics for agitation 2,146 patients in haloperidol group • median age: 38 • 1,656 male/490 female • etiologies: alcohol (1,979), drug intoxication (154), psychiatric (212), and medical (13)	Primary outcome was rescue sedation administered within 1 hour of initial sedative • 390/2,146 (18%) required rescue sedation during initial hour: olanzapine (70), droperidol (0), haloperidol (254), benzodiazepine (63), and ketamine (3) • 560/2,146 (26%) received rescue sedation during ED encounter Patients receiving haloperidol required 7% greater instances of rescue medication compared to both droperidol and olanzapine (95% CI 9% to 5% less)	Need for rescue medication at 1 hour documented but no additional details of time to sedation	In group receiving haloperidol: Respiratory events: • 4/2,146 (0.2%: 95% CI 0.1 to 0.5%) intubated Cardiac events • no cases of torsades de pointes or other cardiac events reported. Extrapyramidal side effects • 0 cases of akathisia • 1 case of dystonia Allergic reactions • none

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Haloperidol					
Chan et al[106] (2021)	Haloperidol 5 mg IM • single optional redose allowed per study protocol • randomized, double-blind Compared to: • olanzapine 5 mg IM • midazolam 5 mg IM	ED patients requiring parenteral drug sedation for acute agitation 57 patients in haloperidol group • mean age 42 years • 24 male/18 female • perceived possible causes: drug/substance abuse (19), alcohol intoxication (13), underlying mental illness (46), medication non-compliance (18), suicidal ideation/attempt (18), exposure to haloperidol (1), concurrent psychotropic medication (13) • baseline sedation scores: 3 (14 patients), 4 (17 patients), and 5 (25 patients) 23 patients in the haloperidol group received a second dose of study drug or alternative sedatives	Agitation/sedation level was measured on a 6-point validated sedation scale: (5=highly aroused, violent; 4=highly aroused, possibly distressed, or fearful; 3=moderately aroused, unreasonable, or hostile; 2=mildly aroused, willing to talk reasonably; 1=minimal agitation; and 0=asleep); adequate sedation was defined as a score of 2 or less Sedation scores were recorded at baseline, at first observed adequate sedation, and at 10, 20, 30, 45, and 60 minutes after the first dose regardless of observed time to sedation • midazolam was superior to haloperidol with significant differences detected in the Kaplan-Meier curves (p=0.002) • no difference for haloperidol compared with olanzapine (p=0.78) At 10 minutes after the initial dose, 21% in the haloperidol group were adequately sedated; at 60 minutes, the proportion of patients adequately sedated increased to 97% Fully-adjusted accelerated factor for haloperidol was compared with midazolam at 1.89 (95% CI 1.28 to 2.80), indicating significantly slower sedation for haloperidol	Median time to sedation: • haloperidol 5 mg IM: 23.0 minutes	3 patients in the haloperidol group experienced an adverse event; 1 patient experienced oxygen desaturation, 1 patient experienced dystonia, and 1 patient experienced a cardiac arrest 3 hours after 2nd dose of haloperidol and died 8 days later 13/57 exhibited QTc prolongation 17 patients receiving haloperidol fell asleep after treatment

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Ziprasidone					
Martel et al[16] (2005)	Ziprasidone 20 mg IM • prospective, randomized, double-blind trial • rescue sedation at treating physician discretion permitted 30 minutes after study drug administration for AMS >0 Compared to: • midazolam 5 mg IM • droperidol 5 mg IM	ED patients with acute undifferentiated agitation requiring emergent sedation as determined by the treating physician. 46 patients in ziprasidone group • mean age 36.8 • 32 male/14 female • initial mean AMS scale score of 3.41 • initial assessment of reason for agitation: alcohol intoxication (43), illicit substance intoxication (5), head injury (9), psychiatric etiology (8), and delirium (1). • discharge diagnoses: acute alcohol intoxication (44), acute drug intoxication (3), and closed head injury (12)	AMS scale score was obtained every 15 minutes from time 0 to 120 minutes following study medication administration with effective sedation defined as an AMS of 0 or less More patients remained agitated at 15 minutes in ziprasidone group compared to the the droperidol and midazolam groups (p=0.01) • droperidol: 20/50 • midazolam: 15/48 • ziprasidone: 28/46 There was no difference between groups at 30 minutes (p=0.08) • droperidol: 6/50 • midazolam: 11/48 • ziprasidone: 14/46 Less patients were agitated at 45 minutes in the droperidol and ziprasidone groups compared to the midazolam group (p=0.03). • droperidol: 9/50 • midazolam: 14/48 • ziprasidone: 9/46 More patients receiving ziprasidone or midazolam required rescue medications at 30 minutes compared to droperidol (p<0.05) • droperidol: 5 patients required 6 doses • ziprasidone: 9 patients requiring 11 doses • midazolam: 24 patients requiring 30 doses	Mean AMS scale scores in the ziprasidone 20 mg IM group: • at 15 minutes: 1.1 (95% CI 0.39 to 1.76) • at 30 minutes: • 0.74 (95% CI -1.34 to -0.14) • at 45 minutes: -1.28 (95% CI -1.89 to -0.69) • at 60 minutes: -2.20 (95% CI -2.61 to -1.78)	Respiratory depression: • 26/46 patients who received ziprasidone • 7 required supplemental oxygen • no difference in proportion with respiratory depression (p=0.26) or supplemental oxygen (p=0.20) when compared to midazolam and droperidol • no patients required intubation for respiratory depression Akathisia: • 4/46 patients who received ziprasidone Cardiac dysrhythmias: • none

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Ziprasidone					
Klein et al[103] (2018)	Ziprasidone 20 mg IM • prospective, observational Compared to: • midazolam 5 mg • olanzapine 10 mg IM • haloperidol 5 mg IM • haloperidol 10 mg IM	ED patients receiving medication to treat acute agitation 145 patients in ziprasidone arm • median age: 40 • 101 male/44 female • cause of agitation: alcohol (90%)/illicit substance (17%)/psychiatric illness (9%)/medical (1%)	Primary endpoint was adequate sedation, defined as Altered Mental Status Score <1 15 minutes after medication administration • ziprasidone 20 mg IM inferior to midazolam 5 mg IM (18% lesser for ziprasidone: 95% CI 29% lesser to 6% lesser) • ziprasidone 20 mg IM not different from olanzapine 10 mg IM (8% lesser for ziprasidone: 95% CI 19% lesser to 3% greater) • ziprasidone 20 mg IM superior to haloperidol 5 mg IM (12% greater for ziprasidone: 95% CI 1% greater to 23% greater) • ziprasidone 20 mg IM not different from haloperidol 10 mg IM (10% greater for ziprasidone: 95% CI 0% difference to 21% greater) Median difference in AMSS score compared to baseline at 15 minutes: • ziprasidone 20 mg IM no different than midazolam 5 mg IM (1 point lesser decrease for ziprasidone: 95% CI 1.5 point lesser decrease to 0.5 point greater decrease) • ziprasidone 20 mg IM no different than olanzapine 10 mg IM (0 point greater decrease for ziprasidone: 95% CI 0.5 point	Median time to adequate sedation: • ziprasidone 20 mg IM: 17 minutes	No difference in adverse events between groups Respiratory distress: • 1 to 3 patients in each arm with hypoxemia • 1 patient intubated in each arm except haloperidol 10 mg with no intubations Cardiovascular: • 1 to 2 patients in each group with hypotension except ziprasidone with no episodes of hypotension • 1 patient in each arm with bradycardia except midazolam with no episodes of bradycardia • no patients in any arm with torsades de pointes or other dysrhythmias Extrapyrimadal symptoms: • 2 patients in haloperidol 10 mg arm with dystonia. No other dystonic reactions in any arm

			greater decrease to 0.5 point lesser decrease) • ziprasidone 20 mg IM superior to haloperidol 5 mg IM (1 point greater decrease for ziprasidone: 95% CI 1.5 point greater decrease to 0.5 point greater decrease) • ziprasidone 20 mg IM superior to haloperidol 10 mg IM (1 point greater decrease for ziprasidone: 95% CI 1.5 point greater decrease to 0.5 point greater decrease) Time to adequate sedation (compared to midazolam 5 mg IM): • ziprasidone 20 mg IM inferior (HR 0.78: 95% CI 0.61 to 0.93) Time to adequate sedation for subset receiving monotherapy and no rescue sedation medications (compared to midazolam 5 mg IM): • ziprasidone 20 mg IM inferior (HR 0.64: 95% CI 0.48 to 0.82)		• no episodes of akathisia in entire study

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Ziprasidone					
Martel et al[108] (2020)	Ziprasidone 10 mg IM • prospective, randomized, double-blind trial • rescue sedation at treating physician discretion permitted 30 minutes after study drug administration for AMSS >0 Compared to: • droperidol 5 mg IM • ziprasidone 20 mg IM • lorazepam 2 mg IM	ED patients with acute undifferentiated agitation requiring emergent sedation as determined by the treating physician 28 patients in ziprasidone 10 mg group • median age: 40 • 19 male/9 female • initial median AMSS scale score of 3 • initial median BARS score of 6 • initial assessment of reason for agitation: alcohol intoxication (19), drug intoxication (2), head injury (3), and primary psychiatric etiology (5). • final diagnoses: acute alcohol intoxication (22), acute drug intoxication (4), head injury (8), psychiatric disease (4), and other (2)	Primary outcome was adequate sedation at 15 minutes • a lesser proportion of ziprasidone 10 mg compared to droperidol treated patients met the primary outcome: 39% lower (95% CI 14% to 64%) while ziprasidone 10 mg did not differ from either lorazepam or the higher dose of ziprasidone • lorazepam: 15/31 • droperidol: 16/25 • ziprasidone 10 mg: 7/28 • ziprasidone 20 mg: 11/31 AMSS scores were obtained every 15 minutes from time 0 to 120 minutes following study medication administration with median AMSS for ziprasidone 10 mg at: • 15 minutes: 1 • 30 minutes: 0 • 45 minutes: -1.5 • 60 minutes: -1.5 Additional sedation was required: • 4/28 before adequate sedation achieved • 7/28 in entire encounter • at a median time of 46 minutes following the initial administration	The post-administration assessment of adequate sedation occurred every 15 minutes post administration. The proportion achieving this endpoint at each check for ziprasidone 10 mg were: • 15 minutes: 7/28 • 30 minutes: 16/28 • 45 minutes: 22/28 • 60 minutes: 24/28	Respiratory depression was greater in both ziprasidone groups along lorazepam with compared to droperidol (p=0.04); for ziprasidone 10 mg: • 2/28 with hypoxemia (SpO_2<90%) • 9/28 with change in $ETCO_2$ • 10/28 with respiratory depression No patients in the ziprasidone 10 mg group required intubation Median QTc: 410 ms • no dysrhythmias in ziprasidone 10 mg group No patients in ziprasidone 10 mg group experienced dystonia

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Antipsychotics					
Ziprasidone					
Martel et al[108] (2020)	Ziprasidone 20 mg IM • prospective, randomized, double-blind trial • rescue sedation at treating physician discretion permitted 30 minutes after study drug administration for AMSS >0 Compared to: • droperidol 5 mg IM • ziprasidone 10 mg IM • lorazepam 2 mg IM	ED patients with acute undifferentiated agitation requiring emergent sedation as determined by the treating physician 31 patients in ziprasidone 20 mg group • median age: 41 years • 24 male/17 female • initial median AMSS scale score of 3 • initial median BARS score of 7 • initial assessment of reason for agitation: alcohol intoxication (25), drug intoxication (4), head injury (5), and primary psychiatric etiology (4). • final diagnoses: acute alcohol intoxication (25), acute drug intoxication (3), head injury (7), psychiatric disease (5), and other (3)	Primary outcome was adequate sedation at 15 minutes • lesser proportion of ziprasidone 20 mg compared to droperidol treated patients met the primary outcome: 29% lower (95% CI 3% to 54%) while ziprasidone 20 mg did not differ from either lorazepam or the lower dose of ziprasidone • lorazepam: 15/31 • droperidol: 16/25 • ziprasidone 10mg: 7/28 • ziprasidone 20 mg: 11/31 AMSS scores were obtained every 15 minutes from time 0 to 120 minutes following study medication administration with median AMSS for ziprasidone 20 mg at: • 15 minutes: 2 • 30 minutes: -1 • 45 minutes: -1 • 60 minutes: -2 Additional sedation was required: • 4/31 before adequate sedation achieved • 5/31 in entire encounter • at a median time of 38 minutes following the initial administration	The post-administration assessment of adequate sedation occurred every 15 minutes post administration; the proportion achieving this endpoint at each check for ziprasidone 20 mg were: • 15 minutes: 11/31 • 30 minutes: 22/31 • 45 minutes: 24/31 • 60 minutes: 25/31	Respiratory depression was greater in both ziprasidone groups along lorazepam with compared to droperidol (p=0.04); for ziprasidone 20 mg: • 6/31 with hypoxemia (SpO_2<90%) • 10/31 with change in $ETCO_2$ • 12/31 with respiratory depression One patient in the ziprasidone 20 mg group exhibited persistent agitation and required intubation for management of a subdural hematoma Median QTc: 428 ms • no dysrhythmias in ziprasidone 20 mg group One patient in ziprasidone 20 mg group experienced dystonia

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines plus Antipsychotics					
Midazolam plus droperidol					
Isbister et al[101] (2010)	Midazolam 5 mg plus Droperidol 5 mg IM • blinded, randomized controlled trial • further sedation allowed at discretion of attending physician Compared to: • midazolam 10 mg IM • droperidol 10 mg IM	ED patients requiring physical restraint and parenteral sedation 29 patients in droperidol plus midazolam group • median age: 30 • 15 male/14 female • initial assessment of agitation due to: alcohol intoxication (19), self-harm (9), drug-induced delirium (3), acute psychosis (2), and other (1)	Primary sedation outcome was time security staff were required according to a security log from the time of initial call to the "all clear." • duration was not different between groups (p=0.66) with median for: midazolam (20 minutes), droperidol (24 minutes), and midazolam plus droperidol (25 minutes) Secondary sedation outcomes were: • time additional sedation was administered: the hazard ratio for additional sedation medications for midazolam versus droperidol was 2.31 (95% CI 1.01 to 4.71; post prob 0.98 for HR>1.0) indicating that midazolam was more likely to require additional sedation compared to droperidol	Secondary outcome of reduction in AMSS by 3 points or to a score of <1 20 minutes after drug administration • midazolam plus droperidol: 23/29	Respiratory events occurred in: • midazolam plus droperidol: 1/29 involving desaturation events (1) plus airway obstruction (1) Hypotension occurred in: • midazolam plus droperidol: 1/29 Abnormal QT-HR pairs occurred in: • midazolam plus droperidol: 4/29 No dystonic reactions were identified Although oversedation was not a secondary endpoint, AMSS scores revealed that both midazolam and midazolam plus droperidol resulted in unpredictable and oftentimes deep sedation while droperidol resulted in consistent moderate sedation

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines plus Antipsychotics					
Midazolam plus haloperidol					
O'Connor et al[146] (2019)	Midazolam 2 mg to 4 mg plus haloperidol 5 mg IM • retrospective chart review • midazolam plus haloperidol group was not separated from lorazepam plus haloperidol group for analysis (unit of analysis was benzodiazepine plus haloperidol) Compared to: • ketamine 4 mg/kg IM per protocol with 3.68 mg/kg mean administered dose	Prehospital patient with standing order administered for combative or agitated behavior 68 patients in benzodiazepine plus haloperidol group • mean age: 35.4/median age 34 • male (69.1%)/female (30.9%) • co-ingestions: alcohol (39.7%)/cannabis (7.4%)/cocaine (10.3%)/opioids (16.1%)/other (14.7%)/none (10.3%)/unknown (26.5%) • trauma (13.2%)	No measure of adequate sedation Benzodiazepine plus haloperidol group less likely to require additional chemical restraint than ketamine (25% versus 49.5%; OR for ketamine 2.94, 95% CI, 1.49 to 5.80)	Not reported	Intubation rate • benzodiazepine plus haloperidol (1.5%) • ketamine (11.6%) • for intubation following ketamine, OR=8.77 (95% CI, 1.10 to 69.68) • indication for intubation in benzodiazepine plus haloperidol group: refractory agitation (1)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines plus Antipsychotics					
Lorazepam plus haloperidol					
Battaglia et al[107] (1997)	Lorazepam 2 mg plus haloperidol 5 mg IM • randomized and double blind • repeat doses allowed but not until after the first post-treatment standardized evaluation at 1 hour Compared to: • lorazepam 2 mg IM • haloperidol 5 mg IM	ED patients with psychosis and behavioral dyscontrol (agitated, aggressive, destructive, assaultive, or restless behavior) to the extent that they were capable of harming themselves or others. 32 ED patients in the lorazepam plus haloperidol group • 25 male/7 female • mean age 34.4 years • mean weight 74.6 kg • final diagnoses were mania, psychoactive substance abuse, psychosis not otherwise specified, schizophrenia, and schizophreniform disorder	Agitation was assessed serially using the Agitated Behavior Scale with a significant reduction in agitation from baseline at 1 hour in the haloperidol arm. The reduction in agitation seen with combination therapy was greater than lorazepam alone (p=0.014) but not greater than haloperidol alone (p=0.064) Approximately 10% of patients in the combination group were asleep at 1 hour, significantly more than the haloperidol alone group and similar to the lorazepam alone group	Serial evaluations occurred for 12 hours with redosing allowed after reevaluations. Only 1-hour endpoints were abstracted as they are most relevant to this review	11 combination therapy patients (34%) reported adverse effects: • ataxia: 3 (9%) • dizziness: 2 (6%) • dry mouth: 3 (9%) • EPS symptoms: 2 (6%) • speech disorder: 3 (9%) "No serious side effects" were reported

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines plus Antipsychotics					
Lorazepam plus haloperidol					
Riddell et al[99] (2017)	Combination of lorazepam (mean dose 2 mg) IV/(mean dose 2 mg) IM plus haloperidol (mean dose 5 mg) IM • prospective, observational Compared to: • midazolam (mean dose 3.08 mg) IV/(mean dose 2.25 mg) IM/(mean dose 2 mg) IN • lorazepam (mean dose 1.9 mg) IV/ (mean dose 2.4 mg) IM • haloperidol (mean dose 5.71 mg) IM • ketamine (mean dose 0.87 mg/kg) IV/(mean dose 2.97 mg/kg) IM	Acutely agitated patients requiring chemical sedation in the ED 10 patients in the combination lorazepam plus haloperidol group • median age: 40.5 • 9 male/1 female • race: African American (1)/Asian (0)/Hispanic (7)/White (2) • drug use: 60.0% • alcohol use: yes (20.0%)/no (20.0%)/unknown (60.0%) • prior psychiatric visits (50.0%) • route of administration: lorazepam IV plus haloperidol IM (5)/lorazepam IM plus haloperidol IM (5)	Primary outcome: agitation score less than or equal to 2 on a six-point agitation scale • recorded prior to medication administration then at 5, 10, and 15 minutes • combination of lorazepam plus haloperidol (and other arms) inferior to ketamine at: 5 minutes ($p=0.001$), 10 minutes ($p<0.001$), and 15 minutes ($p=0.032$) Secondary outcomes of: • provider assessment of time to adequate sedation: No difference between groups ($p=0.107$) • need for redosing of sedative medications ($p=0.199$) • HR/SBP change: HR reduction seen with midazolam ($p=0.026$) but no other significant HR/SBP changes in any other study arms	Mean time to adequate sedation: • combination of lorazepam 2 mg IV/IM plus Haloperidol 5 mg IM: 23.3 minutes	Intubation: • combination lorazepam plus haloperidol: 1/10

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines plus Antipsychotics					
Lorazepam plus haloperidol					
O'Connor et al[146] (2019)	Lorazepam 2 mg to 4 mg plus haloperidol 5 mg IM • retrospective chart review • lorazepam plus haloperidol group was not separated from midazolam plus haloperidol group for analysis (unit of analysis was benzodiazepine plus haloperidol) Compared to: • ketamine 4 mg/kg IM per protocol with 3.68 mg/kg mean administered dose	Prehospital patient with standing order administered for combative or agitated behavior 68 patients in benzodiazepine plus haloperidol group • mean age: 35.4/median age 34 • male (69.1%)/female (30.9%) • co-ingestions: alcohol (39.7%)/cannabis (7.4%)/cocaine (10.3%)/opioids (16.1%)/other (14.7%)/none (10.3%)/unknown (26.5%) • trauma (13.2%)	No measure of adequate sedation Benzodiazepine plus haloperidol group less likely to require additional chemical restraint than ketamine (25% versus 49.5%; OR for ketamine 2.94, 95% CI 1.49 to 5.80)	Not reported	Intubation rate • benzodiazepine plus haloperidol (1.5%) • ketamine (11.6%) • for intubation with ketamine, OR=8.77 (95% CI 1.10 to 69.68) • indication for intubation in benzodiazepine plus haloperidol group: refractory agitation (1)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Benzodiazepines plus Antipsychotics					
Lorazepam plus haloperidol					
Lin et al[126] (2020)	Lorazepam 2 mg IM or IV plus haloperidol 10 mg IM or IV • prospective randomized open-label study Compared to: • ketamine 4 mg/kg IM or 1 mg/kg IV	ED patients with combative agitation 49 patients in the haloperidol plus lorazepam group • median age 45 • 28 male/21 female • -25 White/9 Black/10 Hispanic • median HR: 100 bmp • median BP: 134/79 • 67% with psychiatric condition • 45/49 received IM medication	Primary outcome of adequate sedation at 5 minutes defined as RASS<1 • ketamine group (22%) more likely than haloperidol plus lorazepam group (0%) to meet endpoint (p=0.001) • secondary outcome of • median RASS at 30 minutes • lower in ketamine group (-1) versus haloperidol plus lorazepam (0) (p=0.02) Median time to sedation shorter in ketamine group (15 minutes) versus haloperidol plus lorazepam (36.5 minutes) (p<0.001) Greater proportion in ketamine group (66%) meeting sedation endpoint at 15 minutes versus haloperidol plus lorazepam (7%) (p<0.001) No difference in additional sedative medications required within 30 minutes (p=0.824): • ketamine (22%) • haloperidol plus lorazepam (20%)	Median time to sedation: • lorazepam 2 mg plus haloperidol 10 mg IV/IM: 36.5 minutes	Hypertension Δ > 20 mmHg • haloperidol plus lorazepam: 4/35 Tachycardia Δ >10 bpm • haloperidol plus lorazepam: 4/35 Hypoxia (SpO_2<92%) • haloperidol plus lorazepam: 3/42 • 1 patient was intubated QTc >450 ms • Haloperidol plus lorazepam: 11/22 • 1 patient experienced an arrythmia 1 patient in the haloperidol plus lorazepam group experienced bradycardia, hypoxia, cardiac arrest, and subsequent death deemed possibly related to the study medication

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Burnett et al[139] (2012)	Ketamine 5 mg/kg IM • dose received 3.1 to 7.4 mg/kg • case series	13 Patients given ketamine for agitation in the EMS environment	Adequate sedation • all patients with RASS of -1 or lower at hospital arrival	Mean time to peak sedation: • ketamine 5 mg/kg IM: 3.3 minutes in 11 patients and 20 minutes in 2 patients	3 patients with hypoxia 2 patients required intubation
Ho et al[140] (2013)	Ketamine • case reports	Case #1 – 500 mg IM ketamine (4.85 mg/kg) for patient with agitated behavior Case #2 – 375 mg IM ketamine (4.68 mg/kg) for patient in altercation with law enforcement	Sedation noted by treating paramedics and physicians	Case #1 – 4 minutes Case #2 – 3 minutes	Case #1 – intubated in the ED, discharged 96 days later Case #2 – intubated in ED, discharged 72 hours later
Scheppke et al[141] (2014)	Ketamine 4 mg/kg IM • followed by optional midazolam 2 mg to 2.5 mg IV/IO or IM to prevent emergence reaction after IV established • retrospective chart review/large case series	52 prehospital patients treated with ketamine for violent, aggressive behavior secondary to a psychiatric or substance-abuse issue.	"Medical control" is an adequate level of sedation to allow standard transport and treatment without further violence or agitation. • suitable sedation achieved in 96% of cases	Mean time to effective sedation and medical control: • ketamine 4 mg/kg IM: 2 minutes	5.8% of patients with respiratory depression • all patients with respiratory depression received midazolam 3.8 % of patients required intubation

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Keseg et al[142] (2015)	Ketamine 4 mg/kg IM, or 2 mg/kg IV • retrospective cohort chart review study No comparison group	36 prehospital patients given ketamine for sedation • male: 77% • median age: 29 years • African American (43%)/Caucasian (34%)/Hispanic (2.9%)/unavailable (20%) • reason for ketamine administration: agitation (16%)/combative (14%)/intubation (2.9%)/hostile (2.9%)/violent (2.9%)/excited delirium (2.9%)/suicidal with weapon (2.9%) • 29 IM only injections	Primary endpoint was "improved condition" as defined by treating EMS personnel • 91% (95% CI 77% to 98%) with improved condition Secondary endpoint of administration of additional sedation methods (benzodiazepines or significant physical force) • 40% (95% CI 24% to 58%) with administration of additional sedation methods	Not reported	8/35 (23%) of patients intubated with indications for intubation of: • agitation (4) • lethargic (2) • unresponsiveness (1) • cardiac arrest prior to ketamine administration (1)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Cole et al[118] (2016)	Ketamine 5 mg/kg IM (median dose received 5.2 mg/kg) • prospective open label EMS study Compared to: • haloperidol 10 mg IM	64 acute undifferentiated agitation with AMSS +3 (57 patients) to +2 (7 patients). AMSS +4 excluded as "profound agitation" • median age 36 years • 37 male/27 female • 31 Caucasian, 16 black American, 7 American Indian, 3 Somali, 2 Hispanic, 1 Asian, 4 mixed/unknown • 48 (75%) with history of mental illness, 30 (47%) with history of chemical dependency, and 25 (39%) with both • EMS impressions of: agitated combative (29), substance abuse (7), behavioral (16), AMS (2), and trauma (4)	Primary endpoint of AMSS < +1. • 61/64 patients achieved adequate sedation • 3/64 patients required second injection prehospital: midazolam (1), ketamine IM (1), and ketamine IV (1) Compared to the group receiving haloperidol, 30% more patients in the ketamine group successfully achieved adequate sedation (p<0.0001, 95% CI 18% to 42%) Time to sedation was 12 minutes less for ketamine group compared to the haloperidol group (p<0.0001, 95%CI 9 to 15 minutes)	Median time to adequate sedation: • ketamine 5 mg/kg IM: 5 minutes	38 complications occurred in 27/55 patients where complications recorded: hypersalivation (21), emergence reaction (5), vomiting (5), dystonia (3), laryngospasm (3), and akathisia (1); there were no deaths in the ketamine group • complications occurred in 44% more patients in the ketamine group compared to the haloperidol group (p<0.0001, 95% CI 30% to 57%) Intubation occurred in 25/64 patients for the following indications: not protecting airway (8), hypersalivation (4), refractory agitation (3), apnea (3), aspiration/vomiting (3), laryngospasm (2), seizure (1), and trauma (1) • intubation occurred in 35% more patients in the haloperidol group compared to the ketamine group (p<0.0001, 95% CI 23% to 48%)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Olives et al[143] (2016)	Ketamine 5 mg/kg IM • retrospective cohort No comparison group	135 patients given ketamine prehospital for agitation	Emergency Medical Service providers reported initial improvement in agitation following ketamine administration • 124/135 (91.8%) • no change in 9/135 (6.7%) • worsened agitation in 2/135 (1.5%)	Not reported	85 patients (62%) intubated: • 74.6% patients during overnight shift versus 55% of daytime encounters (p=0.21) • arrival during night shift associated with intubation, adjusted OR 2.57 (95% CI 1.05 to 6.27) • dose intubated (5.25 mg/kg) not different than not intubated (5.14 mg/kg) (p=0.68) Cardiac arrest after ketamine administration in 2 patients: • neither due to ketamine administration
Scaggs et al[35] (2016)	Prehospital ketamine for agitation 5 mg/kg IM or 1.5 mg/kg IV • case series • mean dose of ketamine received: 4.36 mg/kg No comparison group	7 patients given prehospital ketamine for excited delirium • mean age: 24 years • CK: 484.33 • HR: 158 bpm	Skaggs Scale (modified RASS)	Range of reported time to adequate sedation for ketamine IM 5 mg/kg: 1.5 to 2 minutes	1 patient with hypoxia 1 patient with rhabdomyolysis

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Parsch et al[155] (2017)	Ketamine, studied retrospectively pre and post guideline adoption • retrospective cohort study	Mental health patients with acute behavioral disturbance requiring transport: 28 patients receiving ketamine post guideline change • median age: 34 • 26 men/2 women • transport duration: 175 minutes	Need for intubation as a proxy for adequate sedation • 36% intubated before protocol • 7.14% intubated after protocol • OR 0.14 (for post protocol intubation	Not reported	1 patient on a ketamine and propofol infusion suffered a presumed episode of laryngospasm in flight, manifested by a soft stridor; no specific airway intervention was required and the stridor resolved within a few minutes No episodes of hypoxia, nausea, vomiting, aspiration or cardiovascular compromise were observed during the retrievals

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Riddell et al[99] (2017)	Ketamine (mean dose 0.87 mg/kg) IV/(mean dose 2.97 mg/kg) IM • prospective, observational cohort study Compared to: • midazolam (mean dose 3.08 mg) IV/(mean dose 2.25mg) IM/(mean dose 2 mg) IN • lorazepam (mean dose 1.9 mg) IV/ (mean dose 2.4 mg) IM • haloperidol (mean dose 5.71 mg) IM • combination of lorazepam (mean dose 2 mg) IV/(mean dose 2 mg) IM plus haloperidol (mean dose 5 mg) IM	Acutely agitated patients requiring chemical sedation in the ED 24 patients in the ketamine group • median age: 29 • 19 male/5 female • race: African American (3)/Asian (1)/Hispanic (10)/White (10) • drug use: 54.2% • alcohol use: yes (33.3%)/no (52.2%)/unknown (17.4%) • prior psychiatric visits (30.4%) • route of administration: ketamine IV (18)/ketamine IM (6)	Primary outcome: agitation score less than or equal to 2 on a six-point agitation scale • recorded prior to medication administration then at 5, 10, and 15 minutes • ketamine superior to other arms at: 5 minutes (p=0.001), 10 minutes (p<0.001), and 15 minutes (p=0.032) Secondary outcomes of: • provider assessment of time to adequate sedation: No difference between groups (p=0.107) • need for redosing of sedative medications (p=0.199) • HR/SBP change: HR reduction seen with midazolam (p=0.026) but no other significant HR/SBP changes in any other study arms	Mean time to adequate sedation: • ketamine: 6.57 minutes	Intubation: • ketamine: 2/24

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Cole et al[144] (2018)	Ketamine 5 mg/kg IM • prospective observational cohort study No comparison group	EMS patients transported to single urban ED with EMS clinically identified behavioral emergency and AMSS of +4 49 patients received ketamine • median age: 29 • 76% male/24% female • 49% Caucasian/35% Black American/6% American Indian/2% Hispanic/2% Somali/6% unknown or mixed • EMS impressions: agitated combative (23)/behavioral (14)/substance abuse (4)/AMS (3)/Trauma (3)/Seizure • median dose received: 4.9 mg/kg	Primary endpoint was time to adequate sedation defined as AMSS <+1 • adequate sedation prehospital: 90% Secondary endpoint of additional EMS sedatives	Median time to sedation: • ketamine 5 mg/kg IM: 4.2 minutes	Intubation in ED: 57% (over 1/3 of intubations performed by a single ED physician) • indications for intubation: airway unprotected (10)/hypersalivation (5)/respiratory failure (4)/hemodynamic instability or acidosis (3)/failure to treat agitation (2)/ ”expected return of anticipated behavior” (2)/status epilepticus (1)/hypoxia (1) Adverse events: • hypersalivation (18%) • vomiting (6%) • emergence reaction (2%)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Heydari et al[119] (2018)	Ketamine 4 mg/kg IM • if patient was inadequately sedated or additional medication needed per physician discretion then 2 mg/kg repeat dose allowed • randomized, double blind prospective trial Compared to: • haloperidol 5 mg IM	ED patients with acute agitated and aggressive behavior who required chemical sedation for agitation, according to an emergency medicine resident or attending physician were eligible for enrollment. (AMSS +2 or +3) 45 patients in ketamine group: • mean age of 30.37 • male: 73.3%/female: 26.7% • cause of aggressive behavior: psychotropic substances (26.7%)/psychiatric history (28.9%)/alcohol consumption (26.7%)/trauma (17.8%)	The primary outcome was time to adequate sedation (AMSS≤+1) • faster for ketamine compared to haloperidol (p<0.01, difference 3.7 minutes, 95% CI 2.1 to 5.5) Mean AMSS scores: • 5 minutes: ketamine (1.36) was not different from haloperidol (1.70) (p=0.115) • 10 minutes: ketamine (0.67) was higher than haloperidol (1.27) (p=0.001) • 15 minutes: ketamine (0.14) was not different from haloperidol (0.3) (p=0.167) • proportion not adequately sedated at 15 minutes was lower in ketamine group (6.7%) than haloperidol group (28.9%) • difference of 22% (95% CI 11% to 33%; p<0.0001) Physician satisfaction was higher in ketamine group than haloperidol group (p=0.011)	Median time to adequate sedation • ketamine 4 mg/kg IM: 7.73 minutes	Complications: 35.6% for ketamine 17.8% for haloperidol no significant difference between groups (p=0.094, difference 17%, 95% CI 11% to 22%). Ketamine group: • hypersalivation (n=5, 11.1%) • vomiting (n=6, 13.3%) • Laryngospasm (n=2, 4.4%) • Emergence phenomena (n=3, 6.7%) • Intubation (n=6 13.3%) Primary indications for intubation in ketamine group were refractory agitation (n=1), hypersalivation (n=2), and hypoxia (n=3).

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Mankowitz et al[145] (2018)	Ketamine IV or IM • mean IM dose: 4.9 mg/kg • systematic review No comparison to other agents	650 patients receiving ketamine for agitation • ED (110)/air medical transport (61)/ground transport (479) • 67.6% male • mean age: 33 years	Proportion achieving sedation within 5 minutes • 68.5% (95% CI 61.7% to 75.3%) Proportion requiring further sedating medications beyond single dose of ketamine • 24.4% (95% CI 20.5% to 28.3%)	Mean time to adequate sedation: • ketamine: 7.21 minutes	Vomiting • 5.3% (95% CI 2.4% to 8.2%) hypertension • 12.4% (95% CI 5.8% to 18.9%) emergence delirium • 4.0% (95% CI 1.3% to 6.7%) transient hypoxia • 1.8% (95% CI 0.1% to 3.6%) laryngospasm • 1.3% (95% CI 0.3% to 2.3%) hypersalivation • 19% (95% CI 13.2% to 25%) Intubation • 30.5% (95% CI 27.0% to 34.1%)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Li et al[147] (2019)	Ketamine 2 mg/kg IM or 1 mg/kg IV • retrospective chart review after implementation of a ketamine guideline for excited delirium • per protocol, ketamine administered after combination of benzodiazepine and antipsychotic	ED patients being treated with ketamine for excited delirium 31 patients • mean age: 38.5 • male: 77.4% • 19 IM administration (mean initial dose: 3.6 mg/kg)	RASS scores • RASS decreased from +4 to 0 after ketamine (p=0.001) Post ketamine decrease in: • median SBP: 136 mm hg versus 126 mm hg (p=0.03) • median HR: 105 bpm versus 90 bpm (p=0.03)	Not reported	Six (19.4%) patients required intubation
O'Connor et al[146] (2019)	ketamine 4 mg/kg IM per protocol with 3.68 mg/kg mean administered dose • retrospective chart review • lorazepam plus haloperidol group was not separated from midazolam plus haloperidol group for analysis (unit of analysis was benzodiazepine plus haloperidol) Compared to: • Lorazepam 2 mg to 4 mg plus haloperidol 5 mg IM and midazolam 2 mg to 4 mg plus haloperidol 5 mg IM grouped together for analysis (unit of analysis was benzodiazepine plus haloperidol)	Prehospital patient with standing order administered for combative or agitated behavior 95 patients in ketamine group • mean age: 34.2/median age 33 • male (58.9%)/female (41.1%) • co-ingestions: alcohol (38.9%)/cannabis (4.2%)/cocaine (14.7%)/opioids (16.8%)/other (14.7%)/none (21.1%)/unknown (23.2%) • trauma (17.9%)	No measure of adequate sedation Ketamine group more likely to require additional chemical restraint than Benzodiazepine plus haloperidol group (49.5% versus 25%; OR for ketamine 2.94, 95% CI 1.49 to 5.80)	Not reported	Intubation rate • ketamine (11.6%) • benzodiazepine plus haloperidol (1.5%) • For intubation with ketamine, OR=8.77 (95% CI 1.10 to 69.68) • indications for intubation in ketamine group: refractory agitation (6); hypoxia/respiratory distress (2); airway protection (3)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Lebin et al[148] (2019)	Ketamine 1 mg/kg to 2 mg/kg IV or 3 mg/kg to 5 mg/kg IM • retrospective cohort study Compared to • midazolam 1 mg to10 mg IV, 5 mg to 10 mg IM, or 2.5 mg to 10 mg IN • diazepam 2.5 mg to10 mg IV	Patients with severe agitation requiring prehospital sedation with ketamine or benzodiazepine 59 patients in ketamine group • age: 33 • male (79.7%) • Caucasian (49.2%)/Black or African American (16.9%)/Asian (1.7%)/other or not reported (32.2%) • 56 patients received ketamine IM	Not reported	Not reported	Intubation • ketamine (3.8%) • benzodiazepine (63.0%) • 59.1% (95% CI 79.35% to 37.9%) less likely to be intubated after ketamine administration than benzodiazepine administration

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Holland et al[105] (2020)	Ketamine IM • dose per protocol: 150 mg to 300 mg (93/97 patients received 300 mg) • weight based dose: 3.75 mg/kg (95% CI 2.13 mg/kg to 5.37 mg/kg). • retrospective chart review Compared to: • Midazolam 5 mg IV/IM/IN	Patients with acute agitation requiring sedation by paramedics 97 patients in ketamine treated group • mean age of 33.8 years • 76 male/21 female • 46 White, 49 African-American, and 2 other • mean weight: 82.1kg • suspicion of illicit drugs: 74.2%	Primary endpoint was need for repeat sedative dose • 6/97 required repeat sedation at 20 minutes; no difference compared to midazolam (p=0.306) • 46/97 required repeat sedation at 90 minutes; significantly more than midazolam group (p=0.01) • when limiting the analysis to only sedation given via IM route, there was no difference in need for repeat sedation between midazolam and ketamine groups at 20 minutes (p=0.212) or 90 minutes (p=0.503) • secondary endpoints • time to repeat sedation of 77.2 minutes; no difference compared to midazolam group (p=0.658) • total number of sedation doses did not differ between ketamine and midazolam (p=0.084)	Need for repeat sedative dose at 20 minutes used as proxy for adequate control of agitation • 6/97 in ketamine group required repeat sedation	6 patients in the ketamine group were intubated; one patient was found to have an intracranial hemorrhage; another patient in the ketamine cohort received 6 more doses of sedatives before intubation, suggesting a limited impact of prehospital ketamine on the decision to ultimately intubate For patients administered ketamine, median GCS was 13 (IQR 11.25 to 15) prior to administration and 9 (IQR 3.25 to 11.75) after administration (p<0.0001); there was no significant difference compared to the change in GCS achieved with midazolam, p=0.4116) There were no significant differences in use of bag valve mask or intubation, use of physical restraints, admission location/level of care, or length of stay in the ED, hospital, or ICU

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Lin et al[126] (2020)	Ketamine 4 mg/kg IM or Ketamine 1 mg/kg IV • prospective randomized open-label study Compared to: • lorazepam 2 mg IM/IV plus haloperidol 10 mg IM/IV	ED patients with combative agitation 44 patients in the ketamine group • median age 37 years • 30 male/14 female • 29 White/4 Black/7 Hispanic • median HR: 110 bmp • median BP: 132/88 • 43% with psychiatric condition • 42/44 received IM medication	Primary outcome of adequate sedation at 5 minutes defined as RASS<1 • ketamine group (22%) more likely than haloperidol plus lorazepam group (0%) to meet endpoint (p=0.001) Median time to sedation shorter in ketamine group (15 minutes) versus haloperidol plus lorazepam (36.5 minutes) (p<0.001) Greater proportion in ketamine group (66%) meeting sedation endpoint at 15 minutes versus haloperidol plus lorazepam (7%) (p<0.001) Secondary outcomes: Median RASS at 30 minutes • lower in ketamine group (-1) versus haloperidol plus lorazepam (0) (p=0.02) No difference in additional sedative medications required within 30 minutes (p=0.824): • ketamine (22%) • haloperidol plus lorazepam (20%)	Median time to sedation: • ketamine 4 mg/kg IM: 15 minutes	Hypertension Δ >20 mmHg • ketamine: 13/39 Tachycardia Δ >10 bpm • ketamine: 13/38 Hypoxia (SpO_2<92%) • ketamine: 6/39 • 1 patient was intubated QTc >450 ms • ketamine: 11/23 • 1 patient experienced an arrythmia No deaths occurred in the ketamine group

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Parks et al[156] (2020)	Ketamine (mean dose 4.88 mg/kg) IM • 97.6% IM/2.4% IV • retrospective cohort/chart review No comparison group	86 patients receiving prehospital ketamine for agitation • mean age: 42.9 • female (54.7%)	Not reported	Not reported	14/86 (16.3%) of patients intubated • no difference in dose between intubated (4.44 mg/kg) and not intubated (4.96 mg/kg) patients (-0.53 mg/kg difference; 95% CI, -1.49 to 0.43; P=0.278) Adverse events: • abnormal lung sounds (6) • respiratory distress (8) • apnea (4) • vomiting (1) • hypersalivation (2)

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Cunningham et al[157] (2021)	Ketamine 4 mg/kg IM (standard dose) • pre-/post-intervention retrospective chart review Compared to: Ketamine 3 mg/kg IM followed by optional 2nd dose of 1 mg/kg IM (lower dose)	Prehospital patients treated for acute agitation 211 patients in standard dose group • mean age: 35.14/median age: 32 years • male 67.8%/female 32.2% • mean dose received: 3.51 mg/kg • trauma: 21.3%	Not reported	Not reported	Need for additional chemical restraint after 1st dose of ketamine: standard dose: 57.3% Intubation rate standard dose group: 14.2% • indications for intubation: hypoxia or respiratory distress (10)/refractory agitation (9)/airway protection (9)/facilitate imaging (1)/missing (1) Total adverse reaction standard dose group: 22.2%
Cunningham et al[157] (2021)	Ketamine 3 mg/kg IM followed by optional 2nd dose of 1 mg/kg IM (lower dose) • pre-/post-intervention retrospective chart review Compared to: Ketamine 4 mg/kg IM (standard dose)	Prehospital patients treated for acute agitation 81 patients in standard dose group • mean age: 35.65/median age: 31 • male 65.4%/female 34.6% • mean dose received: 3.24 mg/kg • trauma: 21.0%	In the lower dose cohort, adequate sedation without additional dosing was achieved in 79% (64/81) patients	Not reported	Need for additional chemical restraint after 1st dose of ketamine: lower dose: 57.3% Intubation rate lower dose group: 18.5% • indications for intubation: hypoxia or respiratory distress (6)/refractory agitation (5)/airway protection (4) Total adverse reaction lower dose group: 20.9%

Study & Year Published	Drug Studied	Population	Sedation Endpoint	Time to Reach Endpoint	Adverse Events
Ketamine					
Fernandez et al[93] (2021)	Ketamine (median dose 3.7 mg/kg for AMS/behavioral indications) IM • large retrospective analysis of prospectively collected prehospital registry No comparison group	11,291 prehospital ketamine administrations for any indication by any route 3,795 receiving ketamine for AMS/behavioral indications • age: 50% of patients were 20 to 39 years of age • female (34.1%)/male 65.9% • White (64.6%), Black (22.3%), other race (2.6%), Hispanic or Latino (10.4%)	Single administration of ketamine as a proxy for adequate sedation • one dose: 78.7%	Not reported	8 deaths in entire cohort of 11,291 (0.07%) administrations where ketamine could not be fully excluded as cause • 4 deaths in subgroup of 3,795 (0.1%) receiving ketamine for AMS/behavioral indications where ketamine could not be fully excluded as cause Respiratory events in subgroup receiving ketamine for AMS/behavioral indications • hypoxemia: 10.7% prior to and 10.2% after administration • hypoventilation ($EtCO_2$>45): 6.2% prior to and 23% after administration

AMS, altered mental status; *AMSS*, Altered Mental Status Scale; *BARS*, Behavioral Activity Rating Scale; *BPRS*, Brief Psychiatric Rating Scale; *CI*, confidence interval; *dl*, deciliter; *ED*, emergency department; *EMS*, emergency medical services; *EPS*, extrapyramidal symptoms; *GCS*, Glasgow coma scale; *GCI*, Global Clinical Impression; *HR*, heart rate; *HR/SBP*, heart rate/systolic blood pressure; *ICU*, intensive care unit; *IM*, intramuscular; *IN*, intranasal; *IQR*, interquartile ratio; *IV*, intravenous; *kg*, kilogram; *mg*, milligram; *ms*, millisecond; *OR*, odds ratio; *QTc*, corrected QT interval; *RASS*, Richmond Agitation-Sedation Scale; *SAT*, Sedation Assessment Tool.

Mark Kroll, PhD, FACC, FAIMBE

Research review: Naked, but dangerous

An encounter with a naked subject presents an unpredictable and dangerous call, although very rare

Jul 13, 2021

In March 2015, DeKalb County, Georgia, police officer Robert Wilson, was dispatched to check on a naked person (Anthony Hill) who was acting bizarrely and wandering around an apartment building.

When Officer Wilson spotted Hill, he stepped out of his patrol car to make contact. Hill ran directly at him at high speed flailing his arms. Officer Wilson stepped back toward the rear of his patrol car to create distance while yelling for Hill to stop. Hill did not stop and Officer Wilson discharged his firearm killing Hill.

Former Officer Wilson was criminally charged with many counts, including murder. The prosecution's use of force expert admitted that an electrical weapon deployment towards a charging person required a trick shot (since a probe would have to go into the thorax and the other into a thigh). Nevertheless, former officer Wilson was convicted of various charges and sentenced to 20 years in prison.

Researchers Darrell Ross and Michael Brave found 397 incidents of police responding to naked subjects occurring from 1998–2018. (Richmond, Va., Police via AP)

STUDY REVIEWS LE RESPONSE TO THE NAKED SUBJECT

Two well-recognized researchers on police use of force, Darrell Ross and Michael Brave, recently published a peer-reviewed study of police response to naked subjects. [1]

They found 397 incidents occurring from 1998–2018, averaging about 20 incidents annually, and then did a more detailed study of 215 incidents covered in Federal Court (42 U.S.C. § 1983 civil rights litigation) decisions from 1997 to 2019. This study shows how extremely dangerous such thankfully rare incidents can be.

Some of the percentages given below are from the larger (n=397) group and some are from the smaller legal-case group. (For details, you can request the actual paper here.)

In about 80% of the incidents, the subject charged, actively fought, or assaulted the responding law enforcement officer (LEO); in about 36% of the incidents, the person attempted to disarm the LEO. The subject was unarmed in 75% of the incidents, armed with a firearm in 15% and possessed an edged weapon in 10%. The subject was either mentally ill or on illicit drugs in 89% of the cases; and died in 86% of cases.

In the 22 deadly-force incidents the officers had limited reaction time, usually less than 10 seconds, from the beginning of the contact.

Overall, de-escalation techniques were attempted in 80% of the incidents but were never successful.

In the deadly-force incidents, the LEO had limited time to use an electrical weapon, an impact weapon, or an aerosol.

In about 20% of the incidents overall, intermediate weapons were ineffective, requiring LEOs to transition to deadly force based on the subject's assaultive nature.

About 80% of the subjects exhibited clear signs of profound agitation syndrome with 6 to 11 of the classic diagnostic factors such as unexpected strength, pain insensitivity, extreme stamina, sweating, non-compliance, hyperactivity, agitation, and incoherent speech. Because of the danger of these encounters, the authors recommend that a minimum of 2 (ideally 4) officers respond.

This study shows that an encounter with a naked subject presents an unpredictable and dangerous call, although very rare (1 in 10,000,000 citizen encounters). The courts granted summary judgment to LEOs in 70–75% of the cases, primarily based on the threat and danger posed by the subject. Higher levels of force were often used in response to the higher levels of violence and assaultive behaviors exhibited by the subjects who most were either mentally ill, under the influence of an illicit drug, or both.

POLICE TRAINING FOR NAKED SUBJECT CALLS

In the Implications section, the authors focused on policy and training. Since these incidents are very rare, agencies are encouraged to develop and use brief study and memory aides to assist LEOs with applying optimal force methods and procedures, and to document such incidents.

Some links for training and memory aids include:

- Institute for the Prevention of In-Custody Deaths (IPICD)
- Recognizing and managing abnormal breathing: LEO V 2.0. (with Learner Companion)
- Acute behavioural disturbance (ABD): guidelines on management in police custody
- TASER CONDUCTED ENERGY WEAPON (CEW) use guidelines
- Understanding the 4th Amendment's objective reasonableness standard and qualified immunity
- Types of CEW use study-aid/guideline table.

PREDICTORS OF A FATAL OUTCOME

The Ross and Brave study is consistent with the results of a recently published Dutch study that compared excited-behavior restraint deaths with non-fatal excited-behavior restraint incidents. [2] Full or partial nudity increased the risk of death by 4x and thus disrobing should be seen as an independent predictor of a fatal outcome. That, in turn, is consistent with recent studies that have shown that hyperthermia is associated with the most severe agitation cases. [3]

REFERENCES

1. Ross DL, Brave MA. Assessing Use-of-Force Liability and Law Enforcement Response to the Naked Subject. *Law Enforcement Executive Forum*. 2020;20(1):1-21.

2. Dijkhuizen LGM, Kubat B, Duijst W. Sudden death during physical restraint by the Dutch police. *J Forensic Leg Med.* 2020;72:101966.

3. Baldwin S, Hall C, Bennell C, Blaskovits B, Lawrence C. Distinguishing features of Excited Delirium Syndrome in non-fatal use of force encounters. *J Forensic Leg Med.* 2016;41:21-27.

About the author

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12 OCT 2021

Sidestepping the Excited Delirium Debate

BY VON KLIEM, JD, LL.M / FORCE SCIENCE NEWS / 2

Depending on who you ask, excited delirium syndrome (ExDS) is either a group of symptoms that warn of a life-threatening medical condition or it is a diagnosis invented by racist and abusive police to excuse murder.[1,2]

Among those that use the term ExDS, the medical consensus is that ExDS is not a unique disease but a group of symptoms with uncertain and varied causes.[3] Although drug intoxication is a common cause of ExDS, the World Health Organization includes in the ICD-10 more than 30 separate conditions that can

lead to symptoms associated with ExDS.

As mentioned in a previous article, and it's worth repeating here, "The varied causes of ExD, the overlap of its symptoms with other conditions, and its rare occurrence are just some of the reasons first responders are not expected to diagnose ExD. Even so, the absence of a specific diagnosis does not negate the seriousness of the behavioral and physical symptoms."[4]

Fortunately, first responders are not trained to wait for a specific medical diagnosis before responding to potentially life-threatening symptoms. Whether ExDS proves to be a valid diagnosis or not, the need for containment, rapid de-escalation, and medical intervention for people in an agitated state of delirium is well-settled.

Trying to Pull Focus

Law enforcement and EMS training continues to focus on the latest de-escalation strategies, physical restraint techniques, and chemical sedation options to get suspected ExDS patients to advanced medical care safely. Ideally, this training is informed by professional medical groups like the American Medical Association, the National Association of EMS Physicians, and the American College of Emergency Physicians.

Unfortunately, discussions that should be aimed at identifying the best emergency response protocols for suspected ExDS cases have instead become racialized and hyper-politicized. Conversations around ExDS have become volatile, divisive, and no longer reflect purely physiological or psychological considerations.[5]

Instead, added to the "excited delirium" discussion is the theory that ExDS was invented by "racist" police and corrupt medical organizations *for the purpose* of helping officers avoid accountability for excessive force and murder.[6] Proponents of this ahistorical and racialized view of ExDS must still contend with reports that observe, "By itself, ExDS carries an extremely high mortality risk, with approximately 2/3 of ExDS patients dying in the prehospital setting in the absence of any major trauma, physical restraint, *or police intervention* [emphasis added]."[7]

Staying Focused: The American College

of Emergency Physicians

Despite the distractions caused by injecting political and social justice theories into ExDS discussions, first responders and emergency medical personnel still needed to know how to treat delirious, agitated, and combative patients effectively and safely.

In 2009, The American College of Emergency Physicians (ACEP) took the lead in therapeutic options and best practices for ExDS patient care and survival. Although not the first medical organization to formally recognize ExDS, ACEP has been at the forefront of ExDS discussions since publishing the *White Paper Report on Excited Delirium Syndrome*.[8]

In its 2009 paper, the ACEP Task Force identified the features of ExDS and those characteristics that were present in cases where the subject died. They provided expert insight into developing ExDS response protocols, including prioritizing verbal calming and de-escalation techniques. If those techniques failed, ACEP recommended rapid physical control measures that minimized the time patients spent struggling. When medically justified, ACEP recommended aggressive chemical sedation to facilitate evaluation and transport to advanced medical care.

Allegations of Bias, Conflict of Interest

Following ACEP's publication of their ExDS report, police critics continued to question the existence of ExDS and predictably started to question the motives of ACEP itself.

For context, ACEP represents tens of thousands of physicians from 53 chapters, including all 50 states, Puerto Rico, the District of Columbia, and Government Services. The 2009 ACEP Task Force consisted of 19 members who reported to the Board of Directors and Council, with Council members representing all 53 chapters. The Board of Directors and Council approved the report for publication.

Still, concerns about bias in the report were raised when critics learned that 3 of the Task Force members provided expert medical consultation and litigation support to Axon (manufacturer of TASERs). Two of these medical consultants were

also employed as part-time police officers, relationships that were disclosed in the 2009 report.

Bias is certainly a fair question to investigate. However, critics of ExDS were still forced to contend with the scholarly research and experience of ACEP and its highly credentialed Task Force members. ACEP had convincingly argued that ExDS was a medical emergency and that response protocols were critically needed.

Sidestepping the Debate: The American College of Emergency Physicians

In 2021, ACEP returned to the ExDS conversation. Responding to questions from "its membership, the scientific community at large, community leaders, media, and governmental agencies," ACEP published another Task Force Report. This June 2021 report again focused on the prehospital evaluation, treatment, and management of patients who are delirious and agitated to the point where they cannot be safely or reliably evaluated.[9]

To sidestep the ExDS debate, ACEP took several important steps. First, their most recent study was conducted *de novo*, meaning they took a fresh look at the evidence. They were informed by the 2009 paper but did not approach their latest study as an update or refutation of the previous research.

Next, ACEP executed what they described as a "robust global conflict of interest policy." This policy was intended to identify, disclose, and avoid conflicts or potential bias in its members. The conflict-of-interest policy was "not a direct response to critics of the 2009 white paper nor with specific concerns regarding the content of that paper." Even so, the persuasive impact from executing the conflict-of-interest policy was bolstered by the fact that none of the 2009 Task Force members were members of the 2021 Task Force or review panel.

Finally, ACEP publicly recognized the political controversy surrounding the term "Excited Delirium Syndrome." To avoid potential distractions and maintain focus on patient care and survival, ACEP chose to sidestep the issue again and simply refer to ExDS by the more descriptive terminology, "hyperactive delirium with severe agitation."

Problem solved.

The Task Force Report

Behind the brilliant ACEP pivot is an important report: *The ACEP Task Force Report on Hyperactive Delirium with Severe Agitation in Emergency Settings, June 23, 2021*.

The full report focuses on the evaluation, initial treatment, and effective medical interventions (chemical sedation options). Although the report was expressly written for EMS professionals and medical staff, law enforcement and other first responders will undoubtedly benefit from the insights.

Highlights from the report:

- "[A patient experiencing hyperactive delirium with severe agitation] needs rapid de-escalation and calming to allow for definitive medical evaluation and ongoing treatment, in order to avoid preventable fatality due to failure to manage the potential causative life threats, and to treat the danger inherent to the presenting condition."

- "Hyperactive delirium with severe agitation is a life-threatening constellation of signs and symptoms with numerous causes …. The combination of [symptoms] raises serious concerns for rapid physiologic deterioration and death particularly in patients with underlying comorbidities (e.g., coronary artery disease, obesity, asthma)."

- "There are risks associated with empiric treatment [experience-based treatments] for a presumptive diagnosis in all aspects of medicine; nevertheless, such an approach is required when the patient's clinical condition necessitates the need for resuscitative interventions prior to a definitive diagnosis.

- "We strongly recommend that the urgency of intervention not inadvertently exclude simple, effective therapies. In a recent large, preliminary analysis of

patients in law enforcement custody who were documented as combative and required an EMS response, non-pharmacologic intervention was all that was required in over 80% of cases. In nearly all cases, non-pharmacologic interventions may be attempted, *even if in parallel with preparations for pharmaceutical administration* [emphasis added]."

- "[T]he circumstances in which severely agitated patients are encountered may require immediate utilization of pharmacologic and physical interventions, but in many scenarios, it is still feasible to attempt verbal and non-verbal de-escalation initially.

- "It appears [verbal and non-verbal de-escalation] may be most effective when provided within a structured format, likely enhanced by assignment of specialized teams. The failure of these de-escalation techniques may indicate a much more severe form of agitation only amenable to treatment with sedating medications."

Sedation Not Recommended for Purely Law Enforcement Purposes

It is expected that law enforcement officers may be the first to arrive in cases involving patients experiencing hyperactive delirium with severe agitation. Even so, a dual LEO/EMS response is ideal, with de-escalation and restraint efforts conducted in consultation with on-scene emergency medical personnel.

It's worth repeating, to increase patient survivability, ACEP recommends that physical control measures be selected to minimize the patient's exertion and time spent struggling. Physical control should be immediately followed by continuous medical assessment and treatment—including chemical sedation when medically justified.

Both the 2009 and the 2021 ACEP reports support sedation *when justified for medical reasons and to facilitate medical treatment*.

The 2009 report recommends sedation when necessary for medical assessments

The 2009 report recommends sedation when necessary for medical assessments and intervention. It further recommends that chemical sedation "proceed concurrently with evaluation for precipitating causes or additional pathology."

The 2021 report recommends that medical treatment of hyperactive delirium with severe agitation be led by a physician board-certified in EMS Medicine or Emergency Medicine and that appropriately trained medical professionals on a physician-led care team decide prehospital medical treatments.

Neither report should be read to justify sedation for purely law enforcement purposes.

Read the Full ACEP 2021 Report Below.

VIEW FULL REPORT

Research Force Science News Excited Delirium Articles Below.

VIEW ARTICLES

1. See Byju, A.S., *Excited Delirium: How Cops Invented a Disease*, Current Affairs, April 13, 2021. Accessed at https://www.currentaffairs.org/2021/04/excited-delirium-how-cops-invented-a-disease [↩]
2. See Kasha Bornstein, Tim Montrief, MD, Mehruba Anwar Parris, MD. Excited Delirium: Acute Management in the ED Setting. Emer. Mgmt. Resident. April 8, 2019, last accessed at https://www.emra.org/emresident/article/excited-delirium/ [↩]
3. Id. [↩]
4. Kliem, L.V., "I am concerned about excited delirium…" Force Science News, August 25, 2020, at https://www.forcescience.org/2020/08/i-am-concerned-about-excited-delirium/ [↩]
5. See Martin, L., *Agree to Disagree: Excited Delirium Syndrome as Volatile as the Term Itself*, Psychiatric Times, May 13, 2021. Accessed at https://www.psychiatrictimes.com/view/borderline-personality-disorder-3-things-you-need-to-do-for-patients [↩]
6. See note 1. See also, REPORT 2 OF THE COUNCIL ON SCIENCE AND PUBLIC HEALTH (June 2021), Use of Drugs to Chemically Restrain Agitated Individuals Outside of Hospital Settings, (Reference

Committee E [↩]
7. DeBard ML, Adler J, Bozeman W, Chan T, et al: ACEP Excited Delirium Task Force White Paper Report on Excited Delirium Syndrome, September 10, 2009, last accessed on June 24, 2020, at https://www.prisonlegalnews.org/media/publications/acep_report_on_excited_delirium_syndrome_sept_2009.pdf [↩]
8. Note 2. [↩]
9. Hatten BW, Bonney C, Dunne RB, Hail SL, et al: ACEP Task Force Report on Hyperactive Delirium with Severe Agitation in Emergency Settings, June 23, 2021, last accessed on October 7, 2021, at https://www.acep.org/globalassets/new-pdfs/education/acep-task-force-report-on-hyperactive-delirium-final.pdf [↩]

AMA APA EMS Excited Delirium Excited Delirium Syndrome In Custody Death

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2 RESPONSES

Morrell Sipe REPLY

October 13, 2021 at 7:40 am

Thank you for this insightful article.

LEO Round Table REPLY

October 21, 2021 at 3:25 pm

[…] https://www.forcescience.org/2021/10/sidestepping-the-excited-delirium-debate/ […]

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“Excited Delirium” and Deaths in Police Custody

The Deadly Impact of a Baseless Diagnosis

March 2022

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The report was reviewed, edited, and prepared for publication by Claudia Rader, MS, PHR senior communications manager, with assistance from Samantha Peck, PHR executive assistant. Hannah Dunphy, PHR digital communications manager, prepared the digital presentation.

Cover: Pictures of Elijah McClain and George Floyd were placed in front of the Brooklyn Center, MN police station by protesters following the police shooting there of Daunte Wright in April 2021. Photo: Kerem Yucel/AFP via Getty Images

Executive Summary

On December 23, 2020, Bella Quinto-Collins called 911, seeking help for her 30-year-old brother, Angelo Quinto, who was agitated and exhibiting signs of a mental health crisis at their home in Antioch, California. When two police officers arrived, they pulled Quinto from his mother's arms onto the floor. At least twice, Quinto's mother, Cassandra Quinto-Collins, heard him say to the officers, "Please don't kill me." Bella and Cassandra then watched in disbelief and horror as the two officers knelt on Quinto's back for five minutes until he stopped breathing. Three days later, Quinto died in the hospital.[1]

It was not until August 2021 that the family learned the official determination of cause of death: a forensic pathologist testified during a coroner's inquest that Quinto died from "excited delirium syndrome."[2]

Angelo Quinto, a Filipino-American Navy veteran, is one of many people, disproportionately people of color, whose deaths at the hands of police have been attributed to "excited delirium" rather than to the conduct of law enforcement officers. In recent years, others have included Manuel Ellis, Zachary Bear Heels, Elijah McClain, Natasha McKenna, and Daniel Prude.[3] "Excited delirium" even emerged as a defense for the officers who killed George Floyd in 2020.[4]

An *Austin-American Statesman* investigation into each non-shooting death of a person in police custody in Texas from 2005 to 2017 found that more than one in six of these deaths (of 289 total) were attributed to "excited delirium."[5] A January 2020 *Florida Today* report found that of 85 deaths attributed to "excited delirium" by Florida medical examiners since 2010, at least 62 percent involved the use of force by law enforcement.[6] A Berkeley professor of law and bioethics

Mourners at a birthday vigil for Angelo Quinto, who was killed by police in California in December 2020. His death was attributed to "excited delirium syndrome."
Photo: Courtesy of the Quinto-Collins family

The term “excited delirium” cannot be disentangled from its racist and unscientific origins.

conducted a search of these two news databases and three others from 2010 to 2020 and found that of 166 reported deaths in police custody from possible “excited delirium,” Black people made up 43.3 percent and Black and Latinx people together made up at least 56 percent.[7]

When did the term “excited delirium” evolve to describe a distinct type of “delirium?" How did the corresponding term “excited delirium syndrome” become a go-to diagnosis for medical examiners and coroners to use in explaining deaths in police custody? What is the evidence that it is indeed a valid diagnosis? This report traces the evolution of the term from when it appears to have first been coined in the 1980s to the present. Physicians for Human Rights (PHR) reconstructed the history of the term “excited delirium” through a review of the medical literature, news archives, and deposition transcripts of expert witnesses in wrongful death cases. We evaluated current views and applications of the term through interviews with 20 medical and legal experts on deaths in law enforcement custody. Additionally, we spoke to six experts on severe mental illness and substance use disorders to better understand the context in which the term most often arises. Finally, we interviewed members of two families who lost loved ones to police violence for a firsthand account of the harms of the term’s continued use.

This report concludes that the term “excited delirium” cannot be disentangled from its racist and unscientific origins. Dr. Charles Wetli, who first coined the term with Dr. David Fishbain in case reports on cocaine intoxication in 1981 and 1985,[8] soon after extended his theory to explain how more than 12 Black women in Miami, who were presumed sex workers, died after consuming small amounts of cocaine.[9] “For some reason the male of the species becomes psychotic and the female of the species dies in relation to sex,” he postulated.[10] As to why all the women dying were Black, he further speculated, without any scientific basis, “We might find out that cocaine in combination with a certain (blood) type (more common in blacks) is lethal.”[11]

After a 14-year-old girl was found dead in similar circumstances but without any cocaine in her system, Wetli’s supervisor, chief medical examiner Dr. Joseph Davis, reviewed the case files.[12] Davis concluded that all of the women – 19 by that point – had actually been murdered, pointing to evidence of asphyxiation in many of the cases.[13] Investigators eventually came to hold a serial killer responsible for the murders of as many as 32 women from 1980 to 1989.[14]

The year after the suspected killer’s arrest, Wetli continued to assert that at least some of the women had died from a combination of sex and cocaine: “I have trouble accepting that you can kill someone without a struggle when they’re on

It seems that “excited delirium” as a diagnosis and standalone cause of death was originally brought about by one or a few people’s subjective opinions…. It is not a valid, independent medical or psychiatric diagnosis. There is no clear or consistent definition, established etiology, or known underlying pathophysiology.

cocaine … cocaine is a stimulant. And these girls were streetwise.”[15] He also continued to promote a corresponding theory of Black male death from cocaine-related delirium, without any scientific basis: “Seventy percent of people dying of coke-induced delirium are black males, even though most users are white. Why? It may be genetic.”[16]

Wetli’s grave mischaracterization of the murders of Black women in Miami – and the racism and misogyny that seemed to inform it – should have discredited his other equally racialized and gendered theory of sudden death from cocaine. Instead, the use of the term “excited delirium” grew.

A small cohort of authors, many working as researchers or legal defense experts for TASER International (now Axon Enterprise) – a U.S. company that produces technology products and weapons, including the “Taser” line of electroshock weapons marketed as so-called “less-lethal” “stun” weapons – increased the broader use of the term by populating the medical literature with articles about “excited delirium.” In 2007, TASER/Axon purchased many copies of a book entitled *Excited Delirium Syndrome* written by one of its defense experts, Dr. Vincent Di Maio, and his wife Theresa Di Maio, that built on Wetli’s description of “excited delirium” by describing an “excited delirium syndrome.”[17] They distributed the book for free and also gave out other materials on “excited delirium” at conferences of medical examiners and police chiefs.[18] Seven years later, during a deposition, Dr. Di Maio acknowledged that he and his wife had “come up with” the term “excited delirium syndrome.”[19] The term has come to be used as a catch-all for deaths occurring in the context of law enforcement restraint, often coinciding with substance use or mental illness, and disproportionately used to explain the deaths of young Black men in police encounters.[20]

PHR’s review leads to the conclusion that “excited delirium” is not a valid, independent medical or psychiatric diagnosis. There is no clear or consistent definition, established etiology, or known underlying pathophysiology. There are no diagnostic standards, and it is not included as a diagnosis in any version of the *International Classification of Diseases*, the international standard for reporting diseases and health conditions, currently in its tenth revision (*ICD-10*), or in the *Diagnostic and Statistical Manual of Mental Disorders (DSM-5*) criteria for psychiatric illness. Neither the American Medical Association nor the American

Psychiatric Association currently recognize the validity of the diagnosis. In general, there is a lack of scientific data, and the body of literature supporting the diagnosis is small and of poor quality, with homogenous citations rife with conflicts of interest.

The foundations underpinning the diagnosis of "excited delirium" have been misrepresented, misquoted, and distorted. The *ICD-10* and *DSM-5* acknowledge delirium and its subtypes as valid, but these do not align with purported criteria for "excited delirium" and are described as stemming from underlying causes. It seems that "excited delirium" as a diagnosis and standalone cause of death was originally brought about by one or a few people's subjective opinions. The term has since taken on a meaning and life of its own, with a deleterious impact.

In our interviews with clinicians and scientists across disciplines, there was no consensus on the definition of "excited delirium." A review of the medical literature further confirms that the syndrome is not well defined or understood. The term is therefore scientifically meaningless because of this lack of consensus or rigorous evidentiary basis. Many of the studies that have been used to support the diagnosis have serious methodological deficiencies and are laden with conflicts of interest with law enforcement and TASER/Axon. Moreover, the use of "excited delirium" to explain agitated behavior raises the concern that underlying causes of these behaviors, such as a mental illness or substance intoxication, are not being diagnosed or treated. Most significantly, it is disturbing that "excited delirium" as a diagnosis has been used to justify aggressive and even fatal police tactics.

Police in Aurora, CO face off with demonstrators protesting the killing of Elijah McClain. McClain was forcibly subdued by Aurora police while walking home and injected with ketamine by paramedics who diagnosed him with "excited delirium." McClain suffered a heart attack on the way to the hospital and died four days later. Photo: Andy Cross/MediaNews Group/Denver Post via Getty Images

The term has come to be used as a catch-all for deaths occurring in the context of law enforcement restraint … and disproportionately used to explain the deaths of young Black men in police encounters.

It is also concerning that "excited delirium" has come to pervade law enforcement policies and training manuals, at least in part due to the continued acceptance of the term by the American College of Emergency Physicians (ACEP) and National Association of Medical Examiners (NAME). Officers in many law enforcement agencies are trained to respond to an array of medical emergencies as "excited delirium," which in practice have included conditions that may not all warrant the same medical response, including heart attacks, drug or substance overdoses or withdrawals, acute psychosis, and oxygen deprivation. "Excited delirium" has also gained international reach, having received attention in the wake of in-custody deaths in Australia, Canada, and the United Kingdom, among other countries.[21]

The diagnosis of "excited delirium" has come to rest on racist tropes of Black men and other people of color as having "superhuman strength" and being "impervious to pain," while pathologizing resistance to law enforcement, which may be an expected or unsurprising reaction of a scared or ill individual (or anyone who is being restrained in a position that inhibits breathing). Presently, there is no rigorous scientific research that examines prevalence of death for people with "excited delirium" who are not physically restrained.

People who present with symptoms and signs such as agitation, confusion, fear, hyperactivity, acute psychosis, sweats, noncompliance with directions, tachycardia (rapid heart rate), and tachypnea (rapid breathing), which are too often classified by medical examiners and coroners as "excited delirium," must be recognized as having an underlying diagnosis. The specific underlying condition should be identified and treated. Too often, law enforcement officers are called as the sole first responders to medical emergencies and then use violent methods to forcibly restrain people manifesting these signs, methods – such as those that induce asphyxia from prone and other forms of restraint – that themselves may cause death. Consequently, "excited delirium," rather than law enforcement actions, is cited as the cause of death, or as a factor contributing to death, in autopsy reports.

PHR holds that "excited delirium" is a descriptive term of myriad symptoms and signs, not a medical diagnosis, and, as such, should not be cited as a cause of death. It is essential to end the use of "excited delirium" as an officially determined cause of death, particularly in cases of deaths in police custody. This is one critical step among many to stop these preventable deaths.

It is essential to cease the use of "excited delirium" as an officially determined cause of death, particularly in cases of deaths in police custody. This is one critical step among many to stop these preventable deaths.

Key Recommendations

To the American College of Emergency Physicians (ACEP) and National Association of Medical Examiners (NAME):

- Issue statements clarifying that "excited delirium" is not a valid medical diagnosis and cannot be a cause of death.

To State and Local Governments:

- Improve official responses to people experiencing mental and behavioral health challenges, including by bolstering social services and investing in alternative models of crisis response led by health professionals and/or social workers.
- Establish independent oversight systems and mandate independent investigations of deaths in law enforcement custody.

To Congress:

- Exercise Congress's oversight authority to investigate the use of "excited delirium" in various jurisdictions across the United States in the context of deaths in police custody, systemic racism, and the pursuit of justice and accountability.

Introduction

As Minneapolis police officer Derek Chauvin knelt on George Floyd's neck in May 2020, fellow officer Thomas Lane said, "Roll him on his side?... I just worry about the excited delirium or whatever." Officer Lane's comment in the midst of George Floyd's murder is indicative of the extent to which the concept of "excited delirium" has come to pervade U.S. law enforcement training and practice.

This report traces how "excited delirium" has evolved from a description in case reports of people with cocaine intoxication into a term that is used by law enforcement, forensic pathologists, emergency physicians, and in courts. Others have already described the troubled history of "excited delirium."[22] Yet since the term persists, this report reviews the origins, history, medical literature, and views of experts and affected family members in order to evaluate the underlying validity of the diagnosis.

Background

In the United States, people of color are far more likely than white people to be killed by police.[23] The American Medical Association, American Public Health Association, National Medical Association, and many other groups recognize this as a public health crisis.[24] In addition, a significant percentage of police killings – anywhere from 25 to 50 percent – occur while responding to mental health, behavioral health, or substance use disorder crises.[25]

The in-custody killing of George Floyd by Minneapolis police in May 2020 ignited an unprecedented wave of national and global demonstrations in support of the Black Lives Matter movement and against police brutality and systemic racism across many areas of law enforcement. Protesters called for accountability for police killings and reforms, with many urging the reallocation of funding from law enforcement to social and community services, including mental health services. Protesters also drew attention to the ways in which certain health emergencies all too often receive a law enforcement rather than a medical response, which can result in serious harm or death.

A significant percentage of police killings – anywhere from 25 to 50 percent – occur while responding to mental health, behavioral health, or substance use disorder crises.

In Many Areas, the United States Lacks Appropriate Systems to Respond to Mental and Behavioral Health Crises

In 2020, the Substance Abuse and Mental Health Services Administration (SAMHSA), a branch of the U.S. Department of Health and Human Services, reported that more than one in every five American adults (21 percent) experienced a mental illness.[26] Additionally, in 2020, more than one in every 20 adults (5.6 percent) experienced a serious mental health condition, such as schizophrenia or bipolar disorder.[27] Both of these estimates were higher than annual estimates from 2008 through 2019.[28]

Despite the increasing prevalence of mental health conditions in the United States, there remains a lack of appropriate emergency response systems for people in crisis. Moreover, the deinstitutionalization movement, beginning in the 1950s, left many people with severe mental illness with neither proper treatment nor resources. This has led to a number of people finding themselves homeless or in contact with the carceral system rather than appropriate treatment.[29] The norm when someone is experiencing a mental health crisis is to call emergency services through 911, where, in most jurisdictions, the police often respond. Using armed police as first responders in these cases can result in an escalation of the situation while criminalizing or further endangering the person in crisis. Introducing people with mental illness in crisis first to the carceral system by proxy of a police officer, instead of a trained mental health counselor or clinician, can and has led to deaths at the hands of law enforcement.[30] A 2015 report by the Treatment Advocacy Center found that people with untreated mental illness are 16 times more likely to be killed during a law enforcement encounter than other civilians.[31]

In a 2021 report, the Office of the United Nations High Commissioner for Human Rights (OHCHR) observed that law enforcement officers frequently violate the rights of Black people experiencing mental health crises to protection from discrimination on the basis of both race and disability. OHCHR reviewed more than 190 reports of deaths of Black people in law enforcement custody worldwide, including in the United States, finding that one of the three contexts that accounted for 85 percent of the cases that occurred was "the intervention of law enforcement officials as first responders in mental health crises." The report stated:

> "Several incidents analyzed by OHCHR occurred after calls to emergency services seeking assistance for a person experiencing a mental health crisis. According to the analysis, when acting as first responders, police interventions often aggravate the situation including due to the use of restraints, while crises de-escalation protocols may not provide for appropriate crisis support services. Further, police often fail to identify the victims as individuals in distress and in need of rights-based mental

"When you're dealing with severe mental illness, and especially when you're a Black family or a brown family, you pause before you call the police."

Sabah Muhammad, attorney and legislative and policy counsel, Treatment Advocacy Center

health support. Instead, racial bias and stereotypes compounded with disability-based stereotypes appear to lead law enforcement officials to perceive the victim as "dangerous", overriding considerations of the individual's safety and well-being and of delivery of the appropriate care and basic life support."[32]

Standards for Death Investigations in the United States Vary by Jurisdiction

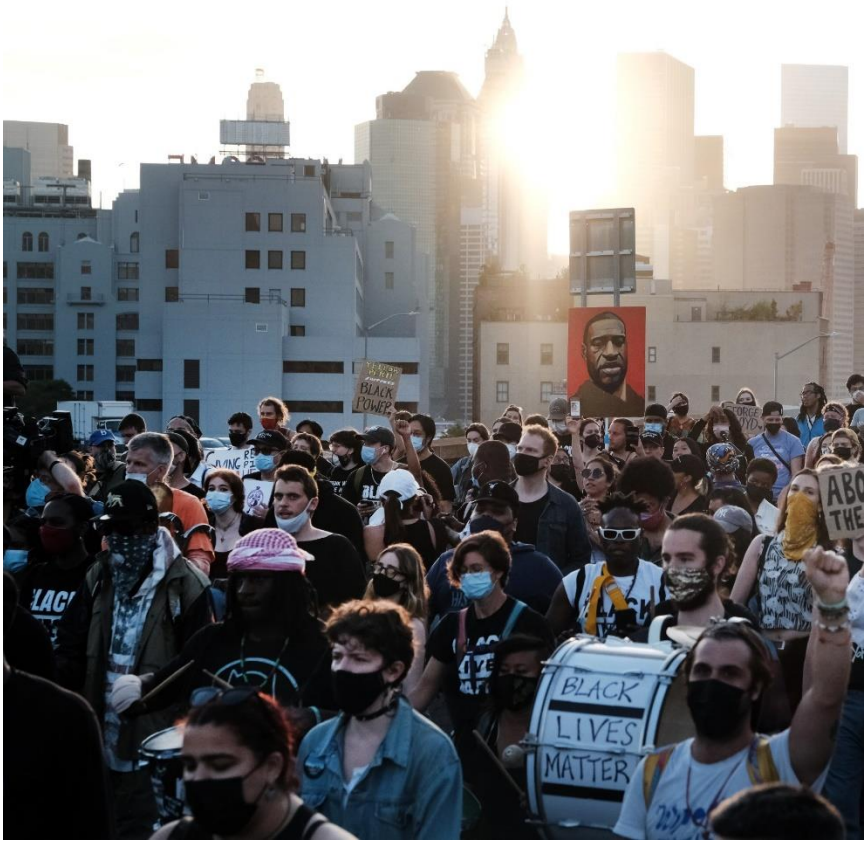
In the United States, official processes for investigating and establishing cause of death vary by state and local jurisdiction. Each state has different requirements for which kinds of deaths require investigations or autopsies.[33] Death investigation systems are highly variable, including both medical examiner systems and coroner systems. In most systems, it is a coroner or medical examiner's responsibility to lead an investigation to determine the circumstances of a person's death in cases of homicide or when there is suspicion of crime or foul play, including police violence.[34] Coroners in most states do not have to be physicians.[35] Medical examiners are physicians but are not always forensic pathologists. Forensic pathologists are physicians that specialize in pathology (study of injured organs, tissues, and cells) and work at the intersection of law and medicine to determine the cause of death. Twenty-three (23) states and Washington, D.C. have appointed medical examiner and/or coroner systems, 11 states have elected coroners and appointed medical examiners, four states have a combination of elected and appointed coroners, and 12 states have a combination of elected and appointed medical examiners.[36]Although there is a lack of national standards and of a universal definition, the consensus for defining deaths in custody is "deaths of persons who have been arrested or otherwise detained" by law enforcement officials.[37]

In 2009, the National Academy of Sciences (NAS) recommended, "Congress should authorize and appropriate incentive funds to the National Institute of Forensic Science (NIFS) for allocation to states and jurisdictions to establish medical examiner systems, with the goal of replacing and eventually eliminating existing coroner systems." NAS further held, "All medicolegal autopsies should be performed or supervised by a board certified forensic pathologist."[38]

Law Enforcement-Related Deaths Are Under-Counted

The age-adjusted mortality rate due to police violence grew by 38.4 percent from the 1980s to the 2000s, and mortality rates due to police violence were highest in non-Hispanic Black people.

There is strong evidence that deaths after or during interaction with law enforcement are not always appropriately reported, monitored, or investigated. A 2017 Harvard study found that more than half of all police killings in 2015 were incorrectly classified as not the result of police officer interactions.[39] Coroners and medical examiners were found to regularly report results that minimized the accountability of police officers.[40] The study compared data from *The Guardian's* "The Counted,"[41] an investigative project on police killings, to data from the National Vital Statistics System (NVSS), a U.S. federal government system that gathers death certificate data, identifies law enforcement-related deaths, and assigns a corresponding diagnostic code: "legal intervention."[42] This same study found that there were significantly more law enforcement-related deaths in *The Guardian's* data set compared to the NVSS. They further discovered that the NVSS had misclassified 55.2 percent of all police killings, and that deaths in low-income areas were disproportionately underreported.[43]



Black Lives Matter protesters march across the Brooklyn Bridge in New York City on May 25, 2021, on the first anniversary of George Floyd's death at the hands of police. Photo: Andy Cross/Spencer Platt/Getty Images

Similarly, a 2021 *Lancet* study compared data from the NVSS to "The Counted" and two other media-based databases on police violence, "Fatal Encounters" and "Mapping Police Violence." The results showed that the NVSS failed to report "55.5 percent of all deaths attributable to police violence," missing about 17,100 deaths from 1980 to 2019.[44] The study also found that the age-adjusted mortality rate due to police violence grew by 38.4 percent from the 1980s to the 2000s, and mortality rates due to police violence were highest in non-Hispanic Black people, followed by Hispanic people of any race, non-Hispanic white people, and finally non-Hispanic people of other races.[45]

System Flaws and the Ability to Manipulate the Reporting System Contribute to Under-Counting of Law Enforcement-Related Deaths

Several factors contribute to under-counting of law enforcement-related deaths. One oft-cited reason is the lack of independence of coroners and medical examiners. In a 2011 survey of National Association of Medical Examiners (NAME) members, 22 percent reported experiencing political pressure from elected or appointed officials to change the cause or manner of death listed on death certificates.[46] Conflicts of interest built into many systems include having medical examiners and coroners work for or be part of police departments.[47] A second contributor to under-counting is the lack of well-established standards and guidelines. There are no standards or explicit instructions to note whether there was police involvement in many death certificates' open-ended sections to "describe how the injury occurred," or to assure correct coding that there was law enforcement involvement, even if the certificate notes police involvement. Moreover, lack of standards to ensure sufficient knowledge and training of coroners and medical examiners further contributes to errors in classification. For example, some medical examiners face difficulty in having to determine whether a restraint case, such as a "hog-tying incident," should be classified as "homicide," "accident," or "undetermined." There is no national definition on manner of death for these police custody killings.[48] Lastly, fear of litigation

In a 2011 survey of National Association of Medical Examiners members, 22 percent reported experiencing political pressure from elected or appointed officials to change the cause or manner of death listed on death certificates. In another survey, 13.5 percent acknowledged modifying their forensic findings because of previous threats of litigation, and 32.5 percent revealed that these considerations would impact their decisions in the future.

resulting from problematic conduct also influences accurate documentation. In another NAME survey with 222 medical examiner respondents, 13.5 percent acknowledged modifying their forensic findings because of previous threats of litigation, and approximately 32.5 percent revealed that these considerations would impact their decisions in the future.[49] Thirty (30) percent expressed that "fear of litigation affected their diagnostic decision-making."[50] In this way, a lack of standards is compounded by a lack of independence of forensic scientists to act without undue pressure from law enforcement or political officials.

*In 2002, the National Association of Medical Examiners (NAME) published its first edition guide for **manner of death** classification; it notes that its guide is not a standard and that death certification requires judgment on a case-by-case basis.[51] It elaborates that manner of death (i.e., determination of how an injury or disease leads to death, such as natural, accident, suicide, homicide, or undetermined) is "circumstance-dependent, not autopsy-dependent."[52] This guide outlines important general principles and definitions:*

> **"Natural deaths** are due solely or nearly totally to disease and/or the aging process. **Accident** applies when an injury or poisoning causes death and there is little or no evidence that the injury or poisoning occurred with intent to harm or cause death. **Homicide** occurs when death results from a volitional act committed by another person to cause fear, harm, or death. Intent to cause death is a common element but is not required for classification as homicide…. **Undetermined** or "could not be determined" is a classification used when the information pointing to one manner of death is no more compelling than one or more other competing manners of death in thorough consideration of all available information. In general, when death involves a combination of natural processes and external factors such as injury or poisoning, preference is given to the non-natural manner of death."

*The **"but-for"** logic is often used as a simple way to determine whether a death should be classified as natural or non-natural.[53] "But-for the injury (or hostile environment), would the person have died when [they] did?" The guide elaborates that "the manner of death is unnatural when injury hastened the death of one already vulnerable to significant or even life-threatening disease." In this guide, the authors call for greater national consistency in death certification.*

In 2017, NAME published a position paper with recommendations for the investigation and reporting of deaths in police custody. In summary, the association calls for an investigation into the facts and circumstances of these deaths, and notes that the investigation has the potential to prevent

similar future deaths and provide educational benefits.[54] *The report elaborates on* ***cause of death*** *and* ***manner of death****:*

> "This committee recommends that the physician consider homicide as the manner of death in cases similar to those that would otherwise meet the threshold of 'death at the hands of another.' While the cause and manner of death designation should be handled the same as any other, the certifying physician/professional should fully utilize the 'How Injury Occurred' section of the death certificate to communicate that the death occurred in custody. For example, wording such as 'Shot by Law Enforcement', 'Driver of Motor Vehicle in Collision with Fixed Object during Pursuit by Law Enforcement', 'Shot Self in the Presence of Law Enforcement', 'Hanged Self while Incarcerated', or 'During Restraint by Law Enforcement' should be included."

Methodology

Physicians for Human Rights (PHR) sought to understand the complex origins, history, current usage, and validity of "excited delirium" by pursuing multiple strands of inquiry.

Documents

As part of PHR's work to systematically document the origins, history, and evolution of the term and concept of "excited delirium," PHR partnered with civil rights attorney Julia Sherwin, who, through nearly two decades of work, has compiled an extensive library of news archives, deposition transcripts, court documents, and articles related to the origins and history of "excited delirium." PHR obtained additional deposition transcripts and court documents from civil rights attorneys John Burton and Ben Nisenbaum.

Medical Literature Review

To examine the extent and quality of evidence for "excited delirium" as a diagnosis and potential cause of death, physician members of the PHR team conducted a scoping review of and analyzed peer-reviewed medical literature.[55] On August 19, 2021, PHR conducted a PubMed/MEDLINE search using the key words "excited delirium" without filters. Two hundred twenty-six abstracts (226) were found between the available date range of January 1956 and August 2021. Titles and abstracts were screened for information on diagnostic criteria for "excited delirium," origins of the term, pathophysiology, and evidence for the syndrome. If the abstract was not available or if the article was unclear after a review of the abstract alone, a full review of the article was performed.

Articles were excluded if they were not peer reviewed, not in English (due to a lack of capacity to translate), or did not provide any of the following: 1) historical information on the origins of "excited delirium;" 2) a definition or description of "excited delirium," which may have included pathology or pathophysiology; or 3) a discussion of evidence for or against "excited delirium" as a distinct syndrome. Articles were also excluded if they focused solely on a case report or series, drugs, or treatment without significant discussion of "excited delirium" as an entity itself. Of the 226 articles, 180 did not meet the above criteria and were excluded from our analysis, leaving 46 peer-reviewed articles. A secondary search was performed on the same database using the term "excited delirium syndrome," which yielded 95 results, all of which had already been captured in the primary search. (Of note, alternate search terms were not employed, such as "Bell's mania," "agitated delirium," "positional asphyxia," "restraint asphyxia," "in-custody deaths," or "police use of force.")

Between August 19, 2021 and October 20, 2021, PHR team members read and abstracted articles that met inclusion criteria. To provide important context to the 46 peer-reviewed articles, other literature, such as letters to the editor and commentary, secondary references, consensus and position papers, and non-peer reviewed material, were also considered and incorporated in this report when germane.

To check for saturation and consistency, results were compared to a general literature review performed in July 2021 by a different PHR team during the concept design stage of this report. The references and conclusions of these two independent literature reviews were complementary and consistent.

Interviews

In light of the continued use of "excited delirium" as a cause of death among medical examiners and coroners, PHR explored the experiences and perspectives of forensic pathologists and other medical and legal experts on deaths in custody. After obtaining exemption from PHR's Ethics Review Board, given the low risk to interviewees, PHR conducted individual semi-structured interviews with 20 experts on deaths in police custody regarding their knowledge and perspective on the use of "excited delirium" as a cause of death. The interviewees included nine forensic pathologists (across the United States, Canada, Chile, and New Zealand, one of whom also trained in Italy and Scotland), one forensic epidemiologist, two emergency physicians, one surgeon who is also a certified medico-legal death investigator, four plaintiff's attorneys, two prosecutors, and one law enforcement trainer.[56] We used snowball sampling to connect with experts and continued reaching out to prospective interviewees until we reached thematic saturation (i.e., no new themes emerged during analysis of interview transcripts). Although the focus of our research was the use of "excited delirium" as a cause of death in the United States, we also interviewed forensic pathologists based outside the

United States considering the global reach of the medical literature on "excited delirium."

In the interviews with physicians, we sought to identify areas of consensus and ongoing discussion regarding "excited delirium" and to learn about their introduction to the term and the evolution of their understanding. We interviewed the attorneys to inform the report background and to seek their views on the prevention of deaths in custody that are attributed to "excited delirium." PHR also held conversations geared toward preventing such deaths with experts on mental health and substance use crisis response, including staff at the Treatment Advocacy Center, National Harm Reduction Coalition, Crisis Assistance Helping Out On The Streets (CAHOOTS), and Portland Street Response.[57]

Finally, PHR received approval from PHR's Ethics Review Board to interview members of families who had lost loved ones to deaths in police custody in the United States where "excited delirium" was designated by medical examiners as the cause of death. We connected with civil rights attorneys who represent families in wrongful death lawsuits against law enforcement officers and asked the attorneys whether any of their clients were interested in speaking with us for our report. Two families conveyed through their attorneys their interest in speaking with PHR, and their attorneys were present for the subsequent interviews.

All interviews took place via video or audio conferencing due to the SARS-CoV-2 public health emergency and wide geographical location of interviewees. All participants gave verbal consent to the interview, and for the interview to be recorded. Notes were also typed during the interviews.

Interviewees were informed of the purpose and voluntary nature of the interview. They were told that they could stop the interview at any time and that all possible measures would be taken to keep their identity confidential unless they wanted to disclose it. They were given the option of remaining anonymous and using a pseudonym in this report. Interviewees received no compensation for participating in interviews. The interviewers used an interview guide, previously agreed upon by the research team. Interview materials and transcripts were stored securely on PHR computers. Team members reviewed the written notes and transcripts to identify key themes across the interviews and pull illustrative quotes.

Limitations

PHR's interviews with forensic pathologists, emergency physicians, lawyers, and others are not intended to be a representative sample of the field. Rather, we sought to speak to experts both in the United States and internationally to gauge areas of consensus and ongoing discussion regarding the continued use of "excited delirium."

The medical literature review was not exhaustive and used one biomedical literature database (PubMed/MEDLINE). Only "excited delirium" and "excited delirium syndrome" were searched and may have not resulted in a comprehensive selection of relevant articles. After articles meeting inclusion criteria were identified and reviewed, a pragmatic research approach was adopted: references of included articles were explored for context and history.

Findings

Origins and History

Key Definitions

*A **syndrome** consists of a group of signs and symptoms that occur together and characterize a discrete abnormality or condition.[58] The cause, pathophysiology, and/or course of a "syndrome" is often not clearly understood. Once medical science identifies a clear causative agent or underlying pathophysiologic process, the group of signs and symptoms are then referred to as a "**disease**." What are considered diseases change over time as a result of advances in technology, diagnostic ability, and expert consensus determinations, among other factors. In psychiatry, maladaptive mental and behavioral disturbances that impair functioning are often referred to as **disorders.** There are well-defined criteria for diagnosing psychiatric disorders, even though some have criticized these criteria as unreliable.[59]*

*The American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5) defines **delirium** as a neurocognitive disorder characterized by a "disturbance in attention and awareness that develops over a short period of time and is not better explained by another preexisting, evolving, or established disorder."[60] Additional features may include hypo- or hyperactivity and emotional disturbances such as fear, agitation, or euphoria, as well as reduced awareness of the environment. The pathophysiology of delirium is poorly understood, but it is generally accepted as a sign of an underlying disease process, such as organ failure, infection, lack of oxygen, metabolic imbalance such as low blood sugar levels, drug side effects, intoxication, or withdrawal, among others. Delirium is medically treated by finding and treating the underlying cause, along with supportive behavioral modifications and medical care such as hydration, psychopharmaceuticals, and pain control.*

Restraints in the medical context are actively discouraged and avoided in the management of delirium, never include prone or neck restraints, and

are monitored by an independent medical oversight organization (the Joint Commission on Accreditation of Healthcare Organizations). Delirium is not itself considered a cause of sudden death.

Bell's Mania

In 1849, Dr. Luther Bell, a Massachusetts physician at the McLean Asylum for the Insane, described cases of primarily female psychiatric patients who experienced symptoms and signs such as overactivity, delusions, transient hallucinations, sleeplessness, and fevers, typically over days to weeks, and in some cases resulting in death.[61] This constellation of signs and symptoms has been called Bell's Mania, delirious mania, acute maniacal delirium, lethal catatonia, and, later, chronic "excited delirium."[62] The Bell's Mania description occurred long before other diagnoses like schizophrenia,[63] bipolar mania, or autoimmune encephalitis were described in their current formulations, and the signs and symptoms of Bell's Mania are consistent with these diagnoses, among others. The disappearance of case reports using these descriptions between the 1950s and 1980s has been attributed to the rise of relatively effective antipsychotic medications and treatment and greater psychiatric diagnostic precision.[64]

Wetli and Fishbain

The introduction of the term "excited delirium" in the 1980s has been attributed to Drs. David Fishbain and Charles Wetli. In the early 1980s, at the University of Miami, Fishbain was director of psychiatric emergency services, and Wetli was a forensic pathologist. In 1981, Wetli and Fishbain co-authored a case report of cocaine intoxication in a person who swallowed packets of cocaine in order to store them within their body, termed "bodypacker."[65] Wetli and Fishbain described the resulting delirium as a medical emergency characterized by a disturbance of attention with impaired perception. They characterized this "acute excited delirium" as reversible, transient, and with an array of possible causes. They elaborated that there are "two types of delirium: stuporous … and excited." Notably, they stated that the treatment of delirium is of the underlying illness and concluded that the delirium presentation hides the "medical nature."

In 1985, Wetli and Fishbain published a case series on cocaine-induced psychosis.[66] This series described seven cocaine users (six men and one woman) who exhibited fear, panic, violent behavior, hyperactivity, hyperthermia, and/or unexpected strength. All of them had been restrained (six by police, in some cases with the assistance of bystanders, and one by emergency room staff) and all died suddenly with respiratory arrest, with five of them reportedly dying in police custody.

Case 1	"The police subsequently restrained his ankles and attached the ankle restraints and handcuffs together."
Case 2	"he was agitated and combative and had to be restrained."
Case 3	"With the aid of several police officers she was finally subdued; handcuffs and ankle restraints were applied and then attached to each other."
Case 4	"with the assistance of several bystanders, the victim was finally subdued. Handcuffs and ankle restraints were placed on the victim and were in turn tied together."
Case 5	"He was removed from the vehicle and his ankles were restrained as well. The ankle and handcuff restraints were then attached to each other."
Case 6	"He was finally apprehended but it took six police officers to restrain him. He was handcuffed, placed in a police vehicle, and kept under observation."
Case 7	"Three officers finally subdued the subject after a violent struggle during which the subject was struck twice in the head with a heavy flashlight. He was handcuffed behind his back and placed prone on the ground. He continued to thrash about for a period of time."

Excerpts from Charles V. Wetli, and David A. Fishbain, 1985, "Cocaine-Induced Psychosis and Sudden Death in Recreational Cocaine Users," Journal of Forensic Sciences, 30, no. 3 (July): 873 – 880.

Autopsies did not reveal any "anatomic cause of death." In this publication, Wetli and Fishbain again described "excited delirium" as a "medical emergency but with a psychiatric presentation" and noted that the "prognosis depends on the underlying cause of the delirium."

Four of the seven people had been either hog-tied (had their hands and feet fastened together) or put into a hobble restraint (a nylon strip that ties a person's ankles together and links them to their wrists handcuffed behind their back) in a prone position, which can impair breathing. Other than mentioning the prone restraint in passing, Wetli and Fishbain did not discuss the role restraint may have played in these victims' deaths.

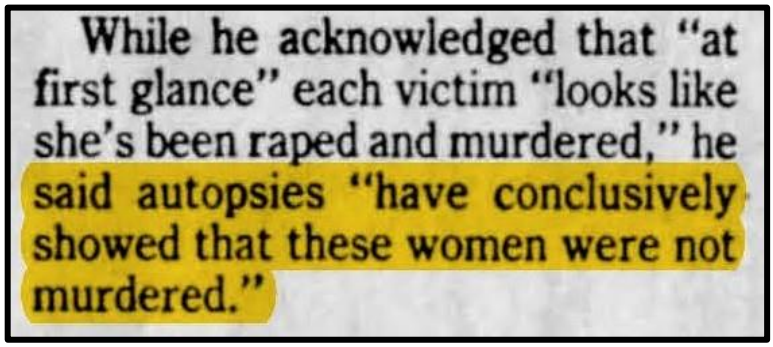
In both these 1981 and 1985 case reports, Wetli and Fishbain reference the *Comprehensive Textbook of Psychiatry*, 3rd edition, chapter 20, pages 1359–1392.[67] This section was written by Dr. Zbigniew J. Lipowski. (PHR obtained the same edition and reviewed these pages.) Wetli and Fishbain cite Lipowski when defining delirium, including the description of a hyperactive and hypoactive delirium: "There are two major types of delirium: stuporous (dull, lethargic, hypoactive, mute, somnolent, and apathetic), and excited (thrashing, shouting, hyperactive, fearful, panicky, agitated, hypervigilant, and violent)."[68] Lipowski does not use the term "excited delirium." It is our conclusion that Wetli and Fishbain initially used "excited" as an adjective to portray the hyperactive form of delirium.

A short time later, Wetli, as will be discussed below, began using "excited delirium" as a cause of death, diagnosis, and unique disease. There is, however, no indication in his writings that he had access to new scientific evidence underpinning this change.

Serial Murders of Black Women in Miami

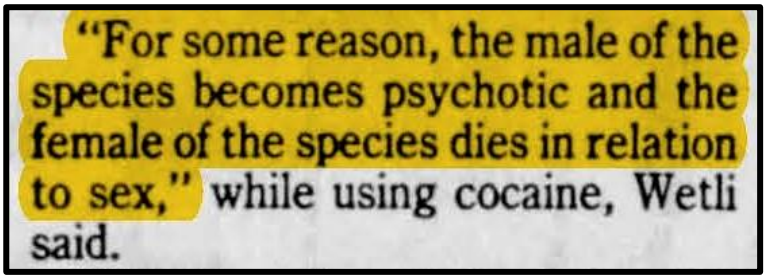
In the years that followed his publications on cocaine-induced "excited delirium," Wetli began to seek new applications of his theories in his work as deputy chief medical examiner in Miami.

Between September 1986 and November 1988, 12 Black women who were presumed sex workers were found dead, one after the other, in the same geographic area of Miami.[69] Wetli and several of his colleagues found that almost all had low levels of cocaine in their systems and classified the majority of the deaths as accidents from cocaine intoxication.[70] On November 24, 1988, Wetli began to publicize his theory that the women had died from combining sex with cocaine use, claiming that autopsies had "conclusively" shown they had not been murdered.[71]

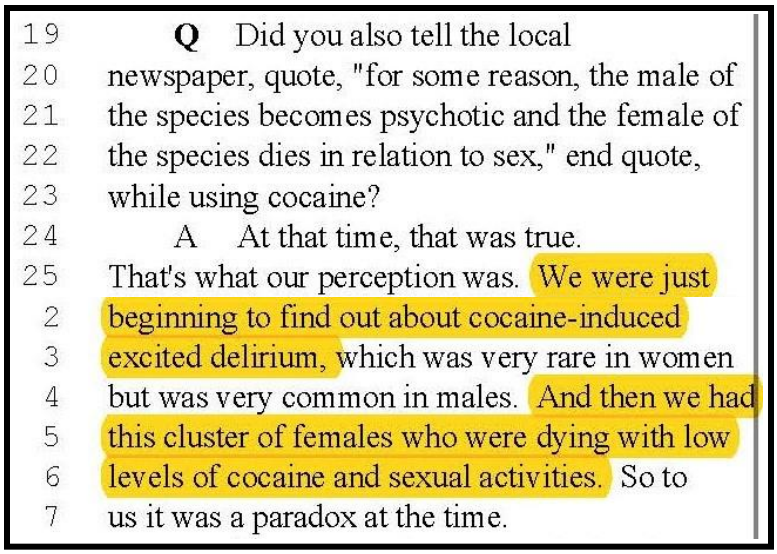

While he acknowledged that "at first glance" each victim "looks like she's been raped and murdered," he said autopsies "have conclusively showed that these women were not murdered."

Excerpt from Donna Gehrke, "Missed Calls, Close Calls Mar Serial Killings Case," Miami Herald, April 26, 1990, page 1A. Highlighting added for emphasis. (Quote first published in Adrian Walker and Heather Dewar, "Cocaine-Sex Deaths in Dade Probed," Miami News, November 24, 1988.)

Wetli speculated that while the women were working as sex workers, they consumed small amounts of cocaine and then died from sexual excitement, which he described as the female manifestation of the "cocaine psychosis" he had previously identified in men.[72] "For some reason, the male of the species becomes psychotic and the female of the species dies in relation to sex," he said.[73]


"For some reason, the male of the species becomes psychotic and the female of the species dies in relation to sex," while using cocaine, Wetli said.

Excerpt from Donna Gehrke, "Missed Calls, Close Calls Mar Serial Killings Case," Miami Herald, April 26, 1990, page 1A. Highlighting added for emphasis. (Quote first published in Adrian Walker and Heather Dewar, "Cocaine-Sex Deaths in Dade Probed," Miami News, November 24, 1988.)



Q Did you also tell the local newspaper, quote, "for some reason, the male of the species becomes psychotic and the female of the species dies in relation to sex," end quote, while using cocaine?

A At that time, that was true. That's what our perception was. We were just beginning to find out about cocaine-induced excited delirium, which was very rare in women but was very common in males. And then we had this cluster of females who were dying with low levels of cocaine and sexual activities. So to us it was a paradox at the time.

Excerpt from deposition of Charles Wetli in Harrison v. County of Alameda, January 15, 2014. Courtesy of Julia Sherwin. Highlighting added for emphasis.

As to why all the women dying were Black, he further speculated, without any scientific basis, "We might find out that cocaine in combination with a certain (blood) type (more common in blacks) is lethal."[74]

The following month, he said, "We know that the deaths are related to crack, but we still don't know the mechanism."[75]

On December 12, 1988 – less than a month after Wetli began to publicize this theory – 14-year-old Antoinette Burns was found dead.[76] Wetli, who performed the initial autopsy, believed that she, too, had died from a combination of sex and cocaine use.[77] For weeks, Burns' family pushed back against this theory, but it was not until the toxicology report came back negative that authorities began to take them seriously.[78]

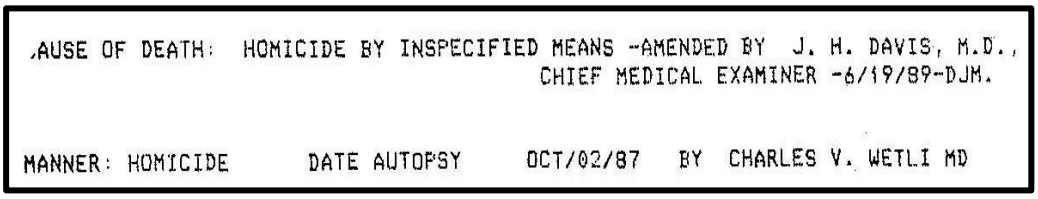
In March of 1989, police investigators confronted Wetli's supervisor, chief medical examiner Dr. Joseph Davis, with evidence they believed pointed to homicide.[79] Davis began to reexamine the case files.[80] In May, a newsweekly reported that the number of Black women found dead had reached at least 17.[81] The article noted that Burns had died without cocaine in her system and cited investigators'

The article described Wetli's sex-cocaine theory for women as the counterpart of his "excited delirium" theory about men. *"The women may be dying after sexual activity,"* Wetli said. *"The men just go berserk."*

beliefs that a serial killer was actually responsible for the women's deaths.[82] Burns' mother told the paper, "I'm always wondering who killed her and how did she die. I want justice to be served."[83]

Wetli, meanwhile, continued to promote his theory that cocaine combined with orgasm produced lethal results: "We still really don't know what's going on. My gut feeling, though, is that this is a terminal event that follows chronic use of crack cocaine affecting the nerve receptors in the brain. I think it's a type of neural exhaustion."[84] The article described Wetli's sex-cocaine theory for women as the counterpart of his "excited delirium" theory about men. "The women may be dying after sexual activity," Wetli said. "The men just go berserk."[85]

Later that month, Davis announced his conclusion that the deaths of all of the women – 19 by that point – were homicides.[86] He reclassified the 14 that had initially been ruled accidents or left unclassified.[87] Only nine women's bodies had been found soon enough to identify concrete signs of strangulation and/or asphyxiation.[88] In those women's cases, Davis found evidence of neck pressure in seven and pressure to the mouth in four, as well as evidence of hemorrhaging in the eyes.[89] He noted that in some of the women's cases, the signs of asphyxiation were so pronounced that one could see them from "ten feet away, it's that clear."[90]



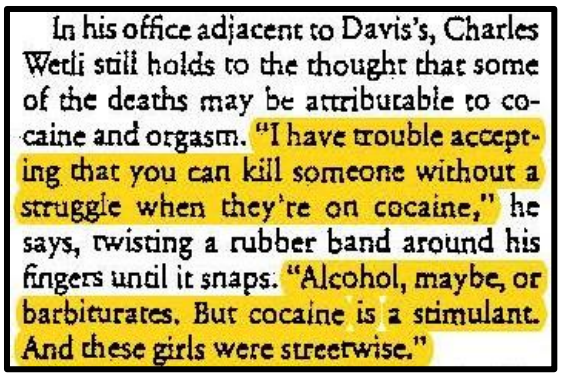
,AUSE OF DEATH: HOMICIDE BY INSPECIFIED MEANS -AMENDED BY J. H. DAVIS, M.D.,
CHIEF MEDICAL EXAMINER -6/19/89-DJM.

MANNER: HOMICIDE DATE AUTOPSY OCT/02/87 BY CHARLES V. WETLI MD

Excerpt from the Metropolitan Dade County Medical Examiner Department's amended investigation report for a woman found dead in October 1987. Her death had been ruled a cocaine intoxication accident in November 1987; Davis changed it to "homicide by inspecified [sic] means" in June 1989. Courtesy of Julia Sherwin.

All but one of the women were believed to have the same killer.[91] Police soon identified Charles Henry Williams, a convicted rapist, as the primary suspect.[92] Arrested in 1989 on an unrelated rape charge, he was eventually believed to be responsible for the deaths of as many as 32 women since 1980.[93] Later charged with one of the murders, he died before he could stand trial.[94]

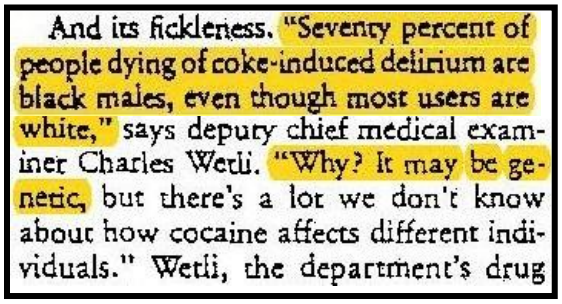
One year after Davis's reclassification of the deaths as homicides, Wetli continued to assert that at least some of the women had died from a combination of sex and cocaine: "I have trouble accepting that you can kill someone without a struggle when they're on cocaine … cocaine is a stimulant. And these girls were streetwise."[95]



In his office adjacent to Davis's, Charles Wetli still holds to the thought that some of the deaths may be attributable to cocaine and orgasm. "I have trouble accepting that you can kill someone without a struggle when they're on cocaine," he says, twisting a rubber band around his fingers until it snaps. "Alcohol, maybe, or barbiturates. But cocaine is a stimulant. And these girls were streetwise."

Excerpt from Russ Rymer, "Murder Without a Trace," In Health, May/June 1990, p.58. Highlighting added for emphasis.

Wetli also continued to promote a corresponding theory of Black male death from cocaine-related delirium, without any scientific basis: "Seventy percent of people dying of coke-induced delirium are black males, even though most users are white. Why? It may be genetic." [96]



And its fickleness. "Seventy percent of people dying of coke-induced delirium are black males, even though most users are white," says deputy chief medical examiner Charles Wetli. "Why? It may be genetic, but there's a lot we don't know about how cocaine affects different individuals." Wetli, the department's drug

Excerpt from Russ Rymer, "Murder Without a Trace," In Health, May/June 1990, p.55-56. Highlighting added for emphasis.

Wetli's grave mischaracterization of the murders of Black women in Miami – and the racism and misogyny that seemed to inform it – failed to discredit his other equally racialized and gendered theory of sudden death from cocaine.[97] Instead, the use of the term "excited delirium" grew.

NAME Position Paper (2004)

More than a decade later, Wetli coauthored a 2004 National Association of Medical Examiners (NAME) position paper that continued to link cocaine use to "excited delirium."[98] That position paper, in a single reference, noted briefly "a catecholamine-mediated excited delirium, similar to cocaine" that was "becoming increasingly recognized and has been detected in patients with mental disorders taking antidepressant medications, and in psychotic patients who have stopped taking their medications." It provided as a citation for this claim the abstract of a presentation by Wetli.[99] Yet, in discussing "sudden death related to police actions," the paper only discussed assessing the involvement of cocaine as a cause of death and asserted that "other obvious causes of death must be carefully ruled

out through a careful scene investigation, meticulous forensic autopsy, and a review of the medical information." The paper also delineated criteria for a diagnosis of "cocaine-induced excited delirium," requiring a "clinical history of chronic cocaine use, typically bizarre and violent psychotic behavior, and the presence of cocaine or its metabolites in body fluids or tissues." It did not discuss at all criteria for diagnosing "excited delirium" from causes other than cocaine use.[100]

In its 2017 position paper on recommendations for the investigation and reporting of deaths in police custody, NAME referenced "excited delirium" in passing, noting, "the more difficult cases are those where the individual is observed to be acting erratically due to a severe mental illness and/or acute drug intoxication. These cases have been defined in the literature as excited delirium and often result in a law enforcement response and restraint of the decedent."[101]

Publication of *Excited Delirium Syndrome*

In 2005, Theresa Di Maio, a psychiatric nurse, and her husband, Dr. Vincent Di Maio, a forensic pathologist who was serving as the chief medical examiner of Bexar County, Texas and editor of the *American Journal of Medicine and Pathology*, published a book on "excited delirium syndrome."[102] They defined the term as "the sudden death of an individual during or following an episode of excited delirium, in which an autopsy fails to … explain the death."[103] They defined "excited delirium" as "delirium involving combative or violent behavior" caused by "normal physiologic reactions of the body to stress gone awry."[104] The Di Maios discussed the history and origins of "excited delirium" via summarized case reports from primarily the 1930s and 1940s, in most cases describing women in psychiatric institutions. In a 2014 deposition in a restraint death case, Dr. Di Maio noted that he and his wife had coined the term "excited delirium syndrome."[105]

Prone Restraint Studies

At the same time that the Di Maios were promoting the concept of "excited delirium syndrome," others were conducting research on the safety of prone restraint tactics. Among the studies most widely used to exonerate law enforcement officials in cases of deaths in custody are those conducted by emergency physicians Theodore Chan and Gary Vilke. Drs. Chan and Vilke are part of what the *New York Times* in a December 26, 2021 investigative report described as a "small but influential cadre of scientists, lawyers, physicians and other police experts whose research and testimony is almost always used to absolve officers of blame for deaths."[106] Forming a "cottage industry of exoneration," many of the dozen or so individuals in this group, including Chan and Vilke, have ties with TASER/Axon and/or work as defense experts in death-in-custody litigation.[107]

In 1997, Chan and Vilke sought to determine whether the "hobble" or "hog-tie" restraint position results in clinically relevant respiratory dysfunction. Fifteen healthy volunteers – a small sample size with a questionable ability to generate valid or reliable results – were hogtied. Measurements of lung function decreased by up to 23 percent, which were statistically significant, but the authors deemed them not clinically significant.[108]

In the early 2000s, Chan and Vilke conducted a study in which they placed 25 pounds and 50 pounds on the backs of 10 participants – again a very small sample size – while they were in a prone position.[109] They obtained Institutional Review Board ("IRB") approval from the University of California's Human Research Protection Program for this study.[110]

In 2001, Vilke served as a plaintiff's expert in a restraint asphyxia case when a man with schizophrenia in psychiatric crisis was restrained in a prone position while officers put their weight on his back. At that time, in his deposition, Vilke opined that the weighted restraint killed the decedent. In referring to his studies involving the placement of 25 and 50 pounds on people's backs, he stated that these were preliminary studies only and seemed to suggest that experimenting with greater weights would be unethical due to the possible danger. He noted, "We don't want to put 200 pounds on people and kill them."[111]

After appearing in that case, Vilke took on work as a defense expert in several wrongful death cases against TASER/Axon and law enforcement. Vilke acknowledged in a 2018 deposition that he had worked as a defense expert on behalf of TASER International in "certainly a number of cases" and said he believed that whenever he had testified in cases involving the use of a Taser, he had always testified on behalf of the defense. [112] Further evincing his defense sympathies, Vilke even told a journalist in 2021 that it was "doubtful" that Minneapolis police officer Derek Chauvin had killed George Floyd by pressing his knee on his neck.[113] The *New York Times* reported that in a deposition in summer 2021, "Dr. Vilke said it had been 20 years since he had last testified that an officer was likely to have contributed to a death."[114]

Likewise, in a 2014 deposition, Chan acknowledged that he had been retained by the defense in cases involving the use of a Taser "probably four or five times."[115]

In 2007, Vilke and colleagues published an article titled "Ventilatory and Metabolic Demands During Aggressive Physical Restraint in Healthy Adults," in which they put up to 225 pounds (102.3 kg) on the backs of 30 healthy adults who were restrained in a "hogtie restraint" prone position, with 27 participants told to "struggle vigorously" for 60 seconds.[116] The authors found no clinically significant impairments in breathing (ventilatory function) among participants who were either prone or struggling. The authors reported that they received IRB approval from San Diego State and the University of California San Diego (UCSD) Human Research Protection Program for the study. However, repeated efforts by Julia Sherwin to subpoena IRB materials related to this study produced no evidence of

a completed IRB review or approval. This raises concerns about whether this study that has since been used as evidence for the safety of prone restraint law enforcement tactics ever passed the ethical and safety hurdles needed to obtain IRB approval.[117]

In two recent restraint death cases handled by Julia Sherwin, the defendant police officers hired Vilke to testify on their behalf.[118] In both cases, Vilke testified that the officers beating and restraining the decedents in a prone position, putting weight on the victims' backs, and even choking one decedent did not cause or contribute to their deaths.[119]

Role of TASER

TASER/Axon is a U.S. company that develops technology products and weapons for the military, law enforcement, and civilians, including "Taser," a line of so-called "less-lethal" electroshock "stun" weapons. In 2007, TASER purchased 1,000 to 1,500 copies[120] of Di Maio's book on "excited delirium syndrome" and distributed free copies.[121] They also gave out other materials on "excited delirium" at conferences of medical examiners and police chiefs.[122] Since there are only about 500 full-time forensic pathologists in the United States,[123] TASER purchased enough copies of Di Maio's book in 2007 alone to easily cover the entire forensic pathology community, ensuring widespread familiarity with his theory on "excited delirium syndrome."[124]

Di Maio has acknowledged testifying as a paid expert for TASER/Axon multiple times and stated in 2014 that in the cases in which he was deposed, he always gave the opinion that the Taser did not cause or contribute to the person's death.[125]

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American College of Emergency Physicians (ACEP) White Paper

In 2005, TASER's national litigation counsel,[126] Michael Brave, co-founded a corporation entitled the Institute for the Prevention of In-Custody Deaths (IPICD) with another TASER defense expert and consultant,[127] John Peters.[128] In October 2008, IPICD held its "3rd Annual Sudden Death, Excited Delirium & In-Custody Death Conference." IPICD advertised the conference as "the first consensus conference that focuses upon excited delirium," and promised that

"attendees will help make law enforcement, medical, and legal history … focused on arriving at a 'consensus' about excited delirium." IPICD stated that the "findings from this seminal event will then be published in leading medical, legal, and law enforcement journals."[129]

The conference speakers included TASER and/or restraint death defense experts and consultants such as Chan, Di Maio, Vilke, and Wetli, as well as Dr. Steven Karch[130] and Dr. Deborah Mash.[131] The results of the 2008 IPICD conference were published as the "White Paper Report on Excited Delirium Syndrome" by the American College of Emergency Physicians on September 10, 2009.[132] The co-authors of the white paper included Chan, Mash, and Vilke, as well as TASER's medical director, Dr. Jeffrey Ho.[133] Despite the close links between the paper's co-authors and TASER, PHR has been unable to find conflict-of-interest statements or disclosures in connection with the conference or the resulting white paper.

The White Paper Report acknowledges that the pathophysiology of "excited delirium syndrome" is not understood, that there are no tests or standard diagnostic criteria, and that the medical treatment for the "syndrome" is unknown. Regarding the term "excited delirium," the authors assert that the "issue of semantics does not indicate that excited delirium does not exist" and provide similar *ICD-9* (*International Classification of Diseases, Ninth Revision*) codes such as manic excitement, delirium of mixed origin, agitation, delirium, and abnormal excitement which "describe the same entity as excited delirium syndrome." They fail to consider that if manic excitement, delirium of mixed origin, agitation, and abnormal excitement (among other *ICD-9* codes listed) are the same entity as "excited delirium," then "excited delirium" cannot be a unique entity. Their Report also does not consider that the forms of delirium or manic excitement in the *ICD-9* are not considered lethal. The *Diagnostic and Statistical Manual of Mental Disorders (DSM-5*), the main diagnostic tool used by clinicians for psychiatric diagnosis, in fact, recognizes "delirium" as a clinical entity, with "hyperactive," "hypoactive," and "mixed" delirium subtypes, but these do not align with "excited delirium." The Task Force elaborates: "In most cases, the underlying disease will be untreated at the time of [excited delirium] presentation," which suggests that "excited delirium" is a presentation or manifestation of another cause.[134]

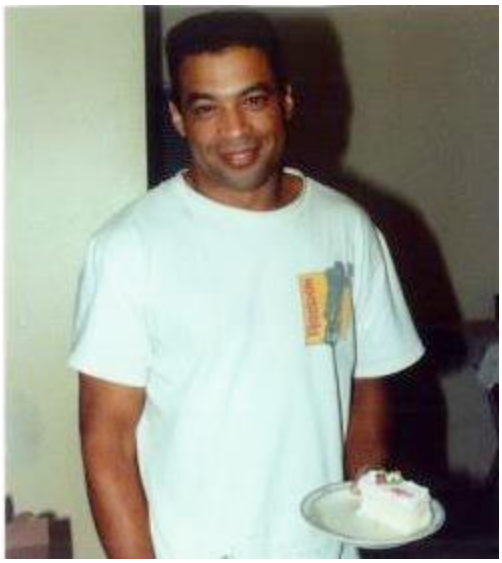
The White Paper Report offers 10 specific features suggesting the presence of "excited delirium" (pain tolerance, agitation, not responding to police presence, superhuman strength, rapid breathing, not tiring despite heavy physical exertion, naked/inappropriately clothed, sweating profusely, hot to the touch, and attraction to/destruction of glass/reflective surfaces). However, it provides no direct citations to the medical literature as to the origins or accuracy of these 10 features in predicting or diagnosing "excited delirium," nor does it comment on the validity of these features as a screening tool. The descriptions of certain symptoms and signs also play into racist tropes that people of color possess "superhuman strength" and are "impervious to pain."[135] This is doubly concerning given that Wetli had asserted without evidence 18 years prior that 70

percent of people who died of cocaine-induced delirium were Black men and that "it may be genetic."[136]

In 2011, the same group of authors published a reiteration of the White Paper Report in the academic, peer-reviewed literature, titled, "Excited delirium syndrome: defining based on a review of the literature."[137] Based on a review of 18 articles, 10 written by the paper's authors, the authors again identified 10 features of "excited delirium."[138] At no point did the authors discuss the lack of and consequent need to develop and test screening tools for "excited delirium" that are valid (able to accurately identify diseased and non-diseased individuals) or reliable (repeat measurements yield the same result). They also provided no statements of conflicts of interest or disclosures.

*A 2008 National Institute of Justice (NIJ) report defined "**excited delirium**" as a "State of extreme mental and physiological excitement, characterized by extreme agitation, hyperthermia, euphoria, hostility, exceptional strength and endurance without fatigue." Of note, the report was written by the then director of the NIJ but included the disclaimer that "Findings and conclusions of the research reported here are those of the authors and do not reflect the official position and policies of their respective organizations or the U. S. Department of Justice."[139]*

The Death of Martin Harrison



Martin Harrison
Photo: Courtesy of the Harrison family.

On August 13, 2010, Martin Harrison was arrested for jaywalking in Oakland, California.[140] A warrant check revealed an outstanding warrant for failing to appear in court on a "driving-under-the-influence" charge, and the police arrested Harrison and took him to the Alameda County Santa Rita Jail.[141] During the intake medical screening process, which occurred at approximately 3:00 p.m., Harrison was visibly intoxicated and smelled of alcohol.[142] He told the licensed vocational nurse (LVN) who conducted the intake medical assessment that he drank every day, that his last drink was that day, and that he had a history of experiencing alcohol withdrawal.[143] The LVN determined Harrison needed no medical care and sent Harrison to the jail's general population without instituting any alcohol withdrawal treatment protocols.[144] Three days later, Harrison experienced severe alcohol withdrawal, or delirium tremens, hallucinating that he was in his apartment and holding his mattress over his head because he perceived people were trying to shoot him. Ten deputies arrived at Harrison's jail cell, Tased him, severely beat him, put a spit hood on him, and forced him into a prone position with officers on top of him, until he died.

Alcohol withdrawal and delirium tremens are considered treatable by medical professionals, yet no medical management was offered at any point during Harrison's stay in jail, including in response to deterioration of his medical condition.

The defendants hired both Di Maio and Wetli as their expert witnesses.

In 2014, Di Maio and Wetli gave sworn deposition testimony in the Harrison case. There was no dispute between the parties that Harrison was experiencing delirium tremens – which, unlike "excited delirium," has

an International Classification of Diseases code – at the time he was severely beaten, Tased, and restrained. Yet Wetli testified in his deposition that Harrison died of "excited delirium" and "is a classic example of death due to excited delirium or the resuscitation that has taken place."[145] Di Maio testified that Harrison's "presentation is of somebody in excited delirium" and "you could argue" that Harrison's death was "a pure excited delirium case."[146]

Despite their assertions regarding "excited delirium," Di Maio and Wetli's depositions confirmed these facts:

- *"Excited delirium" has no International Classification of Diseases (ICD-9 or ICD-10) code, which means it cannot be assigned as a diagnosis or as a cause of death for statistical purposes;[147]*
- *"Excited delirium" has never appeared in any version of the Diagnostic and Statistical Manual of Mental Disorders (DSM), the main diagnostic tool for mental health problems used by physicians and mental health workers in the United States, which is now in its fifth edition;[148]*
- *"Excited delirium" is not recognized by the American Medical Association, American Psychiatric Association, or American Psychological Association.[149]*

The Harrison case settled in 2015 after the first week of an eight-week trial, for $8.3 million, along with changes to policies and training in the fifth largest jail in the United States.[150]

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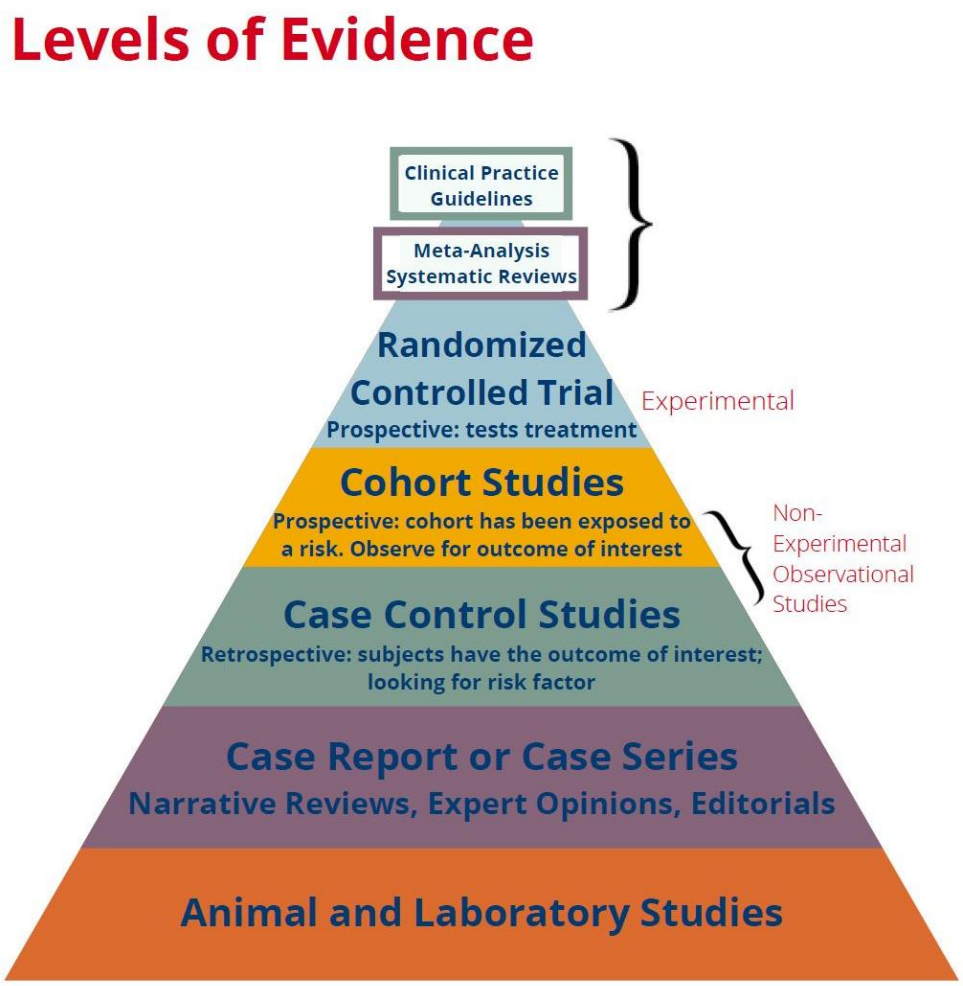
Medical Literature Review

The PHR team explored two main areas of controversy in the peer-reviewed medical literature on "excited delirium": 1) the underlying pathophysiology of "excited delirium;" and 2) "excited delirium" as a cause of death.

Consensus in the Literature that the Pathophysiology of "Excited Delirium" Is Unknown

There is consensus across reviewed articles that the pathophysiology of "excited delirium" is unknown, and that there are no telltale or characteristic autopsy

findings.[151] Many possible causes of the symptoms associated with "excited delirium" are hypothesized. These include a fight-or-flight response (catecholamine surge) resulting in cardiac arrhythmia, disturbances of dopamine and/or dopaminergic pathways, and restraint-related asphyxia or other use of force.[152] Several systematic reviews of the literature on "excited delirium" conclude that the levels of evidence for any postulated etiology are low to very low, and that the overall quality of the studies is poor.[153] For example, a 2018 systematic review found that 65 percent (n = 43) of the articles were retrospective case reports, case series, or case-control studies, all weaker forms of medical evidence.[154]



Hypothesized Roles of Cocaine Intoxication and Neurotransmitters in Symptoms and Signs of "Excited Delirium"

The consensus among the articles included in the review was that Wetli and Fishbain in 1985 introduced into the literature and medical community the concept of "excited delirium" in the context of cocaine use. The authors reported that "excited delirium" was secondary to cocaine intoxication. Therefore, "excited delirium" is a presentation with an underlying cause.[155] Wetli et al. cite the *Comprehensive Textbook of Psychiatry*, chapter 20, written by Dr. Zbigniew J. Lipowski, when defining delirium: "There are two major types of delirium: stuporous … and excited…." Lipowski does not use the term "excited delirium." In fact, cocaine is only referenced in the context of "substance-induced organic mental disorders."[156] It seems that Wetli et al. initially used "excited" as an adjective to portray the hyperactive form of delirium in their case report.

Later, in 1996, Wetli et al. again discussed cocaine-associated delirium and concluded that, "When cocaine users with agitated delirium die, cocaine should be considered the cause of death, unless there is clear physical evidence that death is due to some mechanism other than cocaine toxicity, such as positional or mechanical asphyxia."[157]

The reviewed literature accepts that cocaine interacts with different receptors in the body, including the dopamine system in the brain, by increasing dopamine levels through various mechanisms.[158] Increased release or transport of dopamine is hypothesized in some articles to lead to "excited delirium."[159] However, controversy remains about whether there is any evidence from autopsies that the dopamine system in the brain is associated with "excited delirium."[160]

Other articles have hypothesized that "excited delirium" may be part of a spectrum of other known medical conditions with other neurotransmitters and pathways involved.[161] No reviewed studies provide conclusive evidence for one hypothesized mechanism over another. Similarly, while death from "excited delirium" in reviewed case series were often attributed to acute myocardial dysfunction leading to cardiopulmonary arrest, exact mechanisms leading to this cause of death are not elucidated.

Debate in the Literature on Whether Prone Restraint Positions rather than "Excited Delirium" Are a Cause of Death in Police Custody

Bell and Wetli et al. defined positional asphyxia as the decedent being found in a position that does not allow adequate breathing and having been unable to free themselves.[162]

In 2020, Strommer et al. conducted an extensive review of the literature and converted all relevant "excited delirium" or "agitated delirium" case reports and characteristics in the literature into a numerical dataset for quantitative analysis.[163] They found that some form of restraint was described in 90 percent of all deaths in "excited delirium." Restraint increased the odds of an "excited delirium" diagnosis by between 7 and 29 times.[164]

A central debate has thus been whether restraint positions such as prone restraint can physiologically cause positional asphyxia and death. Some case reports have shown that prone restraint was used during sudden and unexpected in-custody deaths.[165] Studies have attempted reenactment of prone and prone restraint positions, including with compression, with no clear pattern of results.

One of the earliest studies evaluated blood oxygenation and heart rate after recovery from exercise while in a restrained and hogtied position.[166] The study found that it took participants longer to recover in the hogtied position and questioned if this could be worsened during a violent struggle. Later, a different study monitored similar parameters for different types of restraint positions over a longer period of time after exercise, but in obese adults.[167] This study concluded that there were no clinically significant effects. However, its data showed that carbon dioxide elimination was reduced in all restrained positions. None of the studies captured scenarios reflective of police encounters, i.e., involving people who may be struggling and agitated, as opposed to lying at rest, as were the participants in these studies.

Some studies have shown statistically significant decreases in lung function measures during prone restraint positioning, though whether these results were clinically meaningful is not clear.[168] Researchers have found large decreases in lung function and/or other physiologic parameters, such as heart rate and blood pressure, and concluded that some prone restraint positioning should be considered a risk factor for sudden death.[169] Other studies have shown that after applying weight to the torso of prone people, there were reductions in cardiac output, blood flow, and/or the diameter of the inferior vena cava (the large vessel which returns blood to the heart that is then pumped to the lungs to be oxygenated).[170] One study measured the effects of prone positioning and restraint for 10 minutes on adults with chronic obstructive pulmonary disease; almost half were unable to complete the study due to uncontrolled respiratory symptoms.[171]

A 2020 study found that some form of restraint was described in 90 percent of all deaths in "excited" or agitated delirium. Restraint increased the odds of an "excited delirium" diagnosis by between 7 and 29 times.

A 2021 study noted that four prominent factors – physical exertion, prone positioning, restraint, and body compression – had been tested in other studies.[172] The researchers used electrical impedance tomography (EIT) to measure the combined impacts of these parameters on ventilation in 17 healthy human participants. They found that under the combined effects of all these conditions, participants had significant and prolonged declines in lung reserve volumes over time, indicating increased work of breathing compared to the control posture of arms at the side.[173]

The researchers noted that these declines took place with an applied weight of 35 percent participant bodyweight, which the study described as "likely less" than the weight an officer would typically apply in an arrest-related encounter. They hypothesized that in true conditions of weighted restraint, the increasing effort needed to breathe while in a restraint posture would become more relevant to the survival of the participant the longer the weight is applied.[174]

The above studies demonstrated measurable hemodynamic and/or respiratory changes detectable in volunteers who were placed in a prone or prone restraint position in a controlled and mild setting. All of these studies had tiny sample sizes composed of primarily healthy volunteers in well-controlled environments. None of the study participants were intoxicated, fearful, or agitated, within or outside the context of mental illness, and none were being forcibly restrained. Therefore, none of the studies replicated an accurate police encounter with someone supposedly in "excited delirium" who may be struggling and agitated due to restraints, as opposed to laying in rest.

It is not known whether the use of prone restraint in conditions such as the forcible restraint of an agitated person could cause significantly worse hemodynamic or respiratory harms than what was found in these studies.

Regarding all forms of neck restraint, however, a 2009 study found that "A force of only 6kg is needed to compress the carotid arteries, which is about the average weight of a household cat or one-fourteenth the average weight of an adult male."[175] For this reason, among others, the American Academy of Neurology (AAN) has held that neck restraints should be classified, "at a minimum, as a form of deadly force."[176]

Whether Delirium Alone Can Be a Cause of Death

The *DSM-5* recognizes delirium as characterized by "disturbance of consciousness" (i.e., reduced clarity of awareness of the environment), with reduced ability to focus, sustain, or shift attention. The three delirium subtypes are hyperactive, hypoactive, and mixed. Yet, some literature discussed that delirium alone cannot be a cause of death because, by definition, delirium requires an identifiable underlying organic cause that can be ascertained from the clinical presentation, diagnostic studies, or, in the case of death, by autopsy.[177]

Delirium alone cannot be a cause of death because, by definition, delirium requires an identifiable underlying organic cause that can be ascertained from the clinical presentation, diagnostic studies, or, in the case of death, by autopsy.

In their 2020 quantitative analysis on "excited delirium," Strommer et al. discussed the overlap between restraint asphyxia and "excited delirium," in that the characteristics used to describe "excited delirium" are likely to trigger the use of force and restraint, and that risk factors for "excited delirium" overlap with the risk factors for restraint-related asphyxia.[178] This recent review further reinforces that "excited delirium" does not cause death in unrestrained people.

Key Concerns Raised by Review of the Scientific Literature on "Excited Delirium"

The foundations for the diagnosis of "excited delirium" have been misrepresented, misquoted, and distorted. The authors credited with the creation of the term initially used "excited delirium" as a descriptive term for delirium and noted underlying causes. Our examination of the peer-reviewed medical literature on "excited delirium" found that those articles supporting this diagnosis were authored by a small group of people, many of them with ties to TASER/Axon and/or other conflicts of interest. Most of the studies cross-reference each other and highlight non-peer-reviewed sources, such as the Di Maio and Di Maio book *Excited Delirium Syndrome*, which is not a scientific or medical textbook, is not peer reviewed, and draws unsubstantiated conclusions.[179] For example, Di Maio and Di Maio discuss the 1997 study by Chan et al. multiple times. They describe this study as a "death blow" to the positional asphyxia theory and that believing positional asphyxia is possible "involves suspension of common sense and logical thinking." Elsewhere, they state that Chan et al.'s study "disproved" the restraint asphyxia hypothesis. Di Maio and Di Maio are not reporting evidence-based conclusions. Chan's *single* study with a small, non-representative sample size that does not replicate real-life conditions cannot deliver a "death blow."

Most of the reviewed literature suggests a relationship between "excited delirium," death, and restraint. However, these studies have small sample sizes alongside other limitations. The extensive review conducted by Strommer et al. included studies up to April 2020 and summarized all "excited delirium" characteristics. It found that restraint was described in 90 percent of all deaths in the "excited" or agitated delirium medical literature.[180] Notably, they report that asphyxia often lacks pathognomonic signs (clear signs that a particular disease is present) on autopsy.

We found no rigorous scientific research that examines the prevalence of death for people with "excited delirium" who are not physically restrained.

Our review does not allow for conclusive determinations about whether or not restraint or positional asphyxia is the most likely true cause of death for people said to have died from "excited delirium" while agitated and forcibly restrained. All the studies discussed here, however, including those by authors who claim their studies refute restraint asphyxia and those that did not show clinically significant changes in cardiac or respiratory parameters, indeed did demonstrate measurable changes in cardiac and respiratory parameters. It is unknown if they would be clinically significant in a specific real-world situation, but it is notable that there were cardiopulmonary changes even among participants in calm and controlled settings. It is, therefore, reasonable to hypothesize that these cardiopulmonary changes could worsen and become clinically significant in real-world settings. We found no rigorous scientific research that examines the prevalence of death for people with "excited delirium" who are not physically restrained.

Of note, in a December 26, 2021 investigation in the *New York Times*, the authors analyzed more than 230 scientific papers on restraints, body position, and "excited delirium" in the National Library of Medicine database published since the 1980s. They found that nearly three-quarters of the studies that included at least one author who was in the network of TASER/defense experts "regularly supported the idea that restraint techniques were safe or that the deaths of people who had been restrained were caused by health problems." Meanwhile, "only about a quarter of the studies that did not involve anyone from the network backed that conclusion. More commonly, the other studies said some restraint techniques increased the risk of death, if only by a small amount."[181]

Continued Use of "Excited Delirium" to Explain Deaths in Custody and as a Legal Defense to Exonerate Law Enforcement Officials

Despite the problems with its diagnostic underpinnings, "excited delirium" continues to be used to explain deaths in custody. An *Austin-American Statesman* investigation into each non-shooting death of a person in police custody in Texas from 2005 to 2017 found that more than one in six deaths (of 289 total) were attributed to "excited delirium."[182] A January 2020 *Florida Today* report found that of 85 deaths attributed to "excited delirium" by Florida medical examiners since 2010, at least 62 percent involved the use of force by law enforcement.[183] A Berkeley professor of law and bioethics conducted a search of these two news databases and three others from 2010 to 2020 and found that of 166 reported deaths in police custody from possible "excited delirium," Black people made up 43.3 percent and Black and Latinx people together made up at

"Excited delirium" is frequently asserted as a defense by police officers who kill people during the course of restraint.

least 56 percent.[184] Taser use was connected to 47 percent of cases.[185] Similarly, a 2018 study found that the term "excited delirium" has been disproportionately used as a cause of death in cases concerning young Black men.[186]

"Excited delirium" is also frequently asserted as a defense by police officers who kill people during the course of restraint.[187] With notable exceptions, such as the murder prosecutions of the Minneapolis police officers who killed George Floyd, law enforcement officers are usually not criminally prosecuted for restraint-related deaths,[188] and they frequently deploy the "excited delirium" causation defense in civil lawsuits brought against them by decedents' families.[189]

Growing U.S. Medical and Psychiatric Association Opposition to "Excited Delirium" as a Diagnosis

The American Medical Association (AMA) and the American Psychiatric Association (APA) do not recognize "excited delirium" as a valid diagnosis. In 2021 and 2020, respectively, they released statements denouncing a concerning pattern where "excited delirium" is used as a justification for excessive police use of force, particularly when Black men die in law enforcement custody.[190] The AMA elaborated that the term "excited delirium" has been used to justify inappropriate and discriminatory actions. The APA advocated for the U.S. Department of Health and Human Services to conduct a nationwide investigation of all cases labeled "excited delirium." Both associations advocate for cessation of the use of the term "excited delirium" unless a clear set of diagnostic criteria can be established, rigorous studies undertaken, and data made available.

The American College of Emergency Physicians (ACEP), meanwhile, has yet to revise its position that "excited delirium" is a distinct type of delirium. In June 2021, ACEP released a new task force report on "Hyperactive Delirium with Severe Agitation in Emergency Settings" without rescinding the 2009 white paper.[191] The new report emphasized the necessity to "differentiate and treat life-threatening causes of hyperactive delirium," outlined multiple potential underlying causes, and called for additional research to "more fully understand inciting pathways and distinct pathophysiology of individual causes of hyperactive delirium with severe agitation." The report noted concerns about "potential bias" in the 2009 ACEP white paper on "excited delirium syndrome" and stated that since that report's publication, "ACEP enacted a robust global conflict of interest policy, though notably not in direct response to critics of the 2009 white paper nor with specific concerns regarding the content of that paper or others generated before such a policy was in force." Unlike in the 2009 position paper, ACEP this time appended conflict-of-interest disclosures for the members of the task force that produced this new report. However, the 2021

The American Medical Association and the American Psychiatric Association do not recognize "excited delirium" as a valid diagnosis and both advocate for cessation of the use of the term unless a clear set of diagnostic criteria can be established, rigorous studies undertaken, and data made available.

report specified that while its authors were "informed by" the 2009 report, the new report was "de novo and not to be construed as an update *or refutation* [emphasis added] of the 2009 paper."[192]

In February 2022, PHR reached out to ACEP's leadership to clarify their current position in light of their 2009 and 2021 publications.[193] PHR received the following response from Sandy Schneider, ACEP associate executive director, clinical affairs: "We stand by the research presented in our 'ACEP Task Force Report on Hyperactive Delirium with Severe Agitation in Emergency Settings,' published on June 23, 2021."[194]

Equally of concern, the National Association of Medical Examiners (NAME) has not publicly released a statement refuting the validity of "excited delirium" as a diagnosis and cause of death. In February 2022, PHR reached out to NAME's leadership to clarify its current position in light of its 2004 and 2017 position papers[195] referencing the term.[196] PHR received a response from Dr. Kathryn Pinneri, the 2022 NAME president, who attached the 2021 ACEP task force report and said:

> "'Excited delirium' is not recognized as a diagnosis in the World Health Organization International Classification of Diseases (WHO ICD-10). It is a descriptive term used for what is known medically as an acute hyperactive delirium. Acute delirium is a well-recognized diagnosis that is part of both ICD coding and the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) of the American Psychiatric Association.
>
> "A variety of diseases, intoxications, and injuries may result in an acute hyperactive delirium. … Deaths associated with an excited delirium component have also occurred in the absence of police involvement. Forensic pathologists recognize that although a person may be experiencing a hyperactive or excited delirium, that does not mean they died from it. In fact, should a person die after experiencing acute delirium, the cause of death would be the underlying disease, injury or intoxication that caused the delirium.
>
> "Though I suspect it is accepted among many NAME members, we have never issued any type of consensus statement on excited delirium, and as an organization have not formally 'recognized the condition as a

diagnosis.' The NAME Position paper on the Certification of Cocaine-Related Deaths is no longer current and therefore does not reflect our position at this time. We do still support the position paper on in custody deaths."[197]

International Reach of "Excited Delirium"

"Excited delirium" has also received attention in the wake of in-custody deaths in Australia,[198] Canada,[199] the United Kingdom,[200] and elsewhere. The international spread of the term is concerning, but it has far from widespread acceptance.

Australia

According to *The Guardian*, "No Australian medical association recognises 'excited delirium.'"[201] The term has, however, been used by Australian forensic pathologists in specific cases of deaths in custody. Additionally, *The Guardian* identified at least one case in the last five years in which TASER/Axon sent an email to law enforcement the same day as a death that involved Taser use. Law enforcement shared the email with the forensic pathologist on the case, who disregarded it. The email read, "TIMELY AND URGENT AND REQUIRES ACTION WITHIN 24 HOURS OR LESS," offering assistance with the investigation and inviting the police to send brain tissue samples to the University of Miami Brain Endowment Bank to "determine drug abuse and look for excited delirium markers."[202]

Canada

In December 2007, the Commission for Public Complaints Against the Royal Canadian Mounted Police (RCMP) issued recommendations for the use of conducted energy weapons (Tasers), accepting the existence of "excited delirium" as a unique condition and warning that Tasers should not be used against people "experiencing the condition" unless "the behaviour is combative or poses a risk of death or grievous bodily harm to the officer, the individual or the general public."[203]

In June 2008, an independent review of Taser use by the RCMP concluded that "excited delirium" "can be considered to be 'folk knowledge' when used by the police and should not be included in the RCMP's operational manual unless subsequently formally approved by the RCMP after consultation with a mental-health-policy advisory body."[204]

United Kingdom

In May 2016, the Royal College of Emergency Medicine in the United Kingdom issued guidelines for the management of "excited delirium," which they also referred to as "acute behavioural disturbance" (ABD).[205] The term ABD was later

added to the *Maudsley Prescribing Guidelines in Psychiatry*, a handbook for psychiatric medications, prompting the similarly named South London and Maudsley NHS Foundation Trust – the largest public provider of mental health and substance use services in the United Kingdom – to issue a statement noting that the Trust did not recognize either "excited delirium" or ABD as medical terms.[206] The term "excited delirium" is also not recognized by the European Society of Emergency Medicine, an association of emergency physicians from 30 countries.[207]

In 2020, the Royal College of Pathologists in the United Kingdom issued Forensic Science Regulator Guidance about "excited delirium," noting concerns about its use and misuse as a cause of death. The regulator found that "'Excited Delirium' should never be used as a term that, by itself, can be identified as the cause of death. The use of Excited Delirium as a term in guidance to police officers should also be avoided." The regulatory guidance applies in England, Wales, and Northern Ireland.[208]

The Death of Elijah McClain

On August 24, 2019, 23-year-old Elijah McClain was walking home from a convenience store in Aurora, Colorado when he was unlawfully arrested, beaten, and placed in a chokehold. When paramedics arrived, they diagnosed him with "excited delirium" and injected him with ketamine, an anesthetic that can be fatal, in an amount indicated for someone almost twice his weight. McClain went into cardiac arrest in the ambulance on the way to the hospital and died four days later.[209] A forensic pathologist ruled that his death was undetermined but may have been the result of "excited delirium."[210]

"Justice for Elijah McClain" became a rallying cry in the Black Lives Matter movement: a young Black man killed when he was simply walking home had been blamed for his own death at the hands of law enforcement and first responders.[211] McClain's killing also drew nationwide attention to the inappropriate prehospital use of ketamine in response to supposed signs of "excited delirium." A July 2020 investigation by KUNC, a Colorado public radio station, found that medics in Colorado administered ketamine to 902 people for "excited delirium" over two and a half years, and about 17 percent of those people experienced complications.[212] Since then, there have been whistleblower complaints by paramedics reporting that police officers pressured them to administer ketamine against their medical judgment.[213]

In June 2020, the American Society of Anesthesiologists issued a statement opposing the use of ketamine for a law enforcement purpose.[214] In July 2021, Colorado Governor Jared Polis signed a bill

prohibiting the use of ketamine by non-medical professionals and banning its use in response to "excited delirium." In September 2021, a grand jury indicted three police officers and two paramedics for McClain's death, charging them with manslaughter and criminally negligent homicide.[215] In November, the city of Aurora agreed to pay a settlement of $15 million to Elijah McClain's family.[216] The following month, the Colorado Department of Public Health and Environment published a report from its independent ketamine review committee, which stated, "The panel rejected the condition or diagnosis of 'excited delirium' because it lends itself to discriminatory practices that result in systemic bias against communities of color, and because it lacks a uniform definition and specific, validated medical criteria."[217]

> *"The panel rejected the condition or diagnosis of 'excited delirium' because it lends itself to discriminatory practices that result in systemic bias against communities of color, and because it lacks a uniform definition and specific, validated medical criteria."*
>
> Colorado Department of Public Health and Environment, independent ketamine review committee

The Death of Daniel Prude[218]

Daniel Prude arrived at his brother Joe's home in Rochester, New York on March 22, 2020, after his sister reported he had been behaving erratically. When Daniel jumped headfirst down the basement stairs, Joe called 911. Daniel was hospitalized but released later that day.[219] In the middle of the night, Daniel left Joe's home under the influence of phencyclidine (PCP).[220] Joe jumped in his car to try to find Daniel, calling 911 for the second time. Police arrived and told Joe to go home or risk being jailed for violating the coronavirus lockdown.[221]

Soon after, a Rochester police officer arrived at Joe's home. Joe heard on the officer's radio that they had found a man nearby, unclothed. Over the radio, Joe heard an officer at the scene asking the man if he was Daniel Prude, and Daniel responding "Yes." This was the last word Joe heard his brother utter. The officer told Joe that everything was under control. Joe recalled telling him, "My brother doesn't have any weapons on him. And if he's naked, he's no threat to anybody but himself. Don't kill my brother."[222]

When the officers found Daniel, they ordered him onto the ground. He lay face down, putting his hands behind his back, and officers handcuffed him. Police body cameras recorded officers laughing while Daniel was on the ground. When he sat up, officers put a spit hood over his head and face. Soon after, they pinned him face down; he can be heard saying that the officers were "trying to kill me." One officer assumed a three-point "pushup" position with both of his hands on Daniel's head, stretching his legs out and focusing his weight onto Daniel's head.[223] He held that position for more than two minutes, while a second officer put his weight on Daniel's back, and a third officer held Daniel's legs down.[224] Daniel vomited and became unresponsive.[225] After about 18 minutes of resuscitation attempts, Daniel's circulation returned, but he remained unconscious and unable to breathe on his own.[226] He was transported to the hospital, where he was pronounced dead one week later.[227]

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Daniel Prude (right), with his brother Joe Prude. Photo: Courtesy of Joe Prude

The night of the police encounter, an officer falsely told Joe that his brother had died at the scene. "It took seven days for me to find out that my brother was on life support," Joe said.[228]

On May 5, 2020, the Monroe County medical examiner issued an autopsy report describing Daniel Prude's manner of death as homicide but the cause of death as "complications of asphyxia in the setting of physical restraint due to Excited Delirium due to Acute phencyclidine [PCP] intoxication.[229]

For months, the Rochester police chief and other city officials sought to delay the release of video footage from that night, knowing it would ignite public outrage.[230] *A Prude family attorney submitted a Freedom of Information Law request for the video footage in April 2020, but the city did not send him and his team copies of the video until August.*[231]

Grand Jury Proceedings and "Excited Delirium"

In September 2020, after the video became public, New York Attorney General (A.G.) Letitia James announced that she would empanel a grand

"This is something I've got to live with the rest of my life – seeing that video tape playing over in my head."

Joe Prude, brother of Daniel Prude

jury to consider charges against the officers who restrained Daniel.[232] The A.G.'s office retained as one of its prosecution witnesses the defense expert Dr. Gary Vilke to testify about "excited delirium,"[233] unaware that Vilke had previously given multiple interviews in which he expressed doubt about the police officers' responsibility for Daniel's death.[234] Vilke testified at the grand jury that Daniel died from "PCP induced excited delirium, leading to cardiac arrest." He told the grand jury he "wouldn't do anything differently" than what the officers had done. "My opinion is that none of the officers, their impact, individually or collectively, would have caused or contributed to that cardiac arrest."[235]

On February 23, 2021, it was announced that the grand jury had decided not to indict the officers who had restrained Daniel, which James described as "very, very disappointing."[236] Almost two years after his brother's death, Joe reflected on the pain of losing him in this way: "This is something I've got to live with the rest of my life – seeing that video tape playing over in my head."[237]

Training Recommendations and "Excited Delirium"

The same day that the grand jury decision was announced, the A.G.'s office released a report on its investigation into Daniel's death, which included among its recommendations, "Law enforcement officers, emergency communications providers (dispatchers), and emergency medical service personnel must be trained to recognize the symptoms of excited delirium syndrome and to respond to it as a serious medical emergency."[238] The report acknowledged that "excited delirium" "can be controversial and for good reason," noting that the purported symptoms "overlap with racist stereotypes of Black men," which "continue to put Black people in danger." Yet the report gave credence to the medical literature on "excited delirium" and the 2009 ACEP white paper, stating "we are unaware of any scientific studies in peer reviewed literature endorsing the notion that ExDS [Excited Delirium Syndrome] is a concocted, false finding that was generated to shield police misconduct."[239]

One month later, in the wake of media scrutiny related to the office's decision to retain Vilke and its acceptance of "excited delirium" as a valid diagnosis,[240] the A.G.'s office modified its training recommendations in a report of an investigation into the 2019 in-custody death of Troy Hodge.

The office removed the term "excited delirium," instead recommending that "law enforcement officers, dispatchers, and EMS personnel must be trained to recognize that when people display a unique constellation of symptoms, it can signal potential, imminent medical distress; response protocols and training must be structured accordingly."[241]

The A.G.'s office described this constellation of physical signs in the following manner:

> *"The most common type of presentment this office has observed involves individuals under the effect of a stimulant drug – most commonly cocaine. The individuals have generally been observed to be in a condition indicating some sort of detachment from reality and police have been summoned because of bizarre and/or violent and erratic behavior. Further, the individuals involved in our cases have often been highly sweaty or attired in clothing inappropriate for the existing weather conditions and/or surroundings. After police restrain these individuals, they have resisted the restraint and fought, seeming not to tire until, quite suddenly, they have become silent. The death is nearly always attributed to cardiac arrest or acute drug intoxication."*

Although this description appeared to be a re-packaging of some of the purported physical signs of "excited delirium," the report appropriately noted the need for a "coordinated response" to medical emergencies.[242]

However, the report also included the caveat:

> *"In addressing this issue, we are not suggesting that restraint does not contribute to the death of individuals experiencing this condition. To the contrary, our experience with cases over which we have had jurisdiction has informed us that individuals exhibiting these symptoms are particularly vulnerable to the stress and rigor of restraint, particularly when they struggle against it, are largely impervious to pain, and do not fatigue normally."*[243]

While the report importantly noted the possible contribution of restraint to the observed deaths, PHR is concerned that the explanation above continues to pathologize a potentially normal and instantaneous human response ("struggle" against restraint) and uses language that reinforces racist tropes ("impervious[ness] to pain").

The Death of Angelo Quinto[244]

On December 23, 2020, Bella Quinto-Collins called 911 seeking help for her 30-year-old brother Angelo Quinto, who was exhibiting agitation and other signs of a mental health crisis at their home in Antioch, California.[245] When two police officers arrived, they pulled Quinto from his mother's arms onto the floor. At least twice, Quinto's mother, Cassandra Quinto-Collins, heard him say to the officers, "Please don't kill me." Bella and Cassandra then watched in disbelief and horror as the two officers knelt on Quinto's back for five minutes until he stopped breathing. Three days later, Quinto died in the hospital.[246]

Cassandra recalled that shortly before paramedics arrived, the officers turned Quinto on his side, saw blood coming from his mouth, and asked if Quinto had taken any drugs.[247] PHR reviewed Cassandra's video recording of the officers' actions and observed that Quinto did not immediately get cardiopulmonary resuscitation (CPR), despite being unresponsive.[248]

Cassandra said that the paramedics' report stated that law enforcement officers reported that Quinto was on methamphetamine and combative, that they had to restrain him, and that the paramedics had been told not to communicate with the family.[249] Later, however, a toxicology report found no methamphetamine in his system, and his mother said he did not use it.[250] "Angelo was not violent. He was not a threat to anyone. He was following all directions," Angelo's stepfather, Robert Collins, said.[251] The video recording confirms that Quinto was not combative.[252]

Angelo Quinto, who died after police restrained him in his home in Antioch, California. Photo: Courtesy of the Quinto-Collins family

Cassandra and Bella recalled how law enforcement officers deflected responsibility for Quinto's condition, sought to place blame on him or his family, and blocked the family from receiving health status updates from Quinto's medical team.

The police department obtained a felony search warrant and searched the Quinto-Collins residence. During the time the search was being conducted, the family was not allowed to reenter their home for eight hours.[253]

At the police station that night, Bella and Cassandra were each questioned separately. One of the officers asked if Cassandra had hit Quinto because he had a bloody nose. She said she had not. Cassandra recounted how the detective questioning her became visibly disturbed when he discovered she had recorded the police encounter in her home. The officer left the room, and Cassandra heard him cursing outside, insisting that police should not let her leave the station until they got a copy of the video, which Cassandra had already offered to share.[254]

At one point that night, Cassandra got a call from Quinto's doctor at the hospital. She took the call on speaker phone, and an officer rushed over and instructed her to ask for a call-back number and then get off the phone. The officer wrote down the number but never gave it to Cassandra. The family later learned that a detective at the Antioch Police Department had told the hospital not to communicate with the family.[255]

Cassandra and Bella recalled how law enforcement officers deflected responsibility for Quinto's condition, sought to place blame on him or his family, and blocked the family from receiving health status updates from Quinto's medical team.[256]

Angelo Quinto (far right) with his family.
Photo: Courtesy of the Quinto-Collins family

It was not until August 2021, eight months after Quinto's death, that the family learned what was asserted to be the official cause of Quinto's death: a forensic pathologist had testified during a coroner's inquest that Quinto died from "excited delirium syndrome."[257]

Robert Collins, Angelo's stepfather, recalled a previous meeting with the family's attorney: "He told us about 'excited delirium'... when you have nothing else, you go with 'excited delirium.'"[258]

"'Excited delirium' has to be debunked," Cassandra said. She spoke about how painful it was not only to lose Angelo but to see law enforcement repeatedly deny the circumstances of his death. "We're already suffering," she said. To see law enforcement "lying about what happened" was "actually more hurtful."[259]

After Quinto's death, the Quinto-Collins family began working with the Justice for Angelo Quinto! Justice for All! Coalition, advocating for both accountability and legislative changes, focusing on positional asphyxia and mental health crisis response. "Justice for Angelo means it won't happen to the next person," Robert Collins said.[260] *In September 2021, California governor Gavin Newsom signed the Angelo Quinto Act, which bans all forms of law enforcement restraints that can cause positional asphyxia, including the "knee to neck" restraint that killed George Floyd and Angelo Quinto.*[261]

"Justice for Angelo means it won't happen to the next person."

Robert Collins, Angelo Quinto's stepfather

A birthday vigil for Angelo Quinto on March 3, 2021.
Photo: Courtesy of the Quinto-Collins family

Key Themes from Interviews with Forensic Pathologists and Other Experts

Several key themes emerged from PHR interviews with nine forensic pathologists and four other physicians: 1) the debunking of the initial attribution of "excited delirium" as a cause of death in Miami in the 1980s; 2) the role of TASER/Axon in efforts to legitimize and increase use of "excited delirium" as a cause of death; 3) concern about the validity of prone restraint studies; 4) lack of meaning of the term "excited delirium;" 5) optimism about decreasing use of the term "excited delirium;" 6) use of the term "excited delirium" as a proxy for restraint asphyxia; and 7) use of the term "excited delirium" to exonerate law enforcement for deaths in custody. Additionally, interviews with many of these physicians, as well as legal, mental health, and substance use disorder experts, touched on recommendations for alternative responses to people in crisis.

Debunking of "Excited Delirium" after Misclassified Homicides in Miami in the 1980s

A number of forensic pathologists whom PHR interviewed were first introduced to the term "excited delirium" through Wetli's work from the 1980s. Dr. Michael Pollanen, chief forensic pathologist for Ontario, Canada and professor of laboratory medicine and pathobiology at the University of Toronto, noted that Wetli's original discussion of "excited delirium" "occurred in a context where there was a sharp rise of cocaine use in the U.S."[262] He described its evolution from a "very classical clinical pathological description" of cocaine-related psychosis to a cause of death. "The root concept is highly useful and valid and helpful except it was extended too much beyond the original description," he said. "Wetli described in a beautiful series of cases the concept of cocaine-related psychosis with a syndrome which included hyperthermia [high temperatures] and rhabdomyolysis [muscle breakdown]…. It was a very robust concept." Pollanen observed, however, that "it has become overgeneralized to 'excited delirium' as a cause of death."[263]

Dr. Joye Carter, forensic pathologist for San Luis Obispo County, California and the first Black American to be appointed chief medical examiner, described hearing the term during her forensic fellowship from 1987 to 1989 in Miami, where Wetli was deputy chief medical examiner. She recalled Wetli speaking about "excited delirium" quite often, although the chief medical examiner, Dr. Joseph Davis, did not use the term.[264] Carter, whose fellowship coincided with the office's investigation of the series of deaths of Black women in Miami, said that she had performed the autopsy for one of the women.[265]

> "During the time period in my training, there was a string of serial murders, which initially were classified as drug overdoses. While I was there, I remember attending a meeting. Dr. Davis had had a monthly

> homicide meeting with all the homicide detectives and all the police agencies…. During that meeting, they were discussing cases that had similarities. Through that discussion they realized they had a serial killer on hand."[266]

Wetli had described the cases using terms that were "very racialized" and "polarizing," she said, referencing his comment, "For some reason the male of the species becomes psychotic and the female of the species dies in relation to sex." In other words, Carter said, "This happened to Black men. Black women were dying because they were having sex with Black men." Shortly after she left the medical examiner's office, she recalled, Davis reclassified those cases. "I believe this was debunked in Miami because of the ways these cases were handled," she said.

Carter questioned whether other forensic pathologists who view "excited delirium" as a cause of death "even know the origin of it." "I was there," she said. Those who promote the validity of "excited delirium" as a cause of death "don't even acknowledge the fact that we had a string of homicides of Black women that were initially attributed to, 'Oh now we have it in Black women.'"[267]

"I honestly think that we need to get to the historical reference of 'excited delirium,' where it came from, why it was debunked, and why it's so harmful to just throw these categories on individuals," she said.

The Role of TASER/Axon in Efforts to Legitimize and Increase Use of "Excited Delirium" as a Cause of Death

Interviewees described multiple efforts by TASER/Axon to promote the diagnosis of "excited delirium." Dr. Roger Mitchell, chair of the department of pathology at Howard University and a forensic pathologist, recalled first seeing the term in Di Maio's book and then hearing it at an IPICD conference in Las Vegas as a young forensic pathologist.[268] Several other interviewed forensic pathologists noted that Di Maio was well known in the field.[269] Mitchell described him as a "mainstay in forensics. At the time, he was one of the most visible forensic pathologists and productive forensic pathologists."[270] Dr. Michael Baden, a forensic pathologist and former chief medical examiner of New York City, recalled attending an American Academy of Forensic Sciences annual meeting where TASER had a booth and was distributing free copies of Di Maio's book.[271]

Multiple forensic pathologists referenced the chilling effects of TASER's lawsuits against medical examiners who had attributed in-custody deaths in part to Taser use. *"You literally get this letter threatening you if you say Taser was the cause of death."*

Dr. Joye Carter, forensic pathologist, San Luis Obispo County, CA

Dr. Martin Chenevert, an emergency medicine physician at UCLA Santa Monica Medical Center, only recently encountered the ACEP white paper. "It just seemed like kind of junk science.... There's clearly a lot more work that needs to be done. However, it's clear that it's not a real syndrome, more just a collection of symptoms.... [The white paper] clearly had an agenda." He said the paper described findings of lethal toxicity without any kind of clear biological mechanism. He also noted that many of the white paper authors' TASER affiliations were a "huge red flag."[272]

Multiple forensic pathologists referenced the chilling effects of TASER/Axon's lawsuits over the years against medical examiners who had attributed in-custody deaths in part to Taser use.[273] Carter said, "You literally get this letter threatening you if you say Taser was the cause of death. They're literally threatening the medical examiner with lawsuits."[274] Dr. Judy Melinek, CEO of PathologyExpert Inc. and contract forensic pathologist for Communio Inc. in Wellington, New Zealand, also noted the "silencing effect" the lawsuits had on medical examiners: "Nobody wants to get sued."[275]

Concern about the Validity of Prone Restraint Studies

Several forensic pathologists and other physicians expressed concerns about the validity of the studies on which Vilke and colleagues based their arguments that restraint was not dangerous. Dr. Michael Freeman, a forensic epidemiologist and associate professor of forensic medicine at Maastricht University in the Netherlands, described the studies as "blatantly unscientific research that proposes that it's essentially impossible to kill somebody with restraint."[276] He added, "That particular brand of science was developed for litigation support, in order to protect officers who may have been involved in the wrongful death of someone they were restraining."[277]

Others emphasized the studies' artificial conditions with healthy, non-stressed participants. As Dr. Kris Cunningham, the deputy chief forensic pathologist for Ontario, Canada and a cardiovascular pathologist, noted:

> "There are lots of problems with a number of the studies that have been done in the past, where they take a bunch of medical students and put

> them in prone positioning and restrain them. And, lo and behold!, they don't become hypoxic. Well, that's great, but you're also not in pain and upset because a police officer is on your back. It's a very artificial situation."[278]

Pollanen also emphasized how dissimilar conditions in these studies were from real-life conditions:

> "Part of the problem with the restraint asphyxia critique is that a lot of the experiments – all of the experiments – are done with healthy ambulatory people in prone restraint…. How is that medically or physiologically comparable to prone position restraint of someone who is under maximal adrenergic stimulation, whose oxygen demand is high?"[279]

Lack of Meaning of the Term "Excited Delirium"

While the physicians we interviewed did not agree about whether the term "excited delirium" should ever be used to describe signs and symptoms, those interviewed all agreed that there continues to be no consensus on its meaning. Some, such as Freeman, concluded, "'Excited delirium' is a contrived term. It doesn't mean anything as a cause of death."[280] Others described "excited delirium" as "a widely overused term that we don't really have a meaning for" (Dr. Jared Strote, an emergency physician and professor of emergency medicine at the University of Washington), a "very nebulous concept" (Cunningham), and "an unfortunate mishmash of concepts when you view it from a critical point of view" (Pollanen).[281]

Pollanen did posit that there is a series of behavioral features that can be abbreviated in short form as "excited delirium," but he concluded that this summary description should not be used for any causal conclusions:

> "We do that all the time in medicine. We find denoting terms that describe something, and we use that. When we do that in medicine, we usually don't attach causal relevance to it. It's just a short form. The problem with 'excited delirium,' if you then apply a causal relevance, i.e., it can be a cause of death, the problem is there's no way of differentiating someone with 'excited delirium' from someone who is just really agitated."[282]

Pollanen, therefore, described the use of "excited delirium" as "almost a nomenclatural error": "It goes without saying that the whole thing has just become progressively modified in an inappropriate manner. The concept has evolved in a way that the evidence does not support in fact."[283] Others described the term as a "generic term that applies to a confluence of symptoms" (Melinek) and "a controversial theory that describes the final common path triggered by

different substrates resulting in an increased level of catecholamines" (Dr. Enrico Risso, deputy chief medical examiner in Edmonton, Alberta, Canada).[284]

Regardless of their views on whether or not "excited delirium" should ever be used to describe any particular constellation of symptoms and signs, the majority of the experts interviewed held that "excited delirium" should not be considered a cause of death. As Chenevert said, "As a primary cause of death, I just can't see it."[285]

Optimism about Decreasing Use of the Term "Excited Delirium"

Some forensic pathologists and other physicians were optimistic that the term "excited delirium" was falling increasingly out of favor in recent years.[286] Cunningham characterized it as "a concept that had much more appeal in the past than for a lot of pathologists today."[287]

Pollanen said, "'Excited delirium' as a cause of death is not fit for purpose in the 21st century, based on all the things we know now."[288]

Several respondents speculated about possible reasons that the term may be less frequently used. Mitchell cited better research: "As we get more information, the medical community, particularly the forensic pathology community, needs to be able to adjust to the information in front of them versus being dogmatic in our diagnosis."[289] Some attributed the increasing skepticism about the term to the rise of cellphone videos that capture the reality of police encounters, as Freeman has noted.[290]

Mitchell elaborated on this possible explanation:

> "It's a diagnosis that was used when you didn't have cameras. We didn't have direct objective evidence of the altercation with police or its severity. It is as if we are saying someone self-combusted. They started shaking, and they blew up, and now they're dead. Now we're seeing the actual footage of what is happening, law enforcement is standing on people's backs. Imagine five grown men physically subduing an individual. Yes, he may have been intoxicated, but he would have gone home intoxicated, had he not been in that altercation…. It's 2021. We have cellphone video … eyewitnesses. People are not scared to say what they're seeing. It's a different world."[291]

Even forensic pathologists we interviewed who did not object to others using the term "excited delirium," such as Risso and Dr. Soledad Martinez, a forensic pathologist with Chile's Medical Legal Service, noted they would not use it themselves.[292] Risso said, "In the majority of cases, it is not provable at autopsy, and I prefer to describe the underlying pathologic findings."[293] Martinez said, "I

"'Excited delirium' as a cause of death is not fit for purpose in the 21st century, based on all the things we know now."

Dr. Michael Pollanen, chief forensic pathologist for Ontario, Canada

try to use not a single diagnosis: death in a man with cocaine, agitation, and physical restraint. [I'm] trying to show the complete spectrum of the death."[294]

Other forensic pathologists also expressed a preference for a descriptive narrative and referring to the underlying disease or circumstances. Cunningham said that when he determines a cause of death, "It's circumstance-dependent."[295] Mitchell said, "I've been more descriptive of what my findings are. An example may be, blunt force trauma with acute cocaine toxicity during police restraint. Homicide .... I would rather describe the pathology than put it into a syndrome like excited delirium."[296] Carter explained, "When you tell the story of death, you have an opportunity to put down the primary cause of death. Then you have underlying conditions."[297]

Use of the Term "Excited Delirium" as a Proxy for Restraint Asphyxia

Several forensic pathologists and other physicians criticized the use of "excited delirium" as a proxy for restraint asphyxia during law enforcement encounters. As Freeman said, "The evidence indicates that it's used improperly or unknowingly as a proxy for restraint-related asphyxia." He proposed that one should consider so-called "excited delirium" deaths through the lens of counterfactual causation, a concept borrowed from epidemiology. "Take away the restraint, what are the chances the conditions present in the restrained individual kill him at that discrete point in time?"[298] He added:

> "There is this unproven hypothesis that 'excited delirium' is this unique pathophysiologic process that causes sudden death, and it's the decedent's fault because they took drugs, leveraged by the absurd theory that restraint can't kill you if it is applied by law enforcement."[299]

Many of the interviewed forensic pathologists linked use of the term "excited delirium" with maneuvers that could cause asphyxia. Cunningham said "excited delirium" "may be associated with certain things like chest compression, neck compression, prone positioning, restraint."[300]

Carter said that if cocaine is present, but the person would not have died without the restraint, "I'd say call it what it is. It's still a result of restraint asphyxia."[301]

"A cause of death that can only happen at the hands of cops is not a pathophysiologic process, but rather a semantic ploy designed to immunize police against scrutiny of deaths occurring during restraint."

Dr. Michael Freeman, forensic epidemiologist and associate professor of forensic medicine, Maastricht University

The interviewed forensic pathologists noted that it is still unknown but likely that a person exhibiting physical signs attributed to "excited delirium syndrome" would also have a heightened risk of death by restraint. Freeman described both the uncertainty and the possibility as follows: "The unknown variable is what that person's oxygen needs are at that specific point in time." A "person most likely to be adversely affected by restraint" is the "person with highest oxygen needs, person who is agitated, has been running around screaming."[302] Strote also noted the possibility of increased risk of death for a restrained person who is agitated and under stress: "Is it more likely that an 'excited delirium' patient would die than one of the three of us [referring to himself and his PHR interviewers]?" Yes, he said, "But because they are already in a hyper-adrenergic state. Adrenaline going. Already a stress on their heart."[303]

Mitchell provided an illustrative example to reinforce that predisposing conditions cannot be used to mitigate the responsibility of the perpetrator for a death: "We use an example in forensic pathology…. If an 87-year-old woman is walking down the street, and an assailant puts a gun in her face … and she dies [of fright], what's the manner of death? Homicide." [304]

Use of "Excited Delirium" to Exonerate Law Enforcement for Deaths in Custody

Several physicians noted the prevalence of "excited delirium" as an exculpatory term for police killings. Freeman said:

> "It is a term that allows us to ignore police use of force, no matter how extreme, because we have taken the possibility that the police caused the death out of the picture…. A cause of death that can only happen at the hands of cops is not a pathophysiologic process, but rather a semantic ploy designed to immunize police against scrutiny of deaths occurring during restraint."[305]

Strote also expressed this view: "At some point, 'excited delirium' began to be used by police officers and pathologists to explain deaths in restraint, which can spare the officers a potential homicide diagnosis and pathologists the need to describe a clear cause of death."[306]

Other forensic pathologists highlighted the implications of "excited delirium" mainly being used as a cause of death for deaths in police custody. As Baden noted, "If you have a condition or disease, it cannot be due to a boutique, unique condition that almost always causes a death only during a struggle between police officers and a civilian."[307]

No National Standards for Death Investigations

"Right now, there's no federal oversight to medicolegal death investigation in [this] country. It is a county-by-county, city-by-city, state-by-state construct, and it's a milieu of sheriff-run organizations to politician-run organizations to forensic pathologist-run organizations. We need one system. Uniformity of practice should be our goal … [ensuring] accreditation and oversight of forensic pathology ... and medicolegal death investigation.

"There has been no report [or data] from the Death in Custody Reporting Act passed in 2013 … and that's mandated as law. My solution is ... a checkbox on the U.S. standard death certificate … to allow physicians, whose job it is to sign a death certificate, to ... identify deaths in custody. It's so critically important for there to be an objective measure of deaths in custody, and that needs to happen at the level of the physician, in addition to circumstantial data from the law enforcement agency.

"Then there needs to be death-in-custody fatality reviews. We know as a public health construct how to research a problem and then set standards in place.... It's time for the public health infrastructure … to define deaths in custody as a public health issue."

Dr. Roger Mitchell, chair of the department of pathology at Howard University

Recommendations for Alternative Responses to People in Crisis

Many interviewees – physicians, lawyers, mental health experts, and others – emphasized the need for a different kind of emergency response for individuals in crisis.

Changes in Police Procedures and Emergency Response Protocols

Some focused on the particulars of police training, such as the need to place individuals in a recovery position or to avoid prone restraint. According to Strote, the goal of the emergency response should be "to maximize the best balance of protection for others and minimizing harm to that person."[308]

"It's time for the public health infrastructure … to define deaths in custody as a public health issue."

Dr. Roger Mitchell, chair, department of pathology, Howard University

Melinek also advocated for changes to police procedures: "In many cases, police officers aren't taught or aren't trained that if they do a carotid hold, they can kill somebody.... During the lectures I was giving, I made a point of saying if the medical examiner is saying that something you have done has killed the patient/subject, that is another opportunity to ask: is there something in our procedures that needs to change?"[309]

Other interviewees discussed the need for better training and protocols for dispatchers and other first responders to mobilize appropriate resources beyond or instead of police to respond to an emergency call. Jack Ryan, a retired captain from the Providence, Rhode Island police department who now conducts trainings for law enforcement and policy and procedure audits for law enforcement agencies, recommended that Emergency Medical Services (EMS) dispatchers be trained to recognize signs that an individual is experiencing a health crisis and coordinate a multi-disciplinary response, where the objective is for the person to receive medical help as soon as possible.[310]

> "Remember you can restrain somebody on the stretcher to the gurney rather than prone on their stomach and holding them down. And many times, these EMTs have soft restraints as opposed to some of our hard mechanical restraints."[311]

He said that for these types of crises, "the plan should be similar. Can we slow this thing down?.... Let's get sufficient resources there. Let's try to diminish the prolonged struggle. Let's try to turn them [over] to medical.... We don't stabilize by putting a knee on someone's neck or on someone's back or crushing their heads into the ground." He added, "I do think we should train officers on symptomology of crisis... But remember that symptomology seems to run across the board between mental health crisis, sometimes medical crisis, sometimes drug-induced crisis."[312]

Ryan further noted that officers should be trained to avoid putting weight on an arrestee's back while they are prone, and once the arrestee is handcuffed, officers should turn them on their side or sit them upright, to facilitate breathing. Ryan also stated:

> "I think some of the issues go beyond law enforcement. We know with de-institutionalization ... law enforcement has become the catchall at the end of the day. They say LA County Jail is the largest mental health institution in the U.S. I do some audits of jails. It is so disheartening to see the jail

> stuck with people because there's no other place for them to go.... I think we should have a better system so that all of these folks don't fall at the hands of law enforcement."[313]

Medical and Behavioral Health Response Teams and Support Systems

Civil rights attorney Dale Galipo agreed with the need for medical responses to many requests for help that currently go to law enforcement: "One could argue when the police encounter someone that they claim is in this 'excited delirium' state, that's a medical emergency, so that person needs medical treatment. That person doesn't need force used against them. They don't need to be held down. That is the worst thing you can do for someone in a medical emergency."[314]

Others emphasized the limits of seeking to improve police training to respond to mental and behavioral health crises. Civil rights attorney Jim Davy observed, "The majority of violence and law enforcement-created injuries and civil rights violations I have seen primarily fall into two categories: someone was trained and did the thing they were explicitly trained not to do, or they did the thing they were trained to do, and they were trained to do things that violated people's civil rights." Police officers are not the best positioned to respond to a mental health crisis, he said. "I think we have responsibility as a society to be doing something better, different, more responsive."[315]

A federal law passed in March 2021 allocated $25 million to states to support non-law enforcement mobile crisis teams.[316] To better understand what such other models could look like, PHR consulted experts at the National Harm Reduction Coalition, Treatment Advocacy Center, Crisis Assistance Helping Out On The Streets (CAHOOTS), and Portland Street Response.

Dr. Kimberly Sue, medical director of the National Harm Reduction Coalition, offered examples of alternative spaces to support people in substance use crises, including the new San Francisco Drug Sobering Center and the People's Harm Reduction Alliance in Seattle, which provide drop-in spaces for people experiencing the effects of methamphetamine and other substances.[317]

Elizabeth Sinclair Hancq, director of research at the Treatment Advocacy Center, said that the organization's stance is: "It shouldn't be the situation where people are reaching a crisis point, and law enforcement has to intervene." The goal should be "building up an adequate support system and mental health treatment system."[318] As Sabah Muhammad, attorney and legislative and policy counsel at the Treatment Advocacy Center, noted, in supporting the need for systems to be in place to prevent crises: "Families with a loved one with untreated mental illness live with crisis every day.... What is being overlooked is our daily condition of crisis."[319] A September 2020 Treatment Advocacy Center report found that in seven states, a person has to pose an "imminent threat" before they can be involuntarily hospitalized.[320] Muhammad spoke about the way that such state

involuntary commitment statutes force families to call the police to get help for loved ones with severe mental illness. Changing such laws, she said, would help empower families to get treatment for their loved ones before their only remaining option was a potentially life-threatening police encounter.[321]

Hancq identified three types of crisis response models: Crisis Intervention Teams (CIT, law-enforcement-based response), co-responder teams (law-enforcement-based mental health response), and mental health crisis teams (mental-health-based response).[322] Muhammad said of the various models, "All of them are in an infancy. And they are very state-based. If certain models work in one area of the country, they don't necessarily work in another." She expressed hope that the more frequently clinicians and social workers are integrated into these models, the more families can access wraparound services or relationships of trust, and "something can be established that looks more like long-term treatment… because when you just sit around and wait for crisis, you are just expecting entire communities to suffer until they are going to be maimed or die."[323]

Muhammad emphasized, "If it does turn into an emergency, police just should not be first. They can be part of the team if there is a weapon. Someone else with medical training, crisis training – clinician, doctor, social worker – needs to be informing police of their next step."[324]

She explained, "We're missing so many opportunities to be reasonable. To tap into our humanity. To take the time it takes to realize someone is in the middle of a delusion or hallucination. This is something that should not be done quickly. It is something that should be done to preserve the life of the person."[325]

Tim Black, director of consulting at the White Bird Clinic, which runs the mobile crisis intervention program CAHOOTS in Eugene, Oregon, emphasized, "Any sort of mobile crisis system needs to be first informed by community and then providers." In the context of limited resources, it is more important for the community to strengthen the social safety net than to "bring in mobile crisis teams" because crises are "directly tied to some unmet need." He added, "It's really easy and really popular to talk about mobile crisis [programs] but not about the resources that are needed … rapid access and connection to those resources." Such resources, he said, could include shelter, hygiene, food access, 24-hour mental health resources access, violence interruption, homeless outreach, street medicine, and harm reduction.[326]

Black further noted that the White Bird Clinic does not require its crisis workers to be licensed mental health or health care workers prior to their hiring, which would create impediments to staffing the positions, especially in smaller communities. Instead, the clinic is open to recruiting and then training and credentialing crisis response team members who have a variety of life experiences and educational backgrounds.[327]

Robyn Burek, program manager at Portland Street Response, said she has spoken to "probably 100 different cities" about their models for mobile crisis response. "Everybody has a slight variation in how they're running this. I think that's amazing." She said that the "common thread" that flows through all these models is the need for funding streams at both the federal and state level to allow flexibility to have different models.[328] Black agreed. "There's no one prescribed funding mechanism that works for each community."[329]

Legal Framework

U.S. Law

Allowance of "Excited Delirium" as a Diagnosis in U.S. Courts – Despite No Consensus on its Meaning

A review of legal cases discussing "excited delirium" indicates that the term appears to be limited to cases involving interactions of individuals with law enforcement. Despite significant challenges to "excited delirium's" validity within the medical community – and the limited context in which it arises – the term has been admitted in U.S. courts as a legitimate diagnosis, including as a direct cause of death.[330]

Given the lack of an underlying description of "excited delirium" in diagnostic manuals, legal cases have found a clear definition of the term to be elusive. Consequently, "excited delirium" in a police setting has been considered a reasonable medical diagnosis for an extremely broad array of signs and symptoms. It might be described as a state of agitation, excitability, or paranoia.[331] It might include bizarre behavior, confusion, delusions, hyperactivity, incoherence, or yelling.[332] It is often, although not necessarily, associated with drug use.[333] And, ultimately, it is so broadly defined that it might include the observable manifestation of almost every psychiatric or drug-induced behavior. Beyond even this, "excited delirium" has been described by courts to include superhuman strength and imperviousness to pain.[334] While this is generally asserted to be brought on by an underlying history of drug use or mental illness, it has also been described as being initiated by "physical stress."[335] One court even found excessive "sweating" to be indicative of "excited delirium."[336]

Admission of Expert Testimony on "Excited Delirium"

All courts perform some kind of "gatekeeper" function regarding the admissibility of expert testimony. At the federal level and in many states, in performing this "gatekeeper" function, courts make a preliminary assessment of whether the

expert testimony's underlying methodology is scientifically valid and can properly be applied to the facts at issue.[337] Important factors that have been considered in the context of "excited delirium" include whether the theory "has been subjected to peer review and publication" and whether it has attracted "widespread acceptance" within a "relevant scientific community."[338]

After assessing those factors, courts often admit expert testimony on "excited delirium" as evidence at trial, finding that arguments against the theory should go to its persuasiveness as evidence, rather than to its admissibility. In cases in which plaintiffs have sought to exclude testimony on "excited delirium," courts have pointed to three communities that "generally accept" it as a diagnosis: the American College of Emergency Physicians,[339] forensic pathologists and medical examiners,[340] and many police departments, which train their officers to interpret people's behavior through a lens that assumes many medical or mental health conditions are "excited delirium."[341] Admission of "excited delirium" has also been allowed because "the theory or technique has been published and subjected to peer review."[342] Finally, courts cite the ACEP white paper.[343] One court even described the paper as resulting from ACEP "consensus" that "excited delirium syndrome" "is a unique syndrome which may be identified by the presence of a distinctive group of clinical and behavioral characteristics."[344]

Notably, courts have admitted expert testimony on "excited delirium" even while acknowledging that "excited delirium" is not a validated diagnostic entity in either the *International Classification of Diseases* or the *Diagnostic and Statistical Manual of Mental Disorders*[345] and is not recognized as a medical diagnosis by the American Medical Association, the American Psychiatric Association, or the World Health Organization.[346]

The acceptance of "excited delirium" by U.S. courts underscores the harmful impact of ACEP's 2009 white paper, which it has yet to refute. It also demonstrates the troubling reach of the academic literature on "excited delirium," which persists despite its poor quality, homogenous citations, and embedded conflicts of interest.

Use of "Excited Delirium" as a Defense for Officer Conduct

Given courts' admission of "excited delirium" theory into evidence, law enforcement defendants have also used it as a defense in civil rights cases claiming police brutality or wrongful death. Some courts have used the ever-broadening defense of qualified immunity to shield law enforcement officers from accountability for killing people in the course of restraint, based on claims that the decedent died of "excited delirium." While in other circumstances an officer might be viewed as having used excessive force, the force employed may instead be deemed reasonable when dealing with an individual diagnosed with "excited delirium," with its associations of being "impervious to pain" or having "superhuman" strength.[347] Similarly, deaths which might otherwise be attributed

to asphyxiation as a result of excessive force may instead be explained away as natural or accidental due to the victim's "excited delirium."[348]

Impact of the Pervasiveness of "Excited Delirium" in Police Trainings

The pervasiveness of "excited delirium" within law enforcement policies and training manuals has resulted in a number of lawsuits against police officers for violating their training and mishandling a person claimed to be in "excited delirium." In fact, a number of suits have been brought for not attending to the unique medical needs posed by "excited delirium."[349] Plaintiffs have even gone so far as to state that defendant officers should have recognized that the plaintiff/decedent was experiencing "excited delirium," including its purported "imperviousness to pain, great strength, bizarre behavior, aggression, and hallucinations."[350] By introducing evidence that officers failed to follow trainings in this manner, plaintiff's attorneys validate law enforcement protocols on "excited delirium," perpetuating the term's acceptance in courts at the expense of future victims of police violence.[351]

There are a number of underlying risks presented by the pervasiveness of "excited delirium" within law enforcement policies and training manuals. Myocardial infarctions (heart attacks), drug or substance overdose and withdrawal, oxygen deprivation, and acute psychosis have all been bundled by some law enforcement agencies or trainers under the diagnosis of "excited delirium." Yet, these might require quite different medical interventions in an emergency situation, in contrast to the trained responses to "excited delirium." In the case of *Petro v. Town of West Warwick*, for instance, the dispute as to whether the officers failed to render timely assistance hinged upon whether Mr. Jackson died from "excited delirium syndrome" or sudden cardiac arrest due to primary cardiac disease that had been left untreated.[352] In *Estate of Hezekiah Harvey v. Roanoke City Sheriff's Office*, the defendants' expert, the assistant chief medical officer of West Virginia, concluded that Mr. Harvey "died from natural causes – excited delirium due to chronic schizophrenia with a contributing cause of congestive cardiomyopathy."[353] As such, the defense asserted that it did not matter whether emergency medical personnel had administered antipsychotic medication to Mr. Harvey, who had schizophrenia.[354]

When law enforcement officers are not held accountable for their actions based on a successful defense of "excited delirium," the justice system is doubly hurt. Such a defense not only prevents accountability, it does so on the basis of a diagnosis that has no real medical underpinning. As Physicians for Human Rights explained in a brief to the United States Supreme Court:

> "A civil action under 42 U.S.C. § 1983 is often the only way for a victim of official misconduct to vindicate … federally guaranteed rights. But qualified immunity often bars even those plaintiffs who can prove their case from remedying a wrong: harm, but no foul. Qualified immunity thus

enables public officials who violate federal law to sidestep their legal obligations to the victims of their misconduct."[355]

Indeed, the widespread belief in the existence of "excited delirium" among both law enforcement and the courts has resulted in a perverse paradox: a lack of accountability for police misconduct based on a medically nonexistent explanation for that conduct.

Finally, law enforcement agencies that train their officers on "excited delirium" are doing a disservice to their officers. The agencies are implicitly requiring the officers to diagnose a person's condition, which is not their role. Officers who have concerns about the health status of a person they encounter should instead call for medical back-up.

International Human Rights Law

The United States is also bound by international human rights law, as are the countries to which the term "excited delirium" has spread – Australia, Canada, and the United Kingdom, among others. International law includes important standards related to the multiple contexts in which the term is used, addressing protection from excessive and potentially lethal force; protection from discrimination based on race or disability in encounters with law enforcement; protection from discrimination in accessing treatment for mental health or substance use disorder crises; the necessity of thorough, prompt, and impartial investigations of deaths in law enforcement custody; and the right to an effective remedy.

Right to Life and Protection from Excessive Force by Law Enforcement

The right to life is guaranteed by Article 3 of the Universal Declaration of Human Rights (UDHR)[356] and Article 6 of the International Covenant on Civil and Political Rights (ICCPR), which the United States has ratified and is bound to uphold.[357] All are entitled to equal protection of this right without discrimination, according to Article 7 of the UDHR and Article 26 of the ICCPR.[358]

People of color and people with disabilities, including mental illness or substance use disorders, have the right to protection from discrimination in encounters with law enforcement. Article 5 of the International Convention on the Elimination of All Forms of Racial Discrimination (ICERD), to which the United States is also a party, guarantees "without distinction as to race, colour, or national or ethnic origin … The right to security of person and protection by the State against violence or bodily harm, whether inflicted by government officials or by any individual group or institution."[359] Article 10 of the Convention on the Rights of Persons with Disabilities (CRPD) states, "States Parties reaffirm that every human being has the inherent right to life and shall take all necessary

measures to ensure its effective enjoyment by persons with disabilities on an equal basis with others."[360]

General Comment No. 36 of the Human Rights Committee, the treaty body that oversees implementation of the ICCPR, states, "The use of potentially lethal force for law enforcement purposes is an extreme measure that should be resorted to only when strictly necessary in order to protect life or prevent serious injury from an imminent threat."[361]

The United Nations Basic Principles on the Use of Force and Firearms by Law Enforcement Officials (1990)[362] stipulate that law enforcement agencies should adopt rules and regulations for the use of force within the following parameters:

- The use of force must be minimized, targeted, proportional, and directed at de-escalating violence.
- The use of "less-lethal" incapacitating weapons must be carefully controlled.
- Restraint must be shown in all use of force by law enforcement agents, with a view to minimizing injury and loss of life.

The Basic Principles further state that when the lawful use of force is unavoidable, law enforcement officials should ensure that assistance and medical aid are rendered to any injured or affected persons at the earliest possible moment.[363] Additionally, "Governments shall ensure that arbitrary or abusive use of force and firearms by law enforcement officials is punished as a criminal offence under their law."[364]

Right to Health

Article 12 of the International Covenant on Economic, Social and Cultural Rights (ICESCR) guarantees "the right of everyone to the enjoyment of the highest attainable standard of physical and mental health."[365] The UN Principles for the Protection of Persons with Mental Illness and the Improvement of Mental Health Care further state, "All persons with a mental illness, or who are being treated as such persons, shall be treated with humanity and respect for the inherent dignity of the human person."[366]

Article 5 of ICERD prohibits racial discrimination regarding the right to medical care.[367] Article 25 of the CRPD states that people with disabilities "have the right to the enjoyment of the highest attainable standard of health without discrimination on the basis of disability."[368]

International Standards for Death Investigations and the Right to a Remedy

According to the UN Principles on the Effective Prevention and Investigation of Extra-legal, Arbitrary and Summary Executions:

> "Governments shall prohibit by law all extra-legal, arbitrary and summary executions.... Such executions shall not be carried out under any circumstances including ... situations in which deaths occur in custody.... There shall be thorough, prompt and impartial investigation of all suspected cases of extra-legal, arbitrary and summary executions, including cases where complaints by relatives or other reliable reports suggest unnatural death in the above circumstances."[369]

The UN Manual on the Effective Prevention and Investigation of Extra-Legal, Arbitrary and Summary Executions, commonly known as the Minnesota Protocol and most recently revised in 2016,[370] sets international standards for the investigation of potentially unlawful deaths, including deaths in custody. It states:

> "To discharge these responsibilities properly, forensic doctors, including forensic pathologists, must act independently and impartially. Whether or not they are employed by the police or the State, forensic doctors must understand clearly their obligations to justice (not to the police or the State) and to the relatives of the deceased, so that a true account is provided of the cause of death and the circumstances surrounding the death."[371]

The right to an effective remedy for a violation of the right to life, including the right to judicial remedies, is guaranteed by the UDHR (Article 8), ICCPR (Article 2), ICERD (Article 6), and other international treaties and declarations.[372]

How the Use of "Excited Delirium" in Law Enforcement Protocols, Death Investigations, and Courts Violates International Law

As described above, the term "excited delirium" informs law enforcement responses to people experiencing an array of mental health and substance use disorder crises, as well as other medical emergencies. It is also used by forensic pathologists to explain deaths in law enforcement custody, disproportionately those of Black men, and has absolved officers from liability in both criminal and civil cases.

Some of the purported signs of "excited delirium" that law enforcement officers are trained to recognize ("superhuman strength" and "imperviousness to pain") increase the risk that an officer will employ excessive or lethal force, violating human rights standards on the use of force or, indeed, the right to life itself. These same terms also put Black people and other people of color – in the United States and around the world – at greater risk of harm, given that they exploit racist tropes and perpetuate discrimination against people of color in law

The allowance of “excited delirium” in courts as a defense for officers’ use of lethal force or as an explanation for deaths in custody may foreclose – and has foreclosed – avenues for criminal prosecution or civil liability, violating a core principle of international law: the right to an effective remedy.

enforcement settings. Continued reliance on “excited delirium” thus violates international legal protections from racial discrimination.

People with mental illnesses or substance use disorders also face disproportionate risk of harm – in violation of protections from discrimination based on disability – given that their behavior may overlap with purported signs of “excited delirium.” For someone experiencing a medical emergency, an officer’s belief that the person is experiencing “excited delirium” could also mean denial of access to appropriate medical care – a potential violation of the right to health – and likely a violation of the right to non-discrimination on the basis of race or disability.

The term “excited delirium” is also used by forensic pathologists, medical examiners, and coroners to explain deaths in law enforcement custody, again disproportionately those of Black men. Continued use and acceptance of the term as a cause of death too often impedes a thorough, prompt, and impartial investigation of the death, given that the investigation may end prematurely when “excited delirium” is held to be the cause.

Finally, the allowance of “excited delirium” in courts as a defense for officers’ use of lethal force or as an explanation for deaths in custody may foreclose – and has foreclosed – avenues for criminal prosecution or civil liability, violating a core principle of international law: the right to an effective remedy. Black men are also more likely to have this core right infringed.

Conclusion

“Excited delirium” is not a valid, independent medical diagnosis. There is no clear or consistent definition, established etiology, or agreed upon underlying pathophysiology. As a result, there are no diagnostic standards for “excited delirium.” In general, there is a lack of scientific data, and even the body of literature that mentions “excited delirium” is small and largely written by individuals with rarely disclosed conflicts of interest. Because “excited delirium” is not a valid diagnosis, it should not be used as a cause of death.

The term “excited delirium” cannot be disentangled from its racist and unscientific origins. In the 1980s, “excited delirium” was defined as hyperactive delirium, with aggressive behaviors, and associated with cocaine intoxication. A

Interviewed forensic experts described an alarming pattern of pressure from TASER/Axon when forensic pathologists and/or medical examiners describe law enforcement tactics as contributing to the cause of death.

few years later, Dr. Charles Wetli extended his theory of sudden death from cocaine intoxication to explain the deaths of more than 12 Black women in Miami who, along with at least seven others who were found dead during the same period, were later found to have been murdered by a serial killer. Wetli's grave professional error – and the racism and misogyny that seemed to inform it – should have soundly discredited "excited delirium" as a cause of death at the time, but instead its use grew.

Moreover, the diagnosis of "excited delirium" has been primarily applied to deaths occurring during encounters with law enforcement. If any other medical condition were only or even mostly occurring in a particular environment or context, a scientific approach would require interrogation of that environment as a contributing or causative factor – in this case, police custody.

PHR's review of the literature and interviews with forensic medical and legal experts found that when the diagnosis of "excited delirium" *has* been advanced, it has almost always been by law enforcement and law-enforcement-affiliated organizations, such as TASER International (Axon Enterprise). To the extent that the diagnosis has been raised in the literature by physicians and scientists, they have often been paid by TASER/Axon or law enforcement agencies defending lawsuits arising out of a death, without disclosing these relationships. Interviewed forensic experts also described an alarming pattern of pressure from TASER/Axon when forensic pathologists and/or medical examiners describe law enforcement tactics as contributing to the cause of death.

A diagnosis of "excited delirium" also yields no actionable steps toward what treatment an individual might need. For a diagnostic system to establish itself as scientifically useful, the system itself must be created from reliable and valid definitions and criteria. In the case of "excited delirium," this label certainly does not aid in treatment and has not invited or welcomed research that may better define it or aid in diagnosis, research, or treatment. That a person experiencing agitation, mental illness, or intoxication would need to be restrained, beaten, or choked rather than first treated medically is contrary to medical standards.

People presenting with agitation, confusion, rapid breathing, elevated heart rate, or sweats have an underlying diagnosis. Their signs and symptoms should be named as they are, and the underlying condition should be found and treated medically. Law enforcement should acknowledge that restraint asphyxia is highly possible, if not the most probable cause of death, and, consequently, law

enforcement officials should make every effort not to put a person in a prone restraint or neck restraint.

PHR is concerned that the unscientific diagnosis of "excited delirium" has been used repeatedly over decades to mask deaths caused by inappropriate and often violent law enforcement responses to medical or mental health crises, and to exonerate perpetrators or cover up homicides.

"Excited delirium" is a descriptive term, not a medical diagnosis, and should not be used as a cause of death. PHR has concluded that it is essential to end the use of "excited delirium" as an officially determined cause of death in cases of deaths in police custody or in any other case. This is one critical step among many to stop these preventable deaths, which have to be acknowledged for what they are before a remedy can be found.

PHR is concerned that the unscientific diagnosis of "excited delirium" has been used repeatedly over decades to mask deaths caused by inappropriate and often violent law enforcement responses to medical or mental health crises, and to exonerate perpetrators or cover up homicides.

Recommendations

To the American College of Emergency Physicians (ACEP):

- Revise position on "excited delirium" based on the evidence, recognizing that it is not a valid medical diagnosis and cannot be a cause of death;
  - Note the racist origins and usage of "excited delirium" and the need for further study of racial disparities in its application;
- Rescind all previous white papers that support "excited delirium" as a distinct entity separate from other forms of delirium; and
  - Be transparent about conflicts of interest in previous position statements; implement clear policies on minimizing or eliminating conflicts of interest in future statements.

To the National Association of Medical Examiners (NAME):

- Issue a statement on "excited delirium" based on the evidence, recognizing that it is not a valid medical diagnosis and cannot be a cause of death;

- Note the racist origins and usage of "excited delirium" and the need for further study of racial disparities in its application; and
- Conduct an investigation into structural, political, and other factors affecting the independence of medical examiners when investigating deaths in law enforcement custody, and report the findings publicly.

To Individual Medical Examiners, Forensic Pathologists, and Coroners:

- Ensure that "excited delirium" is not used as either a sole or a contributing cause in death certification.

To Other Medical and Health Professional Associations:

- Study how the involvement of law enforcement in the health context impacts the relationship between patient and health care provider; seek stakeholder input; and
- Establish best practices for communicating with families regarding injuries or deaths of loved ones in law enforcement custody.

To State and Local Governments:

- Address current use of the term "excited delirium:"
  - Instruct state attorneys general to review the use of the term "excited delirium" in all instances by police and correctional services to understand how and when it is applied;
  - Call on police associations and first responders to stop disseminating "excited delirium" protocols and collect data on how the term has been applied, including racial disparities in its use;
- Improve official responses to people experiencing mental and behavioral health challenges:
  - Bolster resources and social services to address community needs, including mental health and harm reduction;
  - Take steps to ensure that medically trained professionals are the primary responders and decision-makers in the management of acute medical emergencies, including mental health and substance use disorder crises;
  - Invest in alternative models of mental and behavioral health crisis response, led by health professionals and/or social workers, rather than law enforcement;
- Enact changes that strengthen oversight and independence of death investigations:
  - Strengthen qualifications and training for medical examiners, forensic pathologists, and coroners;
  - Strengthen institutional protections to ensure the independence of medical examiners, forensic pathologists, and coroners from law enforcement;

- Establish independent oversight systems and mandate independent investigations of deaths in law enforcement custody;
- If a death is indicated on the death certificate as a death in custody, institute rigorous death-in-custody fatality reviews with explicit guidelines;

- Ban the use of neck restraint and weighted or prolonged prone restraint by law enforcement; and
- Fund studies on how the involvement of law enforcement in the health context impacts the relationship between patient and health care provider.

To the Biden Administration:

- Enforce the Death in Custody Reporting Act of 2013 (Pub. L. No. 113-242) that requires law enforcement agencies to report to the Attorney General annually on all deaths in custody within their jurisdiction;
- Enforce the 21st Century Cures Act by requiring the Department of Justice (DOJ) and others to regularly collect and report data related to law enforcement encounters and mental illness;[1]
- Establish national standards across all federal law enforcement agencies for clear procedures in death investigations in federal custody;
- Work with Congress, and state and local governments, to unify national standards for investigations of deaths in custody, including well-supported independent accreditation, investigatory, and oversight mechanisms; and
- Establish a unit within the DOJ to investigate all deaths in custody.

To Congress:

- Exercise Congress's oversight authority in the following ways:
  - Investigate the history and use of "excited delirium" in various jurisdictions across the United States in the context of deaths in police custody, systemic racism, and the pursuit of justice and accountability;
  - Call on the DOJ to enforce the Death in Custody Reporting Act of 2013, which requires law enforcement agencies to report to the Attorney General annually on all deaths in custody within their jurisdiction;
  - Call on the DOJ to enforce the 21st Century Cures Act, which requires the DOJ and others to regularly collect and report data related to law enforcement encounters and mental illness;[2]
  - Develop mechanisms for oversight and tracking of any aggressive tactics used to subjugate or control people in police custody;
- Pass legislation that seeks to direct national standards toward:

[1] We thank the Treatment Advocacy Center for its leadership on this.
[2] Ibid.

    - Quality assurance, and clear required procedures for death investigations and for documenting police violence on death certificates; and
    - Banning the use of neck restraint and weighted or prolonged prone restraint by law enforcement;
- Allocate funding for:
    - A mandated national database tracking law enforcement use of force, including data on mental illness, race, and ethnicity;[3]
    - New or expanded non-law-enforcement emergency mental health services and social services response programs on the state and local levels; and
    - Studies on how the involvement of law enforcement in the health context impacts the relationship between patient and health care provider.

To the U.S. Centers for Disease Control and Prevention:

- Add a required checkbox on the U.S. standard death certificate to enable physicians to report deaths in custody;[4] and
- Undertake a review of deaths in custody as a matter of racial and other disparities in health, including deaths in which the term "excited delirium" was applied to describe the circumstances of death. In this review, analyze the demographics of the people to whom this term is applied, as well as the common situations in which it is invoked.

To UN Human Rights Mechanisms, including the Independent Expert Mechanism on Systemic Racism in Law Enforcement:

- As a function of state reporting and international oversight, study and report on the use of "excited delirium" worldwide to trace the geographic scope of the term's use as an explanation for deaths in custody and its implications for human rights.

[3] Ibid.
[4] This recommendation was suggested to PHR by Dr. Roger Mitchell, chair of the Department of Pathology, Howard University College of Medicine.

Endnotes

[1] Interview with Robert Collins, Bella Quinto-Collins, and Cassandra Quinto-Collins, Oct. 29, 2021. See also, John L. Burris, Ayana C. Curry, Ben Nisenbaum, Dewitt M. Lacy, James A. Cook, Kenneth Chike Odiwe, and Tonia Robinson, Law Offices of John L. Burris, "RE: Request for Endorsed Copy Verifying Administrative Claim Received - Law Offices of John L. Burris," Feb. 18, 2021, https://johnburrislaw.com/Quinto-Claim.pdf; Jacey Fortin, "California Man Died after Police Knelt on Him for 5 Minutes, Family Says," *New York Times*, Feb. 25, 2021, https://www.nytimes.com/2021/02/25/us/angelo-quinto-death-police-kneel.html; Claire Wang, "The Filipino American Family behind Calif.'s New Police Reform Laws Speaks Out," *NBC News*, Oct. 15, 2021, https://www.nbcnews.com/news/asian-america/filipino-american-family-califs-new-police-reform-laws-speaks-rcna3030.

[2] Nate Gartrell and Rick Hurd, "Death of Angelo Quinto, Navy Vet Who Died after Struggle with Antioch Cops, Blamed on 'Excited Delirium,'" *The Mercury News*, Aug. 20, 2021, https://www.mercurynews.com/2021/08/20/death-of-angelo-quinto-after-struggle-with-cops-blamed-on-excited-delirium-a-controversial-diagnosis-the-ama-says-is-used-to-shield-police-violence.

[3] Stacia Glenn, "'Can't breathe': Tacoma police restraint of Manuel Ellis caused his death, medical examiner reports," *Seattle Times*, Jun. 3, 2020, https://www.seattletimes.com/seattle-news/cant-breathe-tacoma-police-restraint-of-manuel-ellis-caused-his-death-medical-examiner-reports/; "Family rallies for man killed in 2017 Omaha police struggle," *Associated Press*, Jun. 6, 2021, https://apnews.com/article/ne-state-wire-omaha-28d4820fecb111234d641f609db031f3; Anica Padilla, "'Excited Delirium': Elijah McClain's Mother Talks To '60 Minutes' About Use Of Ketamine To Sedate Suspects," *CBS Denver*, Dec. 14, 2020; Justin Jouvenal, "'Excited delirium' cited in dozens of deaths in police custody. Is it real or a cover for brutality?" *Washington Post*, May 6, 2015, https://www.washingtonpost.com/local/crime/existence-of-excited-delirium-ruling-in-va-womans-death-has-experts-split/2015/05/06/b1cc9499-ddaa-474c-9e8a-9ae89a9ae679_story.html; Kimberlé Williams Crenshaw and Andrea J. Ritchie, *Say Her Name: Resisting Police Brutality Among Black Women* (New York: African American Policy Forum, 2015) http://static1.squarespace.com/static/53f20d90e4b0b80451158d8c/t/560c068ee4b0af26f72741df/1443628686535/AAPF_SMN_Brief_Full_singles-min.pdf; Lisette Voytko, "Daniel Prude's Autopsy Report Says 'Excited Delirium,' A Controversial Diagnosis, Contributed To His Death," *Forbes*, Dec. 3, 2020, https://www.forbes.com/sites/lisettevoytko/2020/09/03/daniel-prudes-autopsy-report-says-excited-delirium-a-controversial-diagnosis-contributed-to-his-death/?sh=199868b61784.

[4] Alysia Santo, "As George Floyd Died, Officer Wondered About 'Excited Delirium,'" *The Marshall Project*, Jun. 4, 2020, https://www.themarshallproject.org/2020/06/04/as-george-floyd-died-officer-wondered-about-excited-delirium; Steve Karnowski, "EXPLAINER: Why 'excited delirium' came up at Chauvin trial?" *Associated Press*, Apr. 19, 2021, https://apnews.com/article/health-death-of-george-floyd-trials-george-floyd-3b60b3930023a2668e7fc63f903fc3aa; Ja'han Jones, "3 ex-cops charged in George Floyd's death try to blame 'excited delirium' as the cause," *MSNBC*, Jan. 28, 2022, https://www.msnbc.com/the-reidout/reidout-blog/george-floyd-excited-delirium-rcna13990.

[5] Eric Dexheimer and Jeremy Schwartz, "In fatal struggles with police, a controversial killer is often blamed," *Austin-American Statesman*, May 27, 2017, https://www.statesman.com/story/news/investigates/2017/05/27/in-fatal-struggles-with-police-controversial-killer-is-often-blamed/8339157007.

[6] Alessandro Marazzi Sassoon, "Excited delirium: Rare and deadly syndrome or a condition to excuse deaths by police?" *Florida Today*, Oct. 24, 2019,

https://www.floridatoday.com/in-depth/news/2019/10/24/excited-delirium-custody-deaths-gregory-edwards-melbourne-taser/2374304001.

[7] Osagie K. Obasogie, "Excited Delirium and Police Use of Force," *Virginia Law Review* vol. 107, no. 8 (Dec. 2021), https://www.virginialawreview.org/wp-content/uploads/2021/12/Obasogie_Book_107.pdf.

[8] David A. Fishbain and Charles V. Wetli, "Cocaine intoxication, delirium, and death in a body packer," *Annals of Emergency Medicine* vol. 10, (Oct. 1981): 531–532, https://pubmed.ncbi.nlm.nih.gov/7283219/; Charles V. Wetli and David A. Fishbain, "Cocaine-Induced Psychosis and Sudden Death in Recreational Cocaine Users," *Journal of Forensic Sciences* vol. 30, no. 3, (Jul. 1985).

[9] Adrian Walker and Heather Dewar, "Cocaine-Sex Deaths in Dade Probed," *Miami News*, Nov. 24, 1988.

[10] Ibid.

[11] Ibid.

[12] Russ Rymer, "Murder Without a Trace," *In Health*, May/Jun. 1990.

[13] Rosemary Goudreau, "Coroner: Somebody Killed 19 women," *Miami Herald*, May 31, 1989; John Donnelly, "Suspect in Murder of Prostitutes Speaks Out," *Miami Herald*, May 9, 1993; Donna Gehrke, "Missed Calls, Close Calls Mar Serial Killings Case," *Miami Herald*, April 26, 1990; Donna Gehrke, "Cops: Rapist Top Suspect in Murders," *Miami Herald*, Jun. 20, 1989.

[14] Donna Gehrke, "Both Sides Rest in Williams' Rape Trial," *Miami Herald*, Apr. 27, 1990.

[15] Russ Rymer, "Murder Without a Trace," *In Health*, May/Jun. 1990.

[16] Ibid.

[17] Theresa G. Di Maio and Vincent J. Di Maio, *Excited Delirium Syndrome: Cause of Death and Prevention* (New York: CRC Press 2005).

[18] Bernice Yeung, "Taser's Delirium Defense," *Mother Jones*, Mar./Apr. 2009, https://www.motherjones.com/politics/2009/02/tasers-delirium-defense; Gus Garcia-Roberts, "Is excited delirium killing coked-up, stun-gunned Miamians?" *Miami New Times*, Jul. 15, 2010, https://www.miaminewtimes.com/news/is-excited-delirium-killing-coked-up-stun-gunned-miamians-6367399?showFullText=true; interview with Michael Baden, Oct. 7, 2021.

[19] Deposition of Vincent J.M. Di Maio, M.D. at 159:19-21, *Harrison v. County of Alameda*, No. C11-2868 JST (N.D. Cal. Jan. 24, 2014).

[20] Philippe Gonin et al., "Excited Delirium: A Systematic Review," *Academic Emergency Medicine* vol. 25, (May 2018): 552-565, https://doi.org/10.1111/acem.13330.

[21] See, e.g., Royce Kurmelovs, "'Excited delirium': how a disputed US term found its way to Australian deaths-in-custody inquests," *The Guardian*, Jul. 3, 2021, https://www.theguardian.com/australia-news/2021/jul/04/excited-delirium-how-a-disputed-us-term-found-its-way-to-australian-deaths-in-custody-inquests; "Report of the Panel of Mental Health and Medical Experts Review of Excited Delirium", Jun. 30, 2009, https://novascotia.ca/just/public_safety/_docs/Excited%20Delirium%20Report.pdf; Nazia Parveen, "'Excited delirium': term linked to police restraint in UK medical guide condemned," *The Guardian*, Jul. 1, 2021, https://www.theguardian.com/uk-news/2021/jul/01/excited-delirium-term-linked-to-police-restraint-in-uk-medical-guide-condemned.

[22] See, e.g., Arjun S. Byju, "Excited Delirium: How Cops Invented a Disease," *Current Affairs*, Apr. 13, 2021, https://www.currentaffairs.org/2021/04/excited-delirium-how-cops-invented-a-disease; Royce Kurmelovs, "'Excited Delirium': How a Disputed US Term Found Its Way to Australian Deaths-in-Custody Inquests," *The Guardian*, Jul. 3, 2021, https://www.theguardian.com/australia-news/2021/jul/04/excited-delirium-how-a-disputed-us-term-found-its-way-to-australian-deaths-in-custody-inquests; Jennifer K. Brody, Ayana Jordan and Sarah E. Wakeman, "Excited delirium: valid clinical diagnosis or medicalized racism? Organized medicine needs to take a stand," *STAT News*, Apr. 6, 2021, https://www.statnews.com/2021/04/06/excited-delirium-medicalized-racism-organized-medicine-take-a-stand; "Police keep using 'excited delirium' to justify brutality. It's junk science," *Washington Post*, July 17, 2020, https://www.washingtonpost.com/outlook/chokehold-police-excited-delirium/2020/07/17/fe907ec8-c6bc-11ea-b037-f9711f89ee46_story.html; Amnesty

International, "Amnesty International's Continuing Concerns about Taser Use," Mar. 2006, https://www.amnesty.org/en/documents/amr51/030/2006/en.

[23] G. Schwartz and J. Jahn, "Mapping fatal police violence across U.S. metropolitan areas: Overall rates and racial/ethnic inequities, 2013-2017," *PLOS ONE*, vol. 15, no. 6, (Jun. 24, 2020), https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0229686; Frank Edwards, Hedwig Lee, and Michael Esposito, 2019, "Risk of Being Killed by Police Use of Force in the United States by Age, Race–Ethnicity, and Sex," *Proceedings of the National Academy of Sciences* vol. 116, no. 34, (Aug. 20, 2019): 16793-16798, https://www.pnas.org/content/116/34/16793; Stephanie Woodard, "The Police Killings No One Is talking About," *In These Times*, Oct. 17, 2016, https://inthesetimes.com/features/native_american_police_killings_native_lives_matter.html.

[24] American Medical Association, "New AMA policy recognizes racism as a public health threat," Nov. 16, 2020, https://www.ama-assn.org/press-center/press-releases/new-ama-policy-recognizes-racism-public-health-threat; American Public Health Association, "APHA Calls out Police Violence as a Public Health Crisis," Jun. 4, 2020, https://www.apha.org/news-and-media/news-releases/apha-news-releases/2020/apha-calls-out-police-violence; National Medical Association, "NMA Calls for Open Dialogue on Police Brutality," Jun. 2, 2020, https://www.nmanet.org/news/510933/NMA-Calls-for-Open-Dialogue-on-Police-Brutality.htm; see also Rachel R. Hardeman, Eduardo M. Medina, and Rhea W. Boyd, "Stolen Breaths," *New England Journal of Medicine* vol. 383, no. 3, (Jul. 16, 2020): 197–199, https://www.nejm.org/doi/full/10.1056/nejmp2021072.

[25] According to a 2015 Treatment Advocacy Center report, anywhere from 25 to 50 percent of fatal law enforcement encounters end the life of someone with a serious mental illness; individuals with untreated serious mental illness are 16 times more likely to be killed when approached or stopped by law enforcement than other civilians. Doris A. Fuller et al., *Overlooked in the Undercounted*, Treatment Advocacy Center, Dec. 2015, https://www.treatmentadvocacycenter.org/overlooked-in-the-undercounted. A March 2016 study by the Ruderman Family Foundation found that up to half of all people killed by police in the United States have a disability. David M. Perry and Lawrence Carter-Long, "The Ruderman White Paper on Media Coverage of Law Enforcement Use of Force and Disability," Ruderman Family Foundation, Mar. 2016, https://rudermanfoundation.org/media-missing-the-story-half-of-all-recent-high-profile-police-related-killings-are-people-with-disabilities. A June 2020 Mercury News study of 72 police killings in the Bay Area found that two-thirds of people killed had some kind of mental health issue. Thomas Peele, David Debolt, and Robert Solanga, "Exclusive: Blacks Are Only 7% of the Bay Area, but 27% of Those Killed by Police," *The Mercury News*, Jun. 28, 2020, https://www.mercurynews.com/2020/06/28/110-deaths-police-killings-in-the-bay-area-since-2015-result-in-no-prosecutions-of-cops-and-little-discipline See also A. Saleh, A. P. Appelbaum, X. Liu., T. Scott Stroup, and M. Wall, "Deaths of people with mental illness during interactions with law enforcement," *International Journal of Law and Psychiatry* vol. 58, (2018): 110-116 https://pubmed.ncbi.nlm.nih.gov/29853001/.

[26] Mental illness was defined as any mental, behavioral, or emotional disorder in the past year of sufficient duration to meet the diagnostic criteria of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), the main diagnostic tool used by clinicians for psychiatric diagnosis.

[27] A serious mental illness was defined as any mental, behavioral, or emotional disorder that substantially interfered with or limited one or more major life activities.

[28] Substance Abuse and Mental Health Services Administration (SAMHSA), "Key Substance Use and Mental Health Indicators in the United States: Results from the 2020 National Survey on Drug Use and Health," Oct. 2021, https://www.samhsa.gov/data/sites/default/files/reports/rpt35325/NSDUHFFRPDFWHTMLFiles2020/2020NSDUHFFR1PDFW102121.pdf.

[29] Doris A. Fuller et al., *Overlooked in the Undercounted*, 2015.

[30] K. P.Rosenberg, J. DuLong, *Bedlam: An Antimate Journey into America's Mental Health Crisis* (New York: Avery, 2019); C. Montross, *Waiting for an Echo: The Madness of American Incarceration* (New York: Penguin Press, 2020).

[31] Treatment Advocacy Center, "People with Untreated Mental Illness 16 Times More Likely to Be Killed By Law Enforcement," https://www.treatmentadvocacycenter.org/key-issues/criminalization-of-mental-illness/2976-people-with-untreated-mental-illness-16-times-more-likely-to-be-killed-by-law-enforcement; see also David A. Paul, "The Death of Daniel Prude — Reflections of a Black Neurosurgeon," *New England Journal of Medicine* vol. 383, no. 24, e135, https://www.nejm.org/doi/full/10.1056/NEJMpv2030234.

[32] United Nations High Commissioner for Human Rights, "Promotion and protection of the human rights and fundamental freedoms of Africans and of people of African descent against excessive use of force and other human rights violations by law enforcement officers," Jun. 28, 2021, A/HRC/47/CRP.1, https://www.ohchr.org/Documents/Issues/Racism/A_HRC_47_CRP_1.pdf.

[33] Centers for Disease Control and Prevention (CDC), "Investigations and Autopsies," Jan. 15, 2015, https://www.cdc.gov/phlp/publications/coroner/investigations.html.

[34] CDC, "Medicolegal Officers," Jan. 15, 2015, https://www.cdc.gov/phlp/publications/coroner/medicolegal.html.

[35] Radley Balko, "It's time to abolish the coroner," *Washington Post*, Dec. 12, 2017, https://www.washingtonpost.com/news/the-watch/wp/2017/12/12/its-time-to-abolish-the-coroner.

[36] CDC, "DC Medicolegal Officers," Jan. 15, 2015, https://www.cdc.gov/phlp/publications/coroner/medicolegal.html.

[37] M. Hannan, I. Hearnden, K. Grace, T. Bucke, "Deaths in or following police custody. An Examination of the cases 1998/99 – 2008/09," *IPCC Research Series Paper* vol. 17, (2010): 1-93.

[38] National Academy of Sciences, "Strengthening Forensic Science in the United States: A Path Forward," Aug. 2009, https://www.ojp.gov/pdffiles1/nij/grants/228091.pdf.

[39] Harvard T.H. Chan School of Public Health, "More than half of police killings not officially documented on U.S. death certificates," Oct. 10, 2017, https://www.hsph.harvard.edu/news/press-releases/documenting-police-killings; see also Jamiles Lartey, "US police killings undercounted by half, study using Guardian data finds," *The Guardian*, Oct. 11, 2017, https://www.theguardian.com/us-news/2017/oct/11/police-killings-counted-harvard-study.

[40] Justin Feldman, "Want police reform? We need independent medical examiners and coroners," *Washington Post*, Jun. 2, 2020, https://www.washingtonpost.com/opinions/2020/06/02/want-police-reform-we-need-independent-medical-examiners-coroners.

[41] "The Counted: People killed by police in the US," *The Guardian*, https://www.theguardian.com/us-news/ng-interactive/2015/jun/01/about-the-counted.

[42] J. M. Feldman, S. Gruskin, B. A. Coull, N. Krieger, "Quantifying underreporting of law-enforcement-related deaths in United States vital statistics and news-media-based data sources: A capture–recapture analysis," *PLoS Med* vol. 14, no. 10, (2017): 1-20, https://journals.plos.org/plosmedicine/article?id=10.1371/journal.pmed.1002399.

[43] Jamiles Lartey, "US police killings undercounted by half, study using Guardian data finds," *The Guardian*, Oct. 11, 2017, https://www.theguardian.com/us-news/2017/oct/11/police-killings-counted-harvard-study.

[44] "Fatal Police Violence by Race and State in the USA, 1980–2019: A Network Meta-Regression," *The Lancet* vol. 398, no. 10307 (Oct. 2, 2021): 1239–1255, https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(21)01609-3/fulltext.

[45] Ibid.

[46] S.A. Luzi, J. Melinek, W.R. Oliver. "Medical Examiner's Independence is Vital for the Health of the American Legal System," *Academic Forensic Pathology – Sage Journals* vol. 3, no. 1, (Mar. 1, 2019): 84-92, https://journals.sagepub.com/doi/abs/10.23907/2013.012.

[47] M. Singh, "How America's broken autopsy system can mask police violence," *The Guardian*, Jul. 3, 2020, http://www.theguardian.com/us-news/2020/jul/02/autopsies-police-killings-medical-misleading.

[48] L. Sathyavagiswaran, C. Rogers, T. Noguchi, "Restraint asphyxia in in-custody deaths – Medical examiner's role in prevention of deaths," *Legal Medicine* vol. 9, (2007): 88-93, https://doi.org/10.1016/j.legalmed.2006.11.007.

[49] William Russell Oliver, "The effect of threat of litigation on forensic pathologist diagnostic decision making," *American Journal of Forensic Medicine and Pathology*vol. 32, no. 4, (Dec. 2011): 383-386, https://journals.lww.com/amjforensicmedicine/Abstract/2011/12000/The_Effect_of_Threat_of_Litigation_on_Forensic.22.aspx.

[50] Ibid.

[51] R. Hanzlick, J. H. Hunsaker III, G. J. Davis, "A Guide For Manner of Death Classification," *National Association of Medical Examiners*, 2002, https://name.memberclicks.net/assets/docs/MANNEROFDEATH.pdf.

[52] Ibid.

[53] Ibid.

[54] R. A. Mitchell Jr, F. Diaz, G. A. Goldfogel, et al., "National Association of Medical Examiners Position Paper: Recommendations for the Definition, Investigation, Postmortem Examination, and Reporting of Deaths in Custody," *Academic Forensic Pathology – Sage Journals* vol. 7, no. 4, (Dec. 1, 2017): 604-618, https://journals.sagepub.com/doi/abs/10.23907/2017.051.

[55] H. Arksey and L. O'Malley, "Scoping studies: towards a methodological framework," *International Journal of Social Research Methodology* vol. 8, no. 1, (2005): 19-32, https://doi.org/10.1080/1364557032000119616.

[56] The forensic pathologists PHR interviewed were Michael Baden, Joye Carter, Kris Cunningham, Soledad Martinez, Judy Melinek, Roger Mitchell, Michael Pollanen, and Enrico Risso. There were also contributions from forensic pathologists who preferred not to have quotes ascribed to them. The emergency physicians were Martin Chenevert and Jared Strote. The other physicians were Michael Freeman, doctor of medicine and forensic epidemiologist, and Suzan Marshall, surgeon and certified death investigator. The plaintiff's attorneys were John Burton, Jim Davy, Dale Galipo, and Ben Nisenbaum. The prosecutors preferred not to have quotes ascribed to them. The police trainer was Jack Ryan. One additional attorney, Selwyn Pieters, provided written answers to our questions.

[57] PHR spoke to Tim Black, Crisis Assistance Helping Out On The Streets (CAHOOTS); Robyn Burek, Portland Street Response; Elizabeth Sinclair Hanc, Treatment Advocacy Center; Sabah Muhammad, Treatment Advocacy Center; Kimberly Sue, National Harm Reduction Coalition; and Greg Townley, Portland Street Response.

[58] Merriam-Webster, "syndrome," https://www.merriam-webster.com/dictionary/syndrome.

[59] R. Freedman, D. A. Lewis, R. Michels, D. S. Pine, S. K. Schultz, C. A. Tamminga, et al., "The initial field trials of DSM-5: new blooms and old thorns," *American Journal of Psychiatry* vol. 170, no. 1, (Jan. 1, 2013): 1-5, https://ajp.psychiatryonline.org/doi/10.1176/appi.ajp.2012.12091189?url_ver=Z39.88-2003&rfr_id=ori:rid:crossref.org&rfr_dat=cr_pub%20%200pubmed.

[60] American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, (Washington D.C.: APA Press, 2013).

[61] Luther Bell, "On a form of disease resembling some advanced stage of mania and fever," *The American Journal of Insanity* vol. 6, (Oct. 1849): 97–127, https://ajp.psychiatryonline.org/doi/10.1176/ajp.6.2.97; M. Morrison, "Death of a Psychiatric Patient during Physical Restraint. Excited Delirium- A Case Report," *Medicine, Science and the Law* vol. 41, (Jan. 2001): 46-50, https://doi.org/10.1177/002580240104100109; Amanda Truscott, "A knee in the neck of excited delirium," *Canadian Medical Association Journal* vol. 178, (Mar. 2008): 669-670, https://doi.org/10.1503/cmaj.080210; Roger Byard, "Ongoing issues with the diagnosis of excited delirium," *Forensic Science, Medicine and Pathology* vol. 14, (Aug. 2017): 149-151, https://doi.org/10.1007/s12024-017-9904-3; Deborah C. Mash, "Excited Delirium and Sudden Death: A Syndromal Disorder at the Extreme End of the Neuropsychiatric Continuum." *Frontiers in Physiology* vol. 7, (Oct. 13, 2016): 1-9,

https://www.frontiersin.org/articles/10.3389/fphys.2016.00435/full; Matthew D. Sztajnkrycer et al., "Cocaine, excited delirium and sudden unexpected death," *Emergency Medical Services*, vol. 34, (Apr. 2005): 77-81, https://pubmed.ncbi.nlm.nih.gov/15900873/; M. Fink, "Delirious mania," *Bipolar Disorders* vol. 1, no. 1, (Sept. 1999): 54-60, https://doi-org.offcampus.lib.washington.edu/10.1034/j.1399-5618.1999.10112.x; Sandeep Sekkon et al., "Excited Delirium," *StatPearls*, Jul. 2021, https://pubmed.ncbi.nlm.nih.gov/31536280/; Philippe Gonin et al., "Excited Delirium: A Systematic Review," 2018, https://doi.org/10.1111/acem.13330; Gary Vilke et al., "Excited delirium syndrome (ExDS): redefining an old diagnosis," *Journal of Forensic and Legal Medicine* vol. 19, (Jan. 2012): https://doi.org/10.1016/j.jflm.2011.10.006; Maurice Lipsedge, "Excited delirium: A psychiatric review," *Medicine, Science and the Law* vol. 56, no. 2, (Apr. 2016): https://doi.org/10.1177/0025802415579617; Gary Vilke et al., "Excited Delirium Syndrome: Defining Based on a Review of the Literature," *Journal of Emergency Medicine* vol. 43, no. 5, (Nov. 2012): 897-905, https://doi.org/10.1016/j.jemermed.2011.02.017; Christopher Bond et al., "Hot Off the Press: SGEM #218. Excited Delirium: A Systematic Review," *Academic Emergency Medicine* vol. 26, (Jan. 2019): 106-108, https://doi.org/10.1111/acem.13487; J. R. Grant, P. E. Southall, J. Mealey, S. R. Scott, D. R. Fowler, "Excited Delirium Deaths in Custody: Past and Present," *American Journal of Forensic Medicine and Pathology* vol. 30, no. 1, (2009): 1-5, https://journals.lww.com/amjforensicmedicine/Abstract/2009/03000/Excited_Delirium_Deaths_in_Custody__Past_and.1.aspx; David Ranson, "Excited delirium syndrome: a political diagnosis?" *American Journal of Law and Medicine* vol. 19, (Jun. 2012): 667-672, https://pubmed.ncbi.nlm.nih.gov/22908610/..

[62] M. Morrison, "Death of a Psychiatric Patient," 46-50; Matthew D. Sztajnkrycer et al., "Cocaine, excited delirium and sudden unexpected death," 77-81; M. Fink, "Delirious mania," 54-60; Gary Vilke et al., "Excited delirium syndrome (ExDS): redefining an old diagnosis," 2012; Edith Samuel et al., "Excited delirium: Consideration of selected medical and psychiatric issues," *Neuropsychiatric Disease and Treatment* vol. 5, (Jan. 21, 2009): 61-66, https://doi.org/10.2147/ndt.s2883; J. R. Grant, P. E. Southall, J. Mealey, S. R. Scott, D. R. Fowler, "Excited Delirium Deaths in Custody, 1-5; Di Maio and Di Maio, *Excited Delirium Syndrome: Cause of Death and Prevention* (New York: CRC Press, 2005).

[63] Psychiatric diagnoses, prominently schizophrenia, have also been used to stigmatize Black people. See, e.g., J. Metzl, *The Protest Psychosis: How Schizophrenia became a Black Disease* (Boston: Beacon Press, 2009).

[64] Gary Vilke et al., "Excited delirium syndrome (ExDS): redefining an old diagnosis," 2012; J. R. Grant, P. E. Southall, J. Mealey, S. R. Scott, D. R. Fowler, "Excited Delirium Deaths in Custody, 1-5; David Ranson, "Excited delirium syndrome: a political diagnosis?" 2012, 667-672; Deborah C. Mash, "Excited Delirium and Sudden Death, 2016.

[65] David A. Fishbain and Charles V. Wetli, "Cocaine intoxication, delirium, and death in a body packer," *Annals of Emergency Medicine* vol. 10, (Oct. 1981): 531–532, https://pubmed.ncbi.nlm.nih.gov/7283219/.

[66] Charles V. Wetli and David A. Fishbain, "Cocaine-Induced Psychosis and Sudden Death in Recreational Cocaine Users," *Journal of Forensic Sciences* vol. 30, no. 3, (Jul. 1985): 873-880; Sandeep Sekkon et al., "Excited Delirium," 2021; Roger Byard, "Ongoing issues with the diagnosis of excited delirium," *Forensic Science, Medicine and Pathology* vol. 14, (Aug. 2017): 149-151, https://doi.org/ 10.1007/s12024-017-9904-3; Amanda Truscott, "A knee in the neck of excited delirium," *Canadian Medical Association Journal* vol. 178, (Mar. 2008): 669-670, https://doi.org/10.1503/cmaj.080210.

[67] Z.J. Lipowsky, "Organic Mental Disorders: Introduction and Review of Syndromes," in *Comprehensive Textbook of Psychiatry*, 3rd ed., eds. H.F. Kaplan, A. M. Freedman, and B. M. Sodock (Baltimore, Maryland: Williams and Wilkins, 1980), 1359-1392.

[68] Fishbain and Wetli, "Cocaine Intoxication, Delirium, and Death In a Body Packer," 531-532.

[69] Adrian Walker and Heather Dewar, "Cocaine-Sex Deaths in Dade Probed," *Miami News*, Nov. 24, 1988.

[70] Ibid.
[71] Ibid.
[72] Ibid.
[73] Ibid.
[74] Ibid.
[75] Adrian Walker, "Tainted crack suspected in deaths of 9 women," *Miami News*, Dec. 8, 1988.
[76] Pete Collins and Bob McPhail, "Seventeen Dead So Far," *New Times* vol. 4, no. 2, (May 3-9, 1989): 6-22.
[77] Donna Gehrke, "Missed Calls, Close Calls Mar Serial Killings Case," *Miami Herald*, Apr. 26, 1990; Metropolitan Dade County Medical Examiner Department, "Investigation Report (Antoinette Burns. M.E. Case No. 7703575)," Dec. 12, 1988.
[78] Donna Gehrke, "18 Killings Leave Families Struggling with Uncertainty," *Miami Herald*, Jun. 18, 1989; Barry Bearak, "Eerie Deaths of 17 Women Baffle Miami," *Los Angeles Times*, May 14, 1989, https://www.latimes.com/archives/la-xpm-1989-05-14-mn-413-story.html.
[79] Russ Rymer, "Murder Without a Trace," *In Health*, May/Jun. 1990.
[80] Ibid.
[81] Pete Collins and Bob McPhail, "Seventeen Dead So Far," *New Times*, May 3-9, 1989.
[82] Ibid.
[83] Ibid.
[84] Ibid.
[85] Ibid.
[86] Rosemary Goudreau, "Coroner: Somebody Killed 19 women," *Miami Herald*, May 31, 1989.
[87] Donna Gehrke, "Missed Calls, Close Calls Mar Serial Killings Case," *Miami Herald*, April 26, 1990.
[88] Rosemary Goudreau, "Coroner: Somebody Killed 19 women," *Miami Herald*, May 31, 1989.
[89] Ibid. John Donnelly, "Suspect in Murder of Prostitutes Speaks Out," *Miami Herald*, May 9, 1993; Donna Gehrke, "Missed Calls, Close Calls Mar Serial Killings Case," *Miami Herald*, April 26, 1990.
[90] Donna Gehrke, "Cops: Rapist Top Suspect in Murders," *Miami Herald*, Jun. 20, 1989.
[91] Donna Gehrke, "18 Killings Leave Families Struggling with Uncertainty," *Miami Herald*, June 18, 1989.
[92] Ibid.
[93] Donna Gehrke, "Both Sides Rest in Williams' Rape Trial," *Miami Herald*, Apr. 27, 1990.
[94] Tatiana M. With, "Absence of justice: case closed on suspected killer of 32," *Miami Herald*, Sept. 25, 1994.
[95] Russ Rymer, "Murder Without a Trace," *In Health*, May/Jun. 1990. Two decades later, Wetli still held to his theory that a combination of cocaine and sex might have killed the women. The *Miami New Times* reported, "Wetli, who is in private practice in New Jersey, initially downplays his theory. He had to make a diagnosis so that the bodies could be buried, he says. But then it becomes clear he still believes that death-by-sex might have killed those women 20 years ago. 'It's certainly a possibility,' he says. 'The guy never went to trial, so we'll never know. The police had a commendable theory in suspecting him. But believing in something, and proving it, is another story.'" Gus Garcia-Roberts, "Is excited delirium killing coked-up, stun-gunned Miamians?" *Miami New Times*, Jul. 15, 2010, https://www.miaminewtimes.com/news/is-excited-delirium-killing-coked-up-stun-gunned-miamians-6367399?showFullText=true.
[96] Russ Rymer, "Murder Without a Trace," *In Health*, May/June 1990.
[97] In a 2021 law review article on "excited delirium," Professor Osagie K. Obasogie provides important additional context for Wetli's theories: "it is imperative to highlight the role that race and, in particular, perceptions and anxieties regarding Black drug use and Black criminality play in giving legitimacy to an excited delirium diagnosis. … For example, the 1980s crack cocaine epidemic gave birth to horrific tropes about racial minorities and premature death, such as the so-called 'crack baby' myth suggesting that maternal drug use during pregnancy led to high rates of stillbirth and infants with lifelong health

problems. … What connects this spectrum of belief and practice is the notion of pathologizing Blackness, where premature death is seen as a function of Black people's inherent inferiority and is used to exculpate actions by others that may be the more proximate cause of death." Osagie K. Obasogie, "Excited Delirium and Police Use of Force," 2021.

[98] Stephens et al., "National Association of Medical Examiners Position Paper on the Certification of Cocaine-Related Deaths," *American Journal of Forensic Medicine and Pathology* vol. 25, no. 1, (March 2004), http://www.charlydmiller.com/LIB04/2004namecocainedeaths.pdf.

[99] Charles V. Wetli, "Deaths due to cocaine induced excited delirium and psychosis," *American Journal of Clinical Pathology* vol. 104, no. 329, (1995).

[100] Stephens et al., "National Association of Medical Examiners Position Paper," 2004.

[101] R. A. Mitchell Jr., F. Diaz, G. A. Goldfogel, et al., "National Association of Medical Examiners Position Paper: Recommendations for the Definition, Investigation, Postmortem Examination, and Reporting of Deaths in Custody," *Academic Forensic Pathology* vol. 7, no. 4, (2017): 604-618, https://journals.sagepub.com/doi/abs/10.23907/2017.051.

[102] Di Maio and Di Maio, *Excited Delirium Syndrome,* 2005.

[103] Ibid.

[104] Ibid.

[105] Deposition of Vincent J.M. Di Maio, M.D. at 159:19-21, *Harrison v. County of Alameda*, No. C11-2868 JST (N.D. Cal. Jan. 24, 2014). The transcript states:

> **Q.** But excited delirium syndrome is something you and your wife came up with, right?
> **A.** Right. Yes.

[106] Jennifer Valentino-DeVries, Mike McIntire, Rebecca R. Ruiz, Julie Tate, and Michael H. Keller, "How Paid Experts Help Exonerate Police After Deaths in Custody," *New York Times*, Dec. 26, 2021, https://www.nytimes.com/2021/12/26/us/police-deaths-in-custody-blame.html.

[107] Ibid.

[108] T. C. Chan, G. M. Vilke, T. Neuman, J. L. Clausen, "Restraint position and positional asphyxia," *Annals of Emergency Medicine* vol. 30, (1997): 578-86.

[109] T. C. Chan, T. Neuman, J. Clausen, J. Eisele, G. M. Vilke, "Weight Force During Prone Restraint and Respiratory Function," *American Journal of Forensic Medicine and Pathology* vol. 25, (2004): 185-189.

[110] This was necessary because such reviews are mandatory for all research with human participants to certify compliance with established safety and ethical standards.

[111] Deposition of Gary M. Vilke, M.D. at 55:20-21, *Bonnie Keeney v. City of New London*, No. 3:99-CV-2096 (JCH) (D. Conn. Apr. 5, 2001). [Charles Wetli was the defendants' expert in the Keeney case.]

[112] Deposition of Gary M. Vilke at 13:22-14:5, *Neuroth v. Mendocino County*, No. 3:15-cv-03226-RS (N.D. Cal. Mar. 19, 2018).

[113] A.J. Lagoe, Steve Eckert, and Chris Vanderveen, "KARE 11 Investigates: Dueling experts about the danger of holding George Floyd down," *KARE 11*, Apr. 10, 2021, https://www.kare11.com/article/news/local/george-floyd/dueling-experts-about-the-danger-of-holding-george-floyd-down/89-b4cecf93-0ec5-4b91-9a27-5271d1e77db6.

[114] Jennifer Valentino-DeVries, Mike McIntire, Rebecca R. Ruiz, Julie Tate, and Michael H. Keller, "How Paid Experts Help Exonerate Police After Deaths in Custody," *New York Times*, Dec. 26, 2021.

[115] Deposition of Theodore C. Chan, M.D. at 22:25-23:1-9, *Hesterberg v. USA.*, No. C13-1265 JSC (JCS) (N.D. Cal. Apr. 18, 2014).

[116] B.A. Michalewic, T.C. Chan, G.M. Vilke, S.S. Levy, T.S. Neuman, and F.W. Kolkhorst. "Ventilatory and metabolic demands during aggressive physical restraint in healthy adults," *Journal of Forensic Science* vol. 52(1) (2007): 171-175, https://doi.org/10.1111/j.1556-4029.2006.00296.x.

[117] In the *Neuroth* case, Julia Sherwin subpoenaed the IRB materials only from UCSD and San Diego State. In *Martinez v. City of Pittsburg, et al.*, she also subpoenaed IRB materials from Vilke and his co-authors; no one produced anything. San Diego State

informed Sherwin they may have destroyed the information pursuant to their document retention policy. UCSD's policy is to retain such documents "indefinitely wherever possible." In accordance with FDA regulations, an IRB has the authority to approve, require modifications, or disapprove research. See U.S. Food & Drug Administration, "Institutional Review Boards (IRBs) and Protection of Human Subjects in Clinical Trials," Sept. 11, 2019, https://www.fda.gov/about-fda/center-drug-evaluation-and-research-cder/institutional-review-boards-irbs-and-protection-human-subjects-clinical-trials. Julia Sherwin subpoenaed all IRB materials related to all of Vilke et al. prone restraint studies from UCSD. The University produced IRB information for the earlier studies involving 25 pounds and 50 pounds but not for the study with 225 pounds. UCSD had no IRB documents or approval for the more recent study titled "Ventilatory and Metabolic Demands During Aggressive Physical Restraint in Healthy Adults," nor did UCSD have IRB Committee meeting minutes from when the study was considered. Sherwin then subpoenaed all evidence or documents concerning IRB applications or approvals, even including correspondence and emails, for the 225-pound study from all authors of the study; these subpoenas produced nothing.

[118] Depositions of Gary M. Vilke, M.D., *Neuroth v. Mendocino County, et al.*, 3:15-CV-03226-RS, (N. D. Cal. 2018); *Martinez v. City of Pittsburg, et al.*, 3:17-CV-04246-RS, (N.D. Cal. 2019).

[119] Ibid.

[120] Deposition of Vincent J.M. Di Maio, M.D. at 159:19-21, *Harrison v. County of Alameda*, No. C11-2868 JST (N.D. Cal. Jan. 24, 2014). Di Maio stated in this legal deposition in 2014:

> **Q.** By 2007, TASER International had purchased 1,000 or 1,500 copies of your book to hand out free to medical examiners, right?
> **A.** Yes. That's great.

[121] Gus Garcia-Roberts, "Is excited delirium killing coked-up, stun-gunned Miamians?" *Miami New Times*, Jul. 15, 2010; interview with Michael Baden, Oct. 7, 2021. As of December 1, 2021, a new hardcover copy was available for $123.47 and a digital copy for $229.95. https://www.amazon.com/Excited-Delirium-Syndrome-Cause-Prevention/dp/0849316111.

[122] Bernice Yeung, "Taser's Delirium Defense," *Mother Jones*, Mar./Apr. 2009; Gus Garcia-Roberts, "Is excited delirium killing coked-up, stun-gunned Miamians?" *Miami New Times*, Jul. 15, 2010.

[123] Scientific Working Group on Medicolegal Death Investigation (SWGMDI), "Increasing the Supply of Forensic Pathologists in the United States," December 5, 2012, https://www.nist.gov/system/files/documents/2018/04/24/swgmdi_increasing_the_supply_of_forensic_pathologists_in_the_us.pdf.

[124] In 2017, TASER International changed its name to Axon Enterprise. See Laurel Wamsley, "Taser Changes Its Name To Axon And Offers Free Body Cameras For Police," *NPR*, April 7, 2017, https://www.npr.org/sections/thetwo-way/2017/04/07/522878573/we-re-more-than-stun-guns-says-taser-as-it-changes-company-name.

[125] Deposition of Vincent J.M. Di Maio, M.D. at 159:19-21, *Harrison v. County of Alameda*, No. C11-2868 JST (N.D. Cal. Jan. 24, 2014).

[126] Tim Reid, Peter Eisler, Jason Szep and M. B. Pell, "As Taser warns of more and more risks, cities bear a burden in court," *Reuters*, Aug. 23, 2017, https://www.reuters.com/investigates/special-report/usa-taser-legal/; Omar El Akkad and Jessica Leeder, "How Taser International wins in the courtroom," *Globe and Mail*, Nov. 28, 2007, https://www.theglobeandmail.com/news/national/how-taser-international-wins-in-the-courtroom/article20406364/; Shahien Nasiripour, "Zapping Taser," *Reveal News*, Dec. 1, 2008, https://revealnews.org/article/zapping-taser/; Jennifer Valentino-DeVries, Mike McIntire, Rebecca R. Ruiz, Julie Tate, and Michael H. Keller, "How Paid Experts Help Exonerate Police After Deaths in Custody," *New York Times*, Dec. 26, 2021.

[127] In a 2018 deposition, Peters said, "On the consulting side I got paid from time to time retained by independent counsel on Taser associated death cases. And I probably did 15

or 20 of those over the course of six years. So using a flat rate approach of $7,500, that would be an approximate ballpark. Now whether those checks came directly from TASER or it came from counsel like you that TASER might have retained, that varied." Deposition of John G. Peters, Jr. at 37:17-25, *Neuroth v. Mendocino County et al.*, No. 3:15-cv-03226-RS (N.D. Cal. Feb. 15, 2018).

[128] Institute for the Prevention of In-Custody Death, Inc., "Initial List of Officers, Directors and Resident Agent," Apr. 28, 2005. Courtesy of Julia Sherwin.

[129] Institute for the Prevention of In-Custody Deaths, Inc., "IPICD 3rd Annual Sudden Death, Excited Delirium & In-Custody Death Conference—LAS VEGAS," Aug. 15, 2008, https://www.police1.com/police-products/training-products/press-releases/ipicd-3rd-annual-sudden-death-excited-delirium-in-custody-death-conferencelas-vegas-Ted0BlnrSeSTvzd3.

[130] Declaration of Steven B. Karch, MO, FFELM, *Rosa v. TASER International, Inc.*, No. C-05-03577 JF/HRL (N.D. Cal. Jun. 1, 2009).

[131] *Reuters* reported in 2017, "Taser paid Mash around $24,000 for expert testimony in eight lawsuits filed from 2005 to 2009, court records show." Jason Szep, Tim Reid, and Peter Eisler, "How Taser inserts itself into investigations involving its own weapons," *Reuters*, Aug. 24, 2017, https://www.reuters.com/investigates/special-report/usa-taser-experts/.

[132] American College of Emergency Physicians (ACEP) Excited Delirium Task Force, "White Paper Report on Excited Delirium Syndrome," American College of Emergency Physicians, Sept. 2009, https://www.ojp.gov/ncjrs/virtual-library/abstracts/white-paper-report-excited-delirium-syndrome.

[133] Andy Mannix, "After conflict-of-interest concerns, HCMC ends deal with Taser manufacturer," *Star Tribune*, Jun. 19, 2019, https://www.startribune.com/after-conflict-of-interest-concerns-hcmc-to-sever-agreement-with-taser-manufacturer/511528142/; Curtis Gilbert, Angela Caputo, and Geoff Hing, "When Tasers Fail: Tasers are less reliable than their maker has claimed. The results can be deadly," *APM Reports*, May 9, 2019, https://www.apmreports.org/episode/2019/05/09/when-tasers-fail.

[134] Ibid.

[135] Arjun S. Byju, "Excited Delirium: How Cops Invented a Disease," *Current Affairs*, Apr. 13, 2021, https://www.currentaffairs.org/2021/04/excited-delirium-how-cops-invented-a-disease.

[136] Russ Rymer, "Murder Without a Trace," *In Health*, May/June 1990.

[137] Gary Vilke et al, "Excited delirium syndrome: defining based on a review of the literature," 2012, 43-45.

[138] Ibid.

[139] John H. Laub, "Study of Deaths Following Electro Muscular Disruption," National Institute of Justice, May 2011, https://www.ojp.gov/pdffiles1/nij/233432.pdf. Other NIJ-funded but not endorsed reports on "excited delirium" include: "Special Panel Review of Excited Delirium," Less-Lethal Devices Technology Working Group, Dec. 2011, https://nij.ojp.gov/library/publications/special-panel-review-excited-delirium; Cynthia Bir, "Physiological Model of Excited Delirium," National Institute of Justice, Dec. 2011, https://nij.ojp.gov/library/publications/physiological-model-excited-delirium.

[140] Opposition to County Motion for Summary Judgment at 1:2-3:9, *Harrison v. County of Alameda*, No. 3:11-cv-2868-JST (N.D. Cal. Dec. 20, 2013), ECF No. 147; Opposition to Sancho Motion for Summary Judgment at 2:2-5:26, *Harrison v. County of Alameda*, No. 3:11-cv-2868-JST (N.D. Cal. Dec. 20, 2013), ECF No. 146.

[141] Ibid.

[142] Ibid.

[143] Ibid.

[144] Ibid.

[145] Deposition of Charles Wetli, M.D. at 106:17-19, 60:23-25, *Harrison v. County of Alameda*, No. C11-2868 JST (N.D. Cal. Jan. 15, 2014).

[146] Di Maio Deposition at 33:6-7. 32:23-24, *Harrison v. County of Alameda*, No. 3:11-cv-2868-JST (N.D. Cal. Jan. 24, 2014).

[147] Di Maio Deposition at 163:8-13, *Harrison v. County of Alameda*, No. 3:11-cv-2868-JST (N.D. Cal. Jan. 24, 2014).

[148] Wetli Deposition at 62:4-10, 63:23-64:1, *Harrison v. County of Alameda*, No. 3:11-cv-2868-JST (N.D. Cal. Jan. 15, 2014).
[149]Di Maio Deposition at 159:19-160:14, 162:5-17; Wetli Deposition at 62:7-10.
[150] Stipulation and Order of Settlement, *Harrison v. County of Alameda, et al.*, No. 3:11-cv-2868 JST (N.D. Cal. Feb. 27, 2015), ECF No. 442.
[151] Sandeep Sekkon et al, "Excited Delirium," 2021; Philippe Gonin et al, "Excited Delirium: A Systematic Review," 2018; Gary Vilke et al., "Excited delirium syndrome (ExDS): redefining an old diagnosis," 2012; Ellen Strommer et al., "The role of restraint in fatal excited delirium: a research synthesis and pooled analysis," *Forensic Science, Medicine and Pathology* vol. 16, no.4, (Aug. 2020), https://doi.org/10.1007/s12024-020-00291-8; Jared Strote et al., "Medical conditions and restraint in patients experiencing excited delirium," *American Journal of Emergency Medicine* vol. 32, (Sept. 2014): 1093-1096, https://doi.org/10.1016/j.ajem.2014.05.023; James Gill, "The syndrome of excited delirium," *Forensic Science, Medicine and Pathology* vol. 10, no. 2, (Feb. 2014), https://doi.org/10.1007/s12024-014-9530-2; Sarathchandra Kodikara et al., " "Excited delirium syndrome": is it a cause of death," *Legal Medicine* vol. 14, (Sept. 2012): 252-254, https://doi.org/10.1016/j.legalmed.2012.04.003; Christine Alison Hall et al., "Frequency of signs of excited delirium syndrome in subjects undergoing police use of force: Descriptive evaluation of a prospective, consecutive cohort, " *Journal of Forensic and Legal Medicine* vol. 20, (Feb. 2013): 102-107, https://doi.org/10.1016/j.jflm.2012.05.008; William Bozeman et al., "Long QT syndrome unmasked in an adult subject presenting with excited delirium," *Journal of Emergency Medicine* vol. 44, (Feb. 2013): 207-210, https://doi.org/10.1016/j.jemermed.2012.02.054; S. N. Kunz et al., "Arrest-related death on the basis of a drug-induced excited delirium syndrome," *Journal of Forensic and Legal Medicine* vol. 77, (Jan. 2021): https://doi.org/10.1016/j.jflm.2020.102091.
[152] Christopher Bond et al., "Hot Off the Press: SGEM #218. Excited Delirium: A Systematic Review," *Academic Emergency Medicine* vol. 26, (Jan. 2019): 106-108, https://doi.org/10.1111/acem.13487; Solveig Baltzer Nielsen et al., "Can acute stress be fatal? A systematic cross-disciplinary review," *Stress* vol. 22, (May 2019): 286-294, https://doi-org.offcampus.lib.washington.edu/10.1080/10253890.2018.1561847; William Bozeman et al., "Long QT syndrome unmasked in an adult subject presenting with excited delirium," *Journal of Emergency Medicine* vol. 44, (Feb. 2013): 207-210, https://doi.org/10.1016/j.jemermed.2012.02.054; Ashwyn Rajagopalan et al., "Sudden death during struggle in the setting of heterozygosity for a mutation in calsequesterin 2," *Forensic Science, Medicine and Pathology* vol. 12, (2016): 86-89, https://doi.org/10.1007/s12024-015-9733-1; Philippe Gonin et al., "Excited Delirium: A Systematic Review," 2018; Ellen Strommer et al, "The role of restraint in fatal excited delirium," 2020; A. J. Ruttenber et al., "Cocaine-associated rhabdomyolysis and excited delirium: different stages of the same syndrome," *American Journal of Forensic Medicine and Pathology* vol. 20, (Jun. 1999): 120-127, https://doi.org/10.1097/00000433-199906000-00003.
[153] Christopher Bond et al, "Hot Off the Press: SGEM #218. Excited Delirium: A Systematic Review," 2019; Philippe Gonin et al, "Excited Delirium: A Systematic Review," 2018.
[154] Philippe Gonin et al, "Excited Delirium: A Systematic Review," 2018.
[155] Charles V. Wetli and David A. Fishbain, "Cocaine-Induced Psychosis and Sudden Death," 1985.
[156] Z.J. Lipowsky, "Organic Mental Disorders: Introduction and Review of Syndromes," in *Comprehensive Textbook of Psychiatry*, 3rd ed., 1980.
[157] C. V. Wetli et al., "Cocaine-associated agitated delirium and the neuroleptic malignant syndrome," *American Journal of Emergency Medicine* vol. 14, (Jul. 1996): 425-428, https://doi.org/doi:10.1016/S0735-6757(96)90066-2. The literature has often referred to "excited delirium" and agitated delirium as the same. Ellen Strommer et al., "The role of restraint in fatal excited delirium," 2020.
[158] J. K. Staley et al., "High affinity cocaine recognition sites on the dopamine transporter are elevated in fatal cocaine overdose victims," *Journal of Pharmacology and Experimental Therapeutics* vol. 271, (Dec. 1994): 1678-1685, https://pubmed.ncbi.nlm.nih.gov/7996484/; Deborah C. Mash et al., "D3 dopamine and

kappa opioid receptor alterations in human brain of cocaine-overdose victims," *Annals of the New York Academy of Sciences*, 877. (Jun. 1999): 507-522. doi:10.1111/j.1749-6632.1999.tb09286.x; J. K. Staley et al., "Adaptive increase in D3 dopamine receptors in the brain reward circuits of human cocaine fatalities," *Journal of Neuroscience* vol. 16, (Oct. 1996): 6100-6106, https://www.jneurosci.org/content/16/19/6100; A. J. Ruttenber, "Fatal excited delirium following cocaine use: epidemiologic findings provide new evidence for mechanisms of cocaine toxicity," *Journal of Forensic Science* vol. 42, (Jan. 1997): 25-31; B. G. Stephens et al., "Criteria for the interpretation of cocaine levels in human biological samples and their relation to the cause of death," *American Journal of Forensic Medicine and Pathology* vol. 25, (Mar. 2004): 1-10, https://doi.org/10.1097/01.paf.0000118960.58334.a9; A. J. Ruttenber et al., "Cocaine-associated rhabdomyolysis and excited delirium," 1999; Haresh G. Mirchandani et al., "Cocaine-induced agitated delirium, forceful struggle, and minor head injury. A further definition of sudden death during restraint," *American Journal of Forensic Medicine and Pathology* vol. 15, (1994): 95-99, https://doi.org/10.1097/00000433-199406000-00002.

[159] Kevin B. Gerold et al., "Review, clinical update, and practice guidelines for excited delirium syndrome," *Journal of Special Operations Medicine* vol. 15, (2015): 62-69, https://pubmed.ncbi.nlm.nih.gov/25770800/; Deborah Mash, "Brain biomarkers for identifying excited delirium as a cause of sudden death," *Forensic Science International* vol. 190, (Sept. 2009): e13-e19, https://doi.org/10.1016/j.forsciint.2009.05.012; A. J. Ruttenber, "Fatal excited delirium following cocaine use," 1997; Gary Vilke et al., "Excited delirium syndrome: defining based on a review of the literature," 2012, 897-905; Deborah C. Mash, "Excited Delirium and Sudden Death," 2016; Deborah Mash et al., "Dopamine transport function is elevated in cocaine users" *Journal of Neurochemistry* vol. 81, (2002): 292-300.

[160] Deborah Mash et al., "Brain biomarkers for identifying excited delirium as a cause of sudden death," 2009; Magen M. Johnson et al., "Increased heat shock protein 70 gene expression in the brains of cocaine-related fatalities may be reflective of postdrug survival and intervention rather than excited delirium," *Journal of Forensic Sciences* vol. 57, (Nov. 2012): 1519-1523, https://doi.org/10.1111/j.1556-4029.2012.02212.x.

[161] D. L. Ross, "Factors associated with excited delirium deaths in police custody," *Modern Pathology* vol. 11, (Nov. 1998): 1127-1137, https://pubmed.ncbi.nlm.nih.gov/9831212/; Solveig Baltzer Nielsen et al., "Can acute stress be fatal?," 2019; A. J. Ruttenber et al., "Cocaine-associated rhabdomyolysis and excited delirium," 1999.

[162] M. D. Bell et al., "Positional asphyxiation in adults. A series of 30 cases from the Dade and Broward County Florida Medical Examiner Offices from 1982 to 1990," *American Journal of Forensic Medicine and Pathology* vol. 13, (Jun. 1992): 101-107, https://pubmed.ncbi.nlm.nih.gov/1510056/.

[163] Ellen Strommer et al., "The role of restraint in fatal excited delirium," 2020.

[164] Ibid.

[165] Charles V. Wetli and David A. Fishbain. 1985. "Cocaine-Induced Psychosis and Sudden Death," 1985; D. T. Reay et al., "Positional asphyxia during law enforcement transport," *American Journal of Forensic Medicine and Pathology* vol. 13, (Jun. 1992): 90-97, https://doi.org/10.1097/00000433-199206000-00002; Alain Michaud, "Restraint related deaths and excited delirium syndrome in Ontario (2004-2011)," *Journal of Forensic and Legal Medicine* vol. 41, (Jul. 2016): 30-35, https://doi.org/10.1016/j.jflm.2016.04.010; M. S. Pollanen et al., "Unexpected death related to restraint for excited delirium: a retrospective study of deaths in police custody and in the community," *Canadian Medical Association Journal* vol. 158, (Jun. 1998): 1603-1607, https://pubmed.ncbi.nlm.nih.gov/9645173/; Patrick Joseph Maher, "Prehospital resuscitation of a man with excited delirium and cardiopulmonary arrest," *Canadian Journal of Emergency Medicine* vol. 16, (Jan. 2014): 80-83, https://doi.org/10.2310/8000.2013.130824; Jami R. Grant et al., "Excited delirium deaths in custody: past and present," *American Journal of Forensic Medicine and Pathology* vol. 30, (Mar. 2009): 1-5, https://doi.org/10.1097/PAF.0b013e31818738a0; S. J. Stratton, C. Rogers, K. Green, "Sudden death in individuals in hobble restraints during paramedic transportation," *Annals of Emergency Medicine* vol. 25, (1995): 710-712.

[166] Donald T. Reay et al., "Effects of positional restraint on oxygen saturation and heart rate following exercise," *American Journal of Forensic Medicine and Pathology* vol. 9, (Mar. 1988): 16-18, https://doi.org/10.1097/00000433-198803000-00005.

[167] Christian Sloane et al., "Evaluation of the ventilatory effects of the prone maximum restraint (PMR) position on obese human subjects," *Forensic Science International* vol. 237, (Apr. 2014): 86-89, https://doi.org/10.1016/j.forsciint.2014.01.017.

[168] Gary M. Vilke et al., "Spirometry in normal subjects in sitting, prone, and supine positions," *Respiratory Care* vol. 45*, (*2000): 407–410; T. C. Chan et al., "Restraint position and positional asphyxia," *Annals of Emergency Medicine* vol. 30, (1997): 578–586, https://doi.org/ 10.1016/s0196-0644(97)70072-6; Richard Barnett et al., "The physiological impact of upper limb position in prone restraint," *Medicine, Science and the Law* vol. 53, (2013): 161–165, https://doi.org/10.1258/msl.2012.012044; T. C. Chan et al., "Weight force during prone restraint and respiratory function," *American Journal of Forensic Medicine and Pathology* vol. 25, (Sept. 2004): 185–189, https://doi.org/ 10.1097/01.paf.0000136639.69128.bc; Betty A. Michalewicz et al., "Ventilatory and metabolic demands during aggressive physical restraint in healthy adults," *Journal of Forensic Science* vol. 52, (Jan. 2007): 171–175, https://doi.org/10.1111/j.1556-4029.2006.00296.x; Gary M. Vilke et al., "Spirometry in normal subjects in sitting, prone, and supine positions," 407–410.

[169] John Parkes et. al., "Sudden death during restraint: do some positions affect lung function?" *Medicine, Science and the Law* vol. 48*, (*2008): 137–141; M. Roeggla et al., "Cardiorespiratory consequences to hobble restraint," *Wien Klin Wochenschr* vol. 109, (May 1997): 359–361.

[170] Astrid Krauskopf et al., "Does weight force application to the lower torso have an influence on inferior vena cava and cardiovascular parameters?" *American Journal of Emergency Medicine* vol. 26, (Jun. 2008): 603-607, https://doi.org/10.1016/j.ajem.2007.08.017; Jeffrey D. Ho et al., "Effect of position and weight force on inferior vena cava diameter--implications for arrest-related death," *Forensic Science International* vol. 212, (Oct. 2011): 256-259, https://doi.org/ 10.1016/j.forsciint.2011.07.001.

[171] Carolyn Meredith et al., "The cardiopulmonary effects of physical restraint in subjects with chronic obstructive pulmonary disease," *Journal of Clinical Forensic Medicine* vol. 12, (Dec. 2005): 133–136, https://doi.org/ 10.1016/j.jcfm.2004.10.013.

[172] M. Campbell, R. Dakin, S. Stowe, et al., "Thoracic weighting of restrained subjects during exhaustion recovery causes loss of lung reserve volume in a model of police arrest," *Scientific Reports* vol. 11, no. 15166 (2021), https://doi.org/10.1038/s41598-021-94157-w.

[173] Ibid.

[174] Ibid.

[175] J. M. Berkman, J. A. Rosenthal, A. Saadi, "Carotid Physiology and Neck Restraints in Law Enforcement: Why Neurologists Need to Make Their Voices Heard," JAMA Neurology vol. 78, no. 3, (2021): 267–268. https://jamanetwork.com/journals/jamaneurology/article-abstract/2774482, citing S. Yamasaki, I. Takase, N. Takada, K. Nishi, "Measurement of force to obstruct the cervical arteries and distribution of tension exerted on a ligature in hanging," *Legal Medicine* vol. 11, no. 4, (Jul. 2009): 175, https://www.sciencedirect.com/science/article/abs/pii/S134462230900217X?via%3Dihub.

[176] American Academy of Neurology, "AAN Position Statement on the Use of Neck Restraints in Law Enforcement," Jun. 9, 2021, https://www.aan.com/policy-and-guidelines/policy/position-statements/aan-position-statement-on-use-of-neck-restraints-in-law-enforcement/.

[177] Maurice Lipsedge, "Excited delirium: A psychiatric review," *Medicine, Science and the Law* vol. 56, no. 2, (Apr. 2016), https://doi.org/10.1177/0025802415579617; James Gill, "The syndrome of excited delirium," *Forensic Science, Medicine and Pathology* vol. 10, no. 2, (Feb. 2014), https://doi.org/10.1007/s12024-014-9530-2; American Psychiatric Association *Diagnostic and Statistical Manual of Mental Disorders, 5th ed.* (Washington D.C.: APA Press, 2013).

[178] Ellen Strommer et al., "The role of restraint in fatal excited delirium," 2020.
[179] Di Maio and Di Maio, *Excited Delirium Syndrome*, 2005.
[180] Ellen Strommer et al., "The role of restraint in fatal excited delirium," 2020.
[181] Jennifer Valentino-DeVries, Mike McIntire, Rebecca R. Ruiz, Julie Tate, and Michael H. Keller, "How Paid Experts Help Exonerate Police After Deaths in Custody," *New York Times*, Dec. 26, 2021.
[182] Eric Dexheimer and Jeremy Schwartz, "In fatal struggles with police, a controversial killer is often blamed," *Austin-American Statesman*, May 27, 2017, https://www.statesman.com/story/news/investigates/2017/05/27/in-fatal-struggles-with-police-controversial-killer-is-often-blamed/8339157007.
[183] Alessandro Marazzi Sassoon, "Excited delirium: Rare and deadly syndrome or a condition to excuse deaths by police?" *Florida Today*, Oct. 24, 2019, https://www.floridatoday.com/in-depth/news/2019/10/24/excited-delirium-custody-deaths-gregory-edwards-melbourne-taser/2374304001.
[184] Osagie K. Obasogie, "Excited Delirium and Police Use of Force," 2021.
[185] Ibid.
[186] Philippe Gonin et al., "Excited Delirium: A Systematic Review," 2018.
[187] Alisa Chang, "Police Repeatedly Cite 'Excited Delirium' In Killings, But It Has No Real Definition," *NPR*, Apr. 7, 2021, https://www.npr.org/2021/04/07/985128452/police-repeatedly-cite-excited-delirium-in-killings-but-it-has-no-real-definition.
[188] Katie Wedell, Cara Kelly, Camille McManus, and Christine Fernando, "George Floyd is not alone. 'I can't breathe' uttered by dozens in fatal police holds across U.S.," *USA Today*, Jun. 13, 2020, https://www.usatoday.com/in-depth/news/investigations/2020/06/13/george-floyd-not-alone-dozens-said-cant-breathe-police-holds/3137373001.
[189] See, e.g., *Callwood vs. Jones et. al.*, 727 F. App'x at 556; *Peppers v. Washington County, Tenn.*, 686 Fed. App'x. 328 (6th Cir. 2017); *V. W. v. Nichelini,* 2017 WL 34246 (E.D. CA Feb. 2, 2017); *Pirolozzi v. Stanbro*, F. Supp. 2d, 2009 U.S. Dist. LEXIS 42575, 2009 WL 1441070 (N.D. Ohio Mar. 13, 2009). *See* also *Marquez v. City of Phoenix*, 693 F.3d 1167 (9th Cir. 2012); *Gregory v. County of Maui*, 414 F. Supp. 2d 965 (D. Haw. 2006).
[190] American Medical Association, "New AMA Policy Opposes ' Excited Delirium ' Diagnosis," Jun. 14, 2021,https://www.ama-assn.org/press-center/press-releases/new-ama-policy-opposes-excited-delirium-diagnosis; American Psychiatric Association, "Position Statement on Concerns About Use of the Term 'Excited Delirium' and Appropriate Medical Management in Out-of-Hospital Contexts," December 2020, https://www.psychiatry.org/File%20Library/About-APA/Organization-Documents-Policies/Policies/Position-Use-of-Term-Excited-Delirium.pdf.
[191] American College of Emergency Physicians (ACEP) Hyperactive Delirium Task Force, "ACEP Task Force Report on Hyperactive Delirium with Severe Agitation in Emergency Settings," Jun. 23, 2021, https://www.acep.org/globalassets/new-pdfs/education/acep-task-force-report-on-hyperactive-delirium-final.pdf.
[192] Ibid.
[193] PHR, email to ACEP, Feb. 7, 2022.
[194] ACEP Associate Executive Director Sandy Schneider, email to PHR, Feb. 14, 2022.
[195] PHR, email to NAME, Feb. 7, 2022.
[196] Stephens et al., "National Association of Medical Examiners Position Paper," 2004; R. A. Mitchell Jr., F. Diaz, G. A. Goldfogel, et al., "National Association of Medical Examiners Position Paper," 604-618.
[197] 2022 NAME President Kathryn Pinneri, email to PHR, Feb. 10, 2022.
[198] Royce Kurmelovs, "'Excited delirium': how a disputed US term found its way to Australian deaths-in-custody inquests," *The Guardian*, Jul. 3, 2021, https://www.theguardian.com/australia-news/2021/jul/04/excited-delirium-how-a-disputed-us-term-found-its-way-to-australian-deaths-in-custody-inquests.
[199] John Kiedrowski, Michael Petrunik, and Ronald-Frans Melchers, "An Independent Review of the Adoption and Use of Conducted Energy Weapons by the Royal Canadian Mounted Police," Jun. 5, 2008, https://web.archive.org/web/20091231091631/http://www.rcmp-grc.gc.ca/ccaps-spcca/cew-ai/kiedrowski-report-rapport-eng.htm; Gus Garcia-Roberts, "Is excited

delirium killing coked-up, stun-gunned Miamians?" *Miami New Times*, Jul. 15, 2010, https://www.miaminewtimes.com/news/is-excited-delirium-killing-coked-up-stun-gunned-miamians-6367399?showFullText=true; "Report of the Panel of Mental Health and Medical Experts Review of Excited Delirium", Jun. 30, 2009, https://novascotia.ca/just/public_safety/_docs/Excited%20Delirium%20Report.pdf.

[200] The Royal College of Pathologists, "The Use of 'Excited Delirium' as a Cause of Death," *Forensic Science Regulator Guidance* vol. 231, no. 2: 3.

[201] Royce Kurmelovs, "'Excited Delirium': How a Disputed US Term Found Its Way to Australian Deaths-in-Custody Inquests," *The Guardian*, Jul. 3, 2021, https://www.theguardian.com/australia-news/2021/jul/04/excited-delirium-how-a-disputed-us-term-found-its-way-to-australian-deaths-in-custody-inquests.

[202] Ibid.

[203] Commission for Public Complaints Against the Royal Canadian Mounted Police, "RCMP Use of the Conducted Energy Weapon (CEW): Interim Report," Dec. 11, 2007, https://www.crcc-ccetp.gc.ca/pdf/InterimTaserReport.pdf.

[204] John Kiedrowski, Michael Petrunik, and Ronald-Frans Melchers, "An Independent Review of the Adoption and Use of Conducted Energy Weapons by the Royal Canadian Mounted Police," Jun. 5, 2008, https://web.archive.org/web/20091231091631/http://www.rcmp-grc.gc.ca/ccaps-spcca/cew-ai/kiedrowski-report-rapport-eng.htm.

[205] Royal College of Emergency Medicine Independent Advisory Panel on Deaths in Custody, "Guidelines for the Management of Excited Delirium / Acute Behavioural Disturbane (ABD)," May 2016, https://rcem.ac.uk/wp-content/uploads/2021/10/RCEM_Guidelines_for_Management_of_Acute_Behavioural_Disturbance_May2016.pdf.

[206] Nazia Parveen, "'Excited delirium': term linked to police restraint in UK medical guide condemned," *The Guardian*, Jul. 1, 2021, https://www.theguardian.com/uk-news/2021/jul/01/excited-delirium-term-linked-to-police-restraint-in-uk-medical-guide-condemned; South London and Maudsley NHS Foundation Trust, "South London and Maudsley NHS Foundation Trust categorically do not recognise 'Acute Behavioural Disturbance' and 'Excited Delirium' as syndromes or diagnoses," Jun. 23, 2021, https://www.slam.nhs.uk/media/news/south-london-and-maudsley-nhs-foundation-trust-categorically-do-not-recognise-acute-behavioural-disturbance-and-excited-delirium-as-syndromes-or-diagnoses/.

[207] Royce Kurmelovs, "'Excited Delirium': How a Disputed US Term Found Its Way to Australian Deaths-in-Custody Inquests," *The Guardian*, Jul. 3, 2021, https://www.theguardian.com/australia-news/2021/jul/04/excited-delirium-how-a-disputed-us-term-found-its-way-to-australian-deaths-in-custody-inquests.

[208] The Royal College of Pathologists, "The Use of 'Excited Delirium' as a Cause of Death," *Forensic Science Regulator Guidance* vol. 231, no. 2:3.

[209] Claire Lampen, "What We Know About the Killing of Elijah McClain," *The Cut*, Sept. 1, 2021, https://www.thecut.com/2020/07/the-killing-of-elijah-mcclain-everything-we-know.html; Francie Swidler, "Elijah McClain's death and what happened next: a timeline," *CPR News*, Nov. 19, 2021, https://www.cpr.org/2021/11/19/elijah-mcclain-timeline; Ryan W. Miller, "Elijah McClain was injected with ketamine before he died. Is that legal?" *USA Today*, Jul. 1, 2020, https://www.usatoday.com/story/news/nation/2020/06/26/elijah-mcclain-ketamine-may-have-played-role-death-experts-say/3262785001.

[210] Allison Sherry, "The Adams County Coroner Met With Aurora Police Before Elijah McClain's Cause Of Death Was Determined," *CPR News*, Aug. 19, 2020, https://www.cpr.org/2020/08/19/elijah-mcclain-aurora-police-coroner-autopsy-report.

[211] Diane Carman, "Carman: A mother's grief turns to long-overdue action in justice for Elijah McClain," *Colorado Sun*, Sept. 5, 2021, https://coloradosun.com/2021/09/05/elijah-mcclain-aurora-police-opinion.

[212]Michael de Yoanna and Rae Solomon, "Medics In Colorado Dosed 902 People With Ketamine For 'Excited Delirium' In 2.5 Years," *KUNC*, Jul. 21, 2020, https://www.kunc.org/news/2020-07-21/medics-in-colorado-dosed-902-people-with-ketamine-for-excited-delirium-in-2-5-years#stream/0.

[213] Josiah Hesse, "'Weaponization of medicine': police use of ketamine draws scrutiny after Elijah McClain's death," *The Guardian*, Dec. 17, 2021, https://www.theguardian.com/us-news/2021/dec/17/ketamine-law-enforcement-deaths-custody-elijah-mcclain; Krithika Varagur, "Minnesota Paramedic Speaks Out Against Police Use of Ketamine Injections," *The Intercept*, Aug. 25, 2020, https://theintercept.com/2020/08/25/ketamine-police-use-minnesota/.

[214] American Society of Anesthesiologists, "ASA Statement on the Use of Ketamine for a Non-medical Purpose," Jul. 15, 2020, https://www.asahq.org/about-asa/newsroom/news-releases/2020/07/asa-statement-on-the-use-of-ketamine-for-a-non-medical-purpose.

[215] Associated Press, "Officers and Paramedics are Charged in Elijah McClain's 2019 Death in Colorado," *NPR*, Sept. 1, 2021, https://www.npr.org/2021/09/01/1033289263/elijah-mcclain-death-officers-paramedics-charged.

[216] Jeremy Harlan, Lucy Kafanov, and Leslie Perrot, "Aurora, Colorado, to pay $15 million to Elijah McClain's family to settle lawsuit over his 2019 death at the hands of police," *CNN*, Nov. 19, 2021, https://www.cnn.com/2021/11/19/us/elijah-mcclain-and-aurora-police-settlement/index.html.

[217] Colorado Department of Public Health & Environment, "Final Report: Ketamine Investigatory Review Panel," Dec. 1, 2021, https://cdphe.colorado.gov/press-release/ketamine-review-committee-publishes-report-with-several-recommendations.

[218] In February 2022, Physicians for Human Rights interviewed Joe Prude about the death of his brother Daniel in police custody. The narrative in this section is based on that interview as well as media reports and other public records.

[219] Interview with Joe Prude, Feb. 12, 2022.

[220] Brett Dahlberg, "Rochester Hospital Released Daniel Prude Hours Before Fatal Encounter With Police," *NPR*, Sept. 29, 2020, https://www.npr.org/sections/health-shots/2020/09/29/917317141/rochester-hospital-released-daniel-prude-hours-before-fatal-encounter-with-polic; Michael Wilson, "Did This Police Maneuver Lead to Daniel Prude's Death?" *New York Times*, Sept. 15, 2020, https://www.nytimes.com/2020/09/15/nyregion/daniel-prude-video-police-rochester.html; Steve Orr, "How Daniel Prude suffocated as Rochester police restrained him," *Democrat and Chronicle*, Sept. 2, 2020, https://www.democratandchronicle.com/story/news/2020/09/02/daniel-prude-rochester-ny-police-died-march-2020-after-officers-restrained-him/5682948002.

[221] Interview with Joe Prude, Feb. 12, 2022.

[222] Ibid.

[223] Michael Wilson, "Did This Police Maneuver Lead to Daniel Prude's Death?" *New York Times*, Sept. 15, 2020.

[224] Justin Murphy, "Doctor told grand jury that police restraint was 'protective' of Daniel Prude, who later died," *Democrat and Chronicle*, Apr. 20, 2021, https://www.democratandchronicle.com/story/news/2021/04/20/daniel-prude-letitia-james-gary-vilke-grand-jury-restraint/7276344002/; Michael Wilson, "Did This Police Maneuver Lead to Daniel Prude's Death?" *New York Times*, Sept. 15, 2020.

[225] Kim Bellware, "Daniel Prude's death highlights dangers of 'spit hoods' and calls for regulation," *Washington Post*, Sept. 5, 2020, https://www.washingtonpost.com/nation/2020/09/05/daniel-prude-spit-hood.

[226] New York State Office of the Attorney General, Special Investigations and Prosecutions Unit, "Report on the Investigation into the Death of Daniel Prude," Feb. 23, 2021, https://ag.ny.gov/sites/default/files/oag_report_-_prude.pdf.

[227] Ibid.

[228] Interview with Joe Prude, Feb. 12, 2022.

[229] New York State Office of the Attorney General, Special Investigations and Prosecutions Unit, "Report on the Investigation into the Death of Daniel Prude," Feb. 23, 2021, https://ag.ny.gov/sites/default/files/oag_report_-_prude.pdf.; Troy Closson and Ed Shanahan, "Black Man Died of Suffocation After Officers Put Hood on Him," *New York Times*, Sept. 2, 2020, https://www.nytimes.com/2020/09/02/nyregion/daniel-prude-rochester-police.html.

[230] Interview with Elliot Shields, Feb. 12, 2022; Steve Orr and Justin Murphy, "Emails show Rochester officials' attempts to conceal information about Daniel Prude's death," *USA Today*, Sept. 15, 2020, https://www.usatoday.com/story/news/nation/2020/09/15/daniel-prude-death-rochester-police-sought-delay-video-release-mayor-lovely-warren/5803252002; Evan Simko-Bednarski and Lauren del Valle, "Rochester officials intentionally delayed the release of Daniel Prude body cam video," Sept. 16, 2020, https://www.cnn.com/2020/09/16/us/daniel-prude-rochester-emails/index.html; Michael Wilson and Edgar Sandoval, "Documents Reveal How the Police Kept Daniel Prude's Death Quiet," *New York Times*, Sept. 15, 2020, https://www.nytimes.com/2020/09/15/nyregion/rochester-police-daniel-prude.html.

[231] Ibid.

[232] "NY attorney general empanels grand jury in Daniel Prude case," *Associated Press*, Sept. 5, 2020, https://www.wkbw.com/news/national/ny-attorney-general-empanels-grand-jury-in-daniel-prude-case; New York State Office of the Attorney General, "AG James Update on Prude Investigation: Moves to Empanel Grand Jury," Press Release, Sept. 5, 2020, https://ag.ny.gov/press-release/2020/ag-james-update-prude-investigation-moves-empanel-grand-jury.

[233] Justin Murphy and Gary Craig, "Did AG's choice 'guarantee the outcome'? Expert in Prude grand jury draws scrutiny," *Democrat and Chronicle*, Feb. 26, 2021, https://www.democratandchronicle.com/story/news/2021/02/26/excited-delirium-gary-vilke-letitia-james-daniel-prude-grand-jury-rochester-police-not-indicted/6813354002.

[234] Karen Pallarito, "Police Used a 'Spit Hood' While Arresting a Man Who Later Died of Asphyxiation—Here's What That Is," *Health*, Sept. 3, 2020, https://www.health.com/mind-body/health-diversity-inclusion/what-is-spit-hood-daniel-prude; Brandon Lowrey, "Amid Deadly Arrests, 'Delirium' Diagnosis Draws New Scrutiny," *Law360*, Oct. 18, 2020, https://www.law360.com/articles/1320187/amid-deadly-arrests-delirium-diagnosis-draws-new-scrutiny.

[235] State of New York, "Day 5: Investigation into the death of Daniel T. Prude," *Transcript of the Proceedings held before the Monroe County Grand Jury*, Dec. 16, 2020, https://ag.ny.gov/sites/default/files/session_five_-_december_16_2020.pdf.

[236] Justin Murphy and Gary Craig, "Did AG's choice 'guarantee the outcome'? Expert in Prude grand jury draws scrutiny," *Democrat and Chronicle*, Feb. 26, 2021.

[237] Interview with Joe Prude, Feb. 12, 2022.

[238] New York State Office of the Attorney General, Special Investigations and Prosecutions Unit, "Report on the Investigation into the Death of Daniel Prude," Feb. 23, 2021, https://ag.ny.gov/sites/default/files/oag_report_-_prude.pdf.

[239] Ibid.

[240] Justin Murphy and Gary Craig, "Did AG's choice 'guarantee the outcome'? Expert in Prude grand jury draws scrutiny," *Democrat and Chronicle*, Feb. 26, 2021.

[241] New York State Office of the Attorney General, Special Investigations and Prosecutions Unit, "Report on the Investigation into the Death of Troy Hodge, Mar. 19, 2021, https://ag.ny.gov/sites/default/files/oag_report_-_hodge.pdf.

[242] Ibid.

[243] Ibid.

[244] Interview with Robert Collins, Bella Quinto-Collins, and Cassandra Quinto-Collins, Oct. 29, 2021.

[245] Ibid.

[246] Ibid. See also, John L. Burris, Ayana C. Curry, Ben Nisenbaum, Dewitt M. Lacy, James A. Cook, Kenneth Chike Odiwe, and Tonia Robinson. Law Offices of John L. Burris, "RE: Request for Endorsed Copy Verifying Administrative Claim Received - Law Offices of John L. Burris," Feb. 18, 2021, https://johnburrislaw.com/Quinto-Claim.pdf; Jacey Fortin, "California Man Died after Police Knelt on Him for 5 Minutes, Family Says," *New York Times*, Feb. 25, 2021, https://www.nytimes.com/2021/02/25/us/angelo-quinto-death-police-kneel.html; Claire Wang, "The Filipino American Family behind Calif.'s New Police Reform Laws Speaks Out," *NBC News*, Oct. 15, 2021,

https://www.nbcnews.com/news/asian-america/filipino-american-family-califs-new-police-reform-laws-speaks-rcna3030.
[247] Ibid.
[248] John Burris Law Offices, "Video of Angelo Quinto Incident," *YouTube*, Feb. 18, 2021, https://www.youtube.com/watch?v=eB_tR-V9X-M.
[249] Interview with Robert Collins, Bella Quinto-Collins, and Cassandra Quinto-Collins, Oct. 29, 2021.
[250] Ibid.
[251] Ibid.
[252] John Burris Law Offices, "Video of Angelo Quinto Incident," Feb. 18, 2021.
[253] Interview with Robert Collins, Bella Quinto-Collins, and Cassandra Quinto-Collins, Oct. 29, 2021.
[254] Ibid.
[255] Ibid.
[256] Ibid.
[257] Nate Gartrell and Rick Hurd, "Death of Angelo Quinto, Navy Vet Who Died after Struggle with Antioch Cops, Blamed on 'Excited Delirium.'"
[258] Interview with Robert Collins, Bella Quinto-Collins, and Cassandra Quinto-Collins, Oct. 29, 2021.
[259] Ibid.
[260] Ibid.
[261] Claire Wang, "The Filipino American family behind Calif.'s new police reform laws speaks out," *NBC News*, October 15, 2020.
[262] Interviews with Pollanen, Sept. 20, 2021; Strote, Sept. 15, 2021; Carter, Sept. 23, 2021, among others.
[263] Interview with Pollanen, Sept. 20, 2021.
[264] Interview with Carter, Sept. 23, 2021.
[265] Ibid.
[266] Ibid.
[267] Ibid.
[268] Interview with Mitchell, Oct. 29, 2021.
[269] Interview with Freeman, Sept. 8, 2021; Risso, Oct. 5, 2021.
[270] Interview with Mitchell, Oct. 29, 2021.
[271] Interview with Baden, Oct. 7, 2021.
[272] Interview with Chenevert, Oct. 25, 2021.
[273] Eric Balaban, "How officials use a dubious medical condition to explain stun gun deaths," *The Guardian*, Sept. 17, 2015, https://www.theguardian.com/commentisfree/2015/sep/17/dubious-medical-condition-stun-gun-deaths; Tim Reid and Paula Seligson, "Taser's defense tactics include lawsuits against coroners and experts," *Reuters*, Aug. 24, 2017, https://www.reuters.com/article/us-usa-taser-strikeback/tasers-defense-tactics-include-lawsuits-against-coroners-and-experts-idUSKCN1B4182.
[274] Interview with Carter, Sept. 23, 2021.
[275] Interview with Melinek, Sept. 16, 2021.
[276] Interview with Freeman, Sept. 8, 2021.
[277] Ibid.
[278] Interview with Cunningham, Sept. 16, 2021.
[279] Interview with Pollanen, Sept. 20, 2021.
[280] Interview with Freeman, Sept. 8, 2021.
[281] Interviews with Strote, Sept. 15, 2021; Cunningham, Sept. 16, 2021; Pollanen, Sept. 20, 2021.
[282] Interview with. Pollanen, Sept. 20, 2021.
[283] Ibid.
[284] Interviews with Melinek, Sept. 16, 2021; Risso, Oct. 5, 2021.
[285] Interview with Chenevert, Oct. 25, 2021.
[286] Interviews with Freeman, Sept. 8, 2021; Cunningham, Sept. 16, 2021; Pollanen, Sept. 20, 2021; Mitchell, Oct. 29, 2021.
[287] Interview with Cunningham, Sept. 16, 2021.

[288] Interview with Pollanen, Sept. 20, 2021.
[289] Interview with Mitchell, Oct. 29, 2021.
[290] Interviews with Mitchell, Oct. 29, 2021; Freeman, Sept. 8, 2021.
[291] Interview with Mitchell, Oct. 29, 2021.
[292] Interviews with Cunningham, Sept. 16, 2021; Martinez, Sept. 20, 2021; Baden, Oct. 7, 2021; Mitchell, Oct. 29, 2021.
[293] Interview with Risso, Oct. 5, 2021.
[294] Interview with Martinez, Sept. 20, 2021.
[295] Interview with Cunningham, Sept. 16, 2021.
[296] Interview with Mitchell, Oct. 29, 2021.
[297] Interview with Carter, Sept. 23, 2021.
[298] Interview with Freeman, Sept. 8, 2021.
[299] Ibid.
[300] Interview with Cunningham, Sept. 16, 2021.
[301] Interview with Carter, Sept. 23, 2021.
[302] Interview with Freeman, Sept. 8, 2021.
[303] Interview with Strote, Sept. 15, 2021.
[304] Interview with Mitchell, Oct. 29, 2021.
[305] Interview with Freeman, Sept. 8, 2021.
[306] Interview with Strote, Sept. 15, 2021.
[307] Interview with Baden, Oct. 7, 2021.
[308] Interview with Strote, Sept. 15, 2021.
[309] Interview with Melinek, Sept. 16, 2021.
[310] Interview with Ryan, Sept. 23, 2021.
[311] Ibid.
[312] Ibid.
[313] Ibid.
[314] Interview with Galipo, Sept. 27, 2021.
[315] Interview with Davy, Sept. 9, 2021.
[316] Joanne Zuhl, "Wyden takes Oregon street response model nationwide," *Street Roots*, May 5, 2021, https://www.streetroots.org/news/2021/05/05/wyden-takes-oregon-street-response-model-nationwide.
[317] Interview with Sue, Oct. 14, 2021.
[318] Interview with Hancq, Oct. 7, 2021.
[319] Interview with Muhammad, Oct. 12, 2021.
[320] Lisa Dailey et al., "Grading the States: An Analysis of U.S. Psychiatric Treatment Laws," Treatment Advocacy Center, Sept. 2020, https://www.treatmentadvocacycenter.org/storage/documents/grading-the-states.pdf; Sabah Muhammad, "Daniel Prude's death is a nightmare scenario for Black families like mine," *Washington Post*, Sept. 7, 2020, https://www.washingtonpost.com/opinions/2020/09/07/daniel-prude-death-policing-mental-illness.
[321] Interview with Muhammad, Oct. 12, 2021.
[322] Interview with Hancq, Oct. 7, 2021.
[323] Interview with Muhammad, Oct. 12, 2021.
[324] Ibid.
[325] Ibid.
[326] Interview with Black, Dec. 8, 2021.
[327] Ibid.
[328] Interview with Burek, Dec. 16, 2021.
[329] Interview with Black, Dec. 8, 2021.
[330] For this report, PHR's legal team conducted an in-house review of legal cases. For a comprehensive review of law and legal scholarship on "excited delirium," see Osagie K. Obasogie, "Excited Delirium and Police Use of Force," 2021. Professor Obasogie performed a Lexis search for all federal court cases with the terms "1983" and "excited delirium." He obtained 262 results, of which 195 qualified for the sample. He found, "89 of the 195 rulings (45.6%) contain language where the court, in its own voice, affirmatively asserted that excited delirium is a scientifically valid condition. … Fifty-four

cases included this type of discussion [concerning whether or not law enforcement acted reasonably under the Fourth Amendment in restraining someone with this condition], where excited delirium was discussed in a manner that favored the police in twenty-three (almost half) of these cases. … there are twenty-two rulings in the sample where the court said that the presence of excited delirium requires an arresting officer to use more care or less force. … Thirty-seven cases in the sample that we reviewed involved claims regarding the deliberate indifference to medical need, in which thirty-three (89.1%) resulted in one or more defendants being found not liable." He said his study, "highlights the malleable nature in which excited delirium is wielded by federal courts. In short, law enforcement is allowed to have their cake and eat it too. Excited delirium: (1) can be treated as a real entity that justifies the use of force that might be deadly; (2) can be questioned as a real disorder and therefore relieve officers of any duty to treat; and (3) can be used to shield officers from being held accountable for their actions, due to claims of officers' inability to fully observe excited delirium's full manifestations (yet nonetheless participate in questionable uses of force)."

[331] Mann v. Taser Int'l., Inc., 588 F.3d 1291, 1299 (11th Cir. 2009).

[332] *Hoyt v. Cooks, et al.*, 672 F.3d 972, 976 (11th Cir. 2012).

[333] *Goode v. Baggett*, 811 F. App'x 227 (5th Cir. 2020).

[334] See e.g., *Callwood v. Jones et. al.*, 727 F. App'x 552 (11th Cir. 2018); *Hoyt v. Cooks, et al.*, 672 F.3d at 976; *Batiste v. Theriot et. al.*, 458 F. App'x 351 (5th Cir. 2012).

[335] *Davidson v. City of Statesville*, 2012 WL 1441406, at *5 (W.D.N.C. 2012).

[336] *Hoyt v. Cooks, et al.*, 672 F.3d at 976.

[337] Federal Rule of Evidence 702, which has been adopted by many states, governs the admissibility of expert testimony in federal courts. This rule followed a decision by the U.S. Supreme Court holding that trial courts are to perform a "gatekeeper" function regarding the admissibility of expert testimony. *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 589 (1993).

[338] Daubert. 509 U.S. at 593-594. For applications of the *Daubert* test to the admission of medical expert testimony on "excited delirium," see, e.g., *Galack v. PTS of Am., LLC*, 2015 WL 5692327, *18 (N.D. Ga. 2015); *Estate of Barnwell v. Roane City*, 2016 WL 1457928, at *3-4 (E.D. Tenn. Apr. 12, 2016).

[339] *Estate of Barnwell v. Roane City*, 2016 WL 1457928, at *3.

[340] *Mann v. Taser Int'l., Inc.*, 588 F.3d at 976; *Estate of Barnwell v. Roane City*, 2016 WL 1457928, at *3.

[341] See, e.g., *Davidson v. City of Statesville*, 2012 WL 1441406, at *10, *14 discussing the North Carolina Basic Law Enforcement Training Program training which includes "the recognition of the symptoms of excited delirium syndrome"; *Carrier v. Mcknight*, 2017 WL 2533529, at *8 (W.D. Tex. 2017), discussing Williamson County's "Excited Delirium protocol"; *Woodward v. City of Gallatin*, 2013 WL 6092224, at *6-*7 (M.D. Tenn. Nov. 19, 2013) referring to "excited delirium" as part of TASER certification and recertification training.

[342] *Silva v. Chung*, 2019 WL 2195201, at *3; *Estate of Barnwell v. Roane City*, 2016 WL 1457928, at *3.

[343] *Silva v. Chung*, 2019 WL 2195201, at *3; Todero v. Blackwell, supra, 2021 U.S. Dist. LEXIS 188141, *5.

[344] *Todero v. Blackwell*, 2021 WL 4472550, at *2.

[345] *Mann v. Taser Int'l., Inc.*, 588 F.3d 1291.

[346] *Silva v. Chung*, 2019 WL 2195201, at *2 (D. Haw. Feb. 5, 2020); *Todero v. Blackwell*, 2021 WL 4472550, at *2 (S.D. Ind. Oct. 7, 2021).

[347] See, e.g., *Callwood vs. Jones et. al.*, 727 F. App'x at 556.

[348] *Peppers v. Washington County*, Tenn., 686 Fed. App'x. 328 (6th Cir. 2017); V. W. v. Nichelini, 2017 WL 34246 (E.D. CA Feb. 2, 2017); *Pirolozzi v. Stanbro*, F. Supp. 2d, 2009 U.S. Dist. LEXIS 42575, 2009 WL 1441070 (N.D. Ohio Mar. 13, 2009). See also *Marquez v. City of Phoenix*, 693 F.3d 1167 (9th Cir. 2012); *Gregory v. County of Maui*, 414 F. Supp. 2d 965 (D. Haw. 2006).

[349] See *Mann v. Taser*, 588 F.3d 1291; *Davidson v. City of Statesville*, 2012 WL 1441406; *Cook v. Bastin*, 590 F. App'x 523 (6th Cir. 2014); *Mingo v. City of Mobile*, 592 F. App'x 793 (11th Cir. 2014).

[350] *Hoyt v. Cooks*, et al., 672 F.3d at 976.

[351] A recent example is *Estate of Aguirre v. City of San Antonio*, 995 F.3d 395 (5th Cir. 2021). In *Aguirre*, the decedent was handcuffed and not resisting as officers forced him into a prone position with his legs crossed and pressed against his buttocks, similar to a hog-tie. Officers then put their weight on Mr. Aguirre's back, buttocks, and neck for five and a half minutes until they noticed he had stopped breathing. One officer even noted that Mr. Aguirre's lips turned blue, raising concern for hypoxia, and she decided it was because of drugs he was suspected of having consumed. 995 F.3d at 403-404. The autopsy found that Mr. Aguirre died of asphyxiation "[d]ue to the restraint by police," and his death was classified as a homicide. 995 F.3d AT 404. The plaintiffs in *Aguirre* then introduced "excited delirium" into the case, criticizing the defendant officers for not following their training and properly managing the decedent's claimed "excited delirium." 995 F.3d AT 404. The district court granted qualified immunity to the officers on the plaintiff's claims against them and also dismissed the claim against the city for constitutionally inadequate training. Ibid. The Fifth Circuit Court of Appeals reversed the grant of qualified immunity, but extensively relied on the "excited delirium" evidence submitted by the plaintiff, adding credence to this unscientific theory. Ibid. At 405, 413 n 10.

[352] *Petro v. Town of West Warwick*, 889 F. Supp. 2d 292 (D.R.I. 2012).

[353] *Estate of Hezekiah Harvey v. Roanoke City Sheriff's Office*, 2007 BL 184012, at *5 (W.D. Va. Feb. 23, 2007).

[354] Ibid.

[355] Brief of Cross-Ideological Groups Dedicated to Ensuring the Public's Trust in Law Enforcement, and Promoting the Rule of Law as Amici Curiae in Support of Petitioner at 5, *Taylor v. Riojas*, 141 S. Ct. 52 (2020), https://www.supremecourt.gov/DocketPDF/19/19-1261/143596/20200514193421509_Taylor%20v.%20Riojas%20cross-ideological%20brief.pdf.

[356] United Nations, "Universal Declaration of Human Rights (UDHR)," G.A. Res. 217A (III) at 71, art. 3, U.N. GAOR, 3d Sess., 1st plen. Mtg., U.N. Doc. A/810 (Dec. 12, 1948) https://www.un.org/en/about-us/universal-declaration-of-human-rights.

[357] United Nations, "International Covenant on Civil and Political Rights (ICCPR)," Treaty Series vol. 999, (Dec. 16, 1966):171, art. 6, https://www.ohchr.org/en/professionalinterest/pages/ccpr.aspx.

[358] UDHR, art. 7; ICCPR, art. 26.

[359] United Nations, "International Convention on the Elimination of All Forms of Racial Discrimination (ICERD)," G.A. res. 2106 (XX), Annex, 20 U.N. GAOR Supp. (no. 14) at 47, art. 5, U.N. Doc. A/6014, 660 U.N.T.S. 195, (1966), entered into force Jan. 4, 1969, https://www.ohchr.org/en/professionalinterest/pages/cerd.aspx.

[360] United Nations, "International Convention on the Protection and Promotion of the Rights and Dignity of Persons with Disabilities (CRPD)," art. 10, G.A. Res. 61/106, (Dec. 13, 2006), https://www.un.org/development/desa/disabilities/convention-on-the-rights-of-persons-with-disabilities.html.

[361] United Nations, "United Nations Human Rights Committee, General Comment No. 36, Art. 6: right to life," CCPR/C/GC/36 (Sept. 3, 2019) https://tbinternet.ohchr.org/_layouts/15/treatybodyexternal/Download.aspx?symbolno=CCPR/C/GC/36&Lang=en.

[362] United Nations, "UN Basic Principles on the Use of Force and Firearms by Law Enforcement Officials," *adopted by the Eighth United Nations Congress on the Prevention of Crime and the Treatment of Offenders, Havana, Cuba*, (Aug. 27-Sept. 7, 1990), https://www.ohchr.org/en/professionalinterest/pages/useofforceandfirearms.aspx.

[363] Ibid., par. 5(c).

[364] Ibid.

[365] United Nations, "International Covenant on Economic, Social and Cultural Rights (ICESCR)," *United Nations Treaty Series* vol. 993, no.3, art. 12, (Dec. 16, 1966), https://www.ohchr.org/documents/professionalinterest/cescr.pdf.

[366] United Nations General Assembly, "Principles for the protection of persons with mental illness and the improvement of mental health care," art. 2, G.A. res. 46/119, (Dec. 17, 1991), https://www.ohchr.org/EN/ProfessionalInterest/Pages/PersonsWithMentalIllness.aspx.
[367] ICERD, art. 5.
[368] CRPD, art 25.
[369] United Nations, "Principles on the Effective Prevention and Investigation of Extra-legal, Arbitrary and Summary Executions," arts. 1 and 9, res. 1989/65, (May 24, 1989), https://www.ohchr.org/Documents/ProfessionalInterest/executions.pdf
[370] Office of the United Nations High Commissioner for Human Rights, *The Minnesota Protocol on the Investigation of Potentially Unlawful Death (2016)* (New York/Geneva: United Nations, 2017), https://www.ohchr.org/Documents/Publications/MinnesotaProtocol.pdf.
[371] Ibid.
[372] UDHR, art. 8; ICCPR, art. 2; ICERD art. 6. See also, UN Special Rapporteur on extrajudicial, summary or arbitrary executions, "International standards," https://www.ohchr.org/EN/Issues/Executions/Pages/InternationalStandards.aspx; International Commission of Jurists, *The Right to a Remedy and Reparation for Gross Human Rights Violations: A Practitioners' Guide* (Geneva: United Nations, October 2018), https://www.icj.org/wp-content/uploads/2018/11/Universal-Right-to-a-Remedy-Publications-Reports-Practitioners-Guides-2018-ENG.pdf

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BART POLICE DEPARTMENT
EDGARDO ALVAREZ, CHIEF OF POLICE

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USE OF THE RECOVERY POSITION

This bulletin provides guidelines relating to the use of the recovery position, especially that of a restrained subject where force was used to administer the restraint.

Restraint of an individual is sometimes required after force has been applied to control an individual. Per Policy 300.3.5 the following shall apply:

"Terms such as "positional asphyxia," "restraint asphyxia," and "excited delirium" continue to remain the subject of debate among experts and medical professionals, are not universally recognized medical conditions, and frequently involve other collateral or controlling factors such as narcotics or alcohol influence, or pre-existing medical conditions. While it is impractical to restrict an officer's use of reasonable control methods when attempting to restrain a combative individual, officers are not authorized to use any restraint or transportation method which might unreasonably impair an individual's breathing or respiratory capacity for a period beyond the point when the individual has been adequately and safely controlled. Once controlled, the individual should be placed into a recovery position (e.g., supine or seated) and monitored for signs of medical distress (Government Code § 7286.5)."

The side laying fetal position is also considered a position of recovery as it places the subject in a natural position where it does not constrict the ability to breathe.

A restrained person should be placed in the recovery position if the subject is no longer actively resisting or attempting to harm others. The recovery position should also be used or attempted even if the subject is continuing to actively resist when it is safe and feasible to do so. This may be possible when additional officers are available to assist in controlling the subject into the recovery position. When placing a restrained person in the recovery position, police personnel should advise dispatch so that the attempt can be entered into the call log. Also, audible verbal articulation should be stated by on-scene personnel for the purpose of recording the recovery position attempt on body worn camera video.

Edgardo Alvarez
Chief of Police