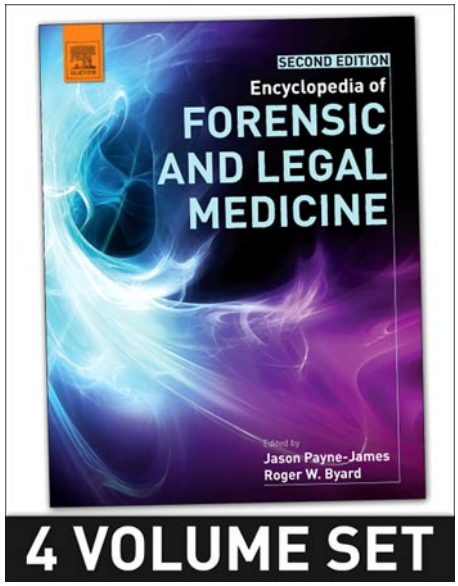

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Excited Delirium

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Abstract

Knowledge surrounding excited delirium syndrome (ExDS)/excited delirium is evolving. ExDS subjects have features that are overwhelmingly similar, especially in the context of sudden in-custody death. Those features are: tolerance to pain, constant or near constant physical activity, failing to respond to police presence, superhuman strength, rapid breathing, not tiring despite heavy exertion, naked/inappropriately clothed, sweating profusely, hot to the touch, and attraction to or destruction of glass. Features are not identical between cases; it is unknown whether any symptom/symptom cluster predicts mortality. Appropriate intervention recognizes a medical emergency that has manifest as a police problem.

Introduction

The current state of knowledge surrounding excited delirium syndrome (ExDS)/excited delirium is based in continuing additions to the work first reported by Wetli and Fishbain in the United States in the 1980s; although much older historical references also exist that may or may not be relevant to today's environment (Fishbain and Wetli, 1981; Wetli and Wright, 1979; Wetli *et al.*, 1996; Wetli and Fishbain, 1985). Literature evolving since these chapters includes recent comprehensive review chapters, pathologic literature, case reports, and emerging prospective clinical study, all of which indicate that individuals in a state of excited delirium have multiple clinical features that are overwhelmingly similar between cases, particularly when that presentation is in the context of sudden in-custody death (Takeuchi *et al.*, 2011; Vilke *et al.*, 2012a,b,c; Hall *et al.*, 2013). Medical associations in the United States, including the National Association of Medical Examiners (NAME) and the American College of Emergency Physicians (ACEP) (Vilke *et al.*, 2012a,b,c) have determined that excited delirium is a definable medical condition with a higher risk of sudden unexpected death than individuals who are not in that condition.

Trivialization of this condition is engendered when its existence is challenged by those who are under the mistaken notion that the death of subjects in a state of excited delirium only occurs when a conducted energy weapon is utilized. It is important to recognize that sudden in-custody death in this condition occurs both with and without the use of a conducted energy weapon (Stanbrook *et al.*, 2008; Truscott, 2008; Ella Baker Center for Human Rights and Edwards, 2007). There is little in medicine that causes sudden and unanticipated death in subjects, often under the age of 40, who cannot be resuscitated in the field despite advanced life support protocols and definitive emergency care. Ongoing debate on the existence of excited delirium as an entity limits funding and interest in the investigation of the heavily marginalized population experiencing this syndrome, a population comprised by and large by psychiatric patients and those intoxicated with stimulant drugs. However, some clinicians, scientists, and many prehospital agencies remain committed to discerning those features necessary to document the case definition and identify the population at risk in order to investigate treatment strategies to mitigate morbidity and mortality.

Definition

DiMaio and DiMaio (2005) described excited delirium as "combative and/or violent behavior" associated with delirium, and in the consideration of the excited delirium state, the essential underpinning is, of course, that the individual is delirious. Delirium is well described by every medical and paramedical textbook and can be defined as "an acute (minutes to hours), transient disturbance in consciousness and cognition…disorientation; disorganized and inconsistent thought processes; inability to distinguish reality from hallucinations; disturbances in speech; disorientation to time and place; misidentification of individuals" (Rosen and Barkin, 1998). For nonmedical practitioners, delirium is most simply described as a condition during which the individual interacts abnormally with the surrounding environment because of altered perception (errors in interpretation of sight, hearing, smell, touch, and taste) combined with impaired cognition. The presence of both errors/impairments is necessary for delirium to exist. Delirium is a fluctuant state, often punctuated by moments of clarity as stream of consciousness ebbs and flows.

In hospital patients, regardless of the reason for it, the presence of delirium is associated with an increase in mortality, the risk of physical injury, the length of hospital stay, and the cost of caring for the patient

(Lonergan *et al.*, 2009; Bathula and Gonzales, 2013; Burns *et al.*, 2009; Dasgupta and Brymer, 2013). In hospital patients, much effort is undertaken to anticipate delirium, thereby preventing it, with the understanding that once delirium occurs, morbidity and mortality necessarily rise even for patients in intensive care units (Burns *et al.*, 2009; Dubin *et al.*, 1979; Dasgupta and Brymer, 2013; Dovjak *et al.*, 2013; Genther and Gourin, 2012; Lee *et al.*, 2013; Morandi *et al.*, 2013). For hospital patients it is still unclear whether different interventions are required or successful in mitigating risk for different types of delirium (Lonergan *et al.*, 2009; Maneeton *et al.*, 2013).

For example, in medical patients with acute alcohol withdrawal, the presence of delirium tremens carries a significant mortality rate even if appropriately treated (Khan *et al.*, 2008). Similarly, perioperative delirium from any cause increases morbidity and mortality significantly, and entire hospital programs are targeted toward the early identification of patients at risk of perioperative delirium (Dovjak *et al.*, 2013; Genther and Gourin, 2012; Lee *et al.*, 2013). However, physicians in hospital settings have the luxury of knowing the patient's medical history accompanied by the completion of a physical examination and ancillary tests to define the nature of the delirium that is accompanied by physiologic excitation. Since the depth of knowledge about the patient varies by the environment and context in which the individual is encountered, the nosology for these events is necessarily different than the description of the same patients in an undifferentiated state in a prehospital environment without the benefit of discerning of underlying conditions and/or intoxicants. Within the hospital environment, classification of varying patient presentations is done through the use of the International Classification of Diseases (ICD) coding system defined by the World Health Organization. This system of coding patient care recognizes that different hospital personnel use different language in describing the same patient attributes. The ICD9/ICD10 manual for the classification of diseases includes at least nine different codes that describe patients with psychomotor agitation in a hospital setting (**Table 1**).

Table 1 ICD 9/10 codes for patients in hospital with psychomotor agitation

ICD9/10 code	*Descriptor*
799.2X	Abnormal excitement
799.2AM	Psychomotor excitement
799.V	Psychomotor agitation
296.00 S	Manic excitement
307.9AD	Agitation
780.09E	Delirium
293.1 J	Delirium of mixed origin
292.81Q	Delirium, drug induced
292.81 R	Delirium, induced by drug

Source: International Classification of Diseases, ICD 9/10; World Health Organization.

When individuals are delirious, whether in hospital or in other settings, there is a range of possible psychomotor manifestations ranging from quiet and introverted to highly agitated and bizarre (Aita, 1968; Detweiler *et al.*, 2009; Fink, 1999, 2000, 2013; Lonergan *et al.*, 2009; Maldeniya and Vasudev, 2013; Peitz *et al.*, 2013; Williams, 2013) (**Figure 1**). A delirious state with mental and physical quiescence can be seen in persons treated in hospital with narcotics or sedatives (central nervous systems depressants), whereas persons on the street may ingest illicit or prescription drugs, for example, narcotics such as heroin, or other 'downers' such as barbiturates. In the prehospital setting, intoxication with heroin, benzodiazepines, or a combination thereof will result in a person who is disoriented, but not active, sometimes deeply sedated enough to require life saving intervention or to be found unconscious in a public space. While there is little question that delirium associated with mental and physical suppression is of significant risk to an individual, calls for service will surround an unwanted or 'found down' person, rather than someone who comes to the attention of law enforcement because of violent activities or who dies following an intense struggle (**Figure 1**).

However, delirium is also commonly accompanied by increased psychomotor activity combined with hemodynamic derangement and metabolic compromise (**Figure 1**). Psychomotor agitation, a term commonly used by psychiatrists, succinctly describes a state of heightened brain activity (hallucinations, paranoia, and agitation) combined with physical activity (fidgeting, restlessness, running, jumping, hiding, trembling, and seizures). While it is important to note that not all individuals with psychomotor agitation are delirious, many are and the detection of a delirious state is important in identifying both risk and appropriate approach.

Identification of the underlying reason for delirium is difficult in the prehospital environment. For example, persons in the throes of delirium tremens because of

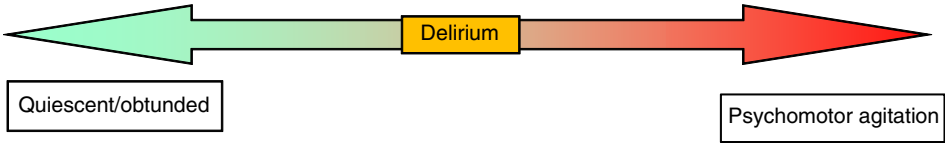

Figure 1 Continuum of possible behaviors in delirium.

Table 2 Excited delirium syndrome brief differential diagnosis

Substance intoxication
Substance withdrawal
Hypoxia
Electrolyte disturbances
Thyroid storm
Infection
Seizures
Head injury
Heat stroke
Serotonin syndrome
Neuroleptic malignant syndrome

Source: Reproduced from Vilke, G.M., Bozeman, W.P., Dawes, D.M., Demers, G., Wilson, M.P., 2012c. Excited delirium syndrome (ExDS): Treatment options and considerations. Journal of Forensic and Legal Medicine 19, 117–121.

alcohol withdrawal can be shaking, agitated, hallucinating, and have severe biophysical and biochemical derangements as a result of physiologic excitation (Hemmingsen and Kramp, 1988). In the prehospital environment, without an accurate medical history or ability to complete ancillary examinations, it would be extremely difficult to differentiate delirium tremens from intoxication with recreational stimulants like lysergic acid diethylamide (LSD), cocaine, or methamphetamine, or to determine whether an acute psychotic illness was at play. Thus the use of the generic term, excited delirium is used in the prehospital setting, rather than a diagnosis specific label, since the underlying diagnosis cannot be accurately discerned (Hall *et al.*, 2013; Storey, 1978; Takeuchi *et al.*, 2011; Vilke *et al.*, 2012a,b,c; Wesley, 2011; **Table 2**).

The adjective 'excited' was used in the first description of excited delirium in modern clinical medicine by Wetli and Fishbain, to specifically document the extreme excitation of physiologic processes that accompanies the delirious state (Fishbain and Wetli, 1981; Wetli and Fishbain, 1985). In other words, the condition is not only psychological derangement (known as agitation) but a physiologic excitation with severe hemodynamic and metabolic changes that threaten the individual's survival. While lay persons would find it easier to understand 'agitation' as a concept rather than 'excitation' because of the connotations of the words in everyday living, such a descriptor only documents the outward physical and mental manifestations of the underlying disruption, and is thus medically inadequate to describe the condition and its complexity at the cellular level.

The combination of impaired sensory input and deranged cognition means the delirious individual can be suffering from hallucinations (visual and/or auditory), abnormal skin sensations (such as the presence of imagined insects or crawling skin or even abnormal items emerging from the skin), and has an inability to respond to biofeedback messages from the body such as pain or exhaustion that would normally alter an individual's physical activity. Even with internal and external cues that what one is feeling/seeing/hearing is not normal and should not be acted upon, the person cannot make appropriate cognitive efforts to limit their psychomotor activities or comply with care provider efforts.

Modern medical literature surrounding excited delirium began with Wetli and Fishbain's descriptions of acutely agitated individuals who died in the prehospital setting and were subsequently found to have succumbed to cocaine toxicity (Fishbain and Wetli, 1981; Wetli and Fishbain, 1985). It should be noted that excited delirium is not simply a replacement term for cocaine intoxication as has been suggested by some. Individuals who use cocaine recreationally do so to improve social function through improved psychostimulation and positive energy; in general recreational cocaine users are not seeking to enter an incoherent state that deteriorates further into systemic collapse. However, excited delirium is a syndrome of devastating psychiatric discord with physiologic excitation, overload, and eventual collapse, which may lead to death; a state for which chronic cocaine use may well be a risk (Hall *et al.*, 2013; O'Halloran and Lewman, 1993; Otahbachi *et al.*, 2010; Pollanen *et al.*, 1998; Ross, 1998; Stratton *et al.*, 2001; Takeuchi *et al.*, 2011; Vilke *et al.*, 2012a,b,c). Excited delirium is not specific to cocaine intoxication and has been documented in individuals with no history of recreational drug abuse but who have died in a state of extreme agitation with delirium, in other words excited delirium (Ross, 1998; Samuel *et al.*, 2009; Takeuchi *et al.*, 2011; Vilke *et al.*, 2012a,b,c). When Wetli and Fishbain used the term excited delirium in the 1980s they were describing the complex constellation of symptoms repetitively encountered in cocaine overdosed individuals who suddenly and unexpectedly died and thus ended up on the autopsy table. Since then DiMaio and DiMaio (2005) have suggested the use of the term excited delirium when no death happens, and ExDS when the features of excited delirium are followed by the death of the subject. It is currently unknown at what point a state of excited delirium transitions from survivable metabolic derangements to a state from which evolution to cardiopulmonary collapse is inevitable.

Epidemiologic study into the actual frequency with which individuals are encountered in a state of excited delirium and undergo police use of force have begun and build on previous works which document the presence of excited delirium in individuals who have died in police custody (Hall *et al.*, 2013; Pollanen *et al.*, 1998; Ruttenber *et al.*, 1997; Vilke *et al.*, 2012a,b,c). There is little doubt that not all excited delirium events are fatal, however further epidemiologic study is required to determine the incidence of excited delirium and its accompanying morbidity and mortality with precision.

Current publication bias centers discussion of excited delirium cases on the use of conducted energy weapons by police officers (Amnesty International, 2008; Anonymous,

2009; Stanbrook *et al.*, 2008; Zimalis, 2007; Zipes, 2012). Despite public allegations and misconceptions, the presence of excited delirium as an entity is not restricted solely to law enforcement cases where conducted energy weapons are utilized. In a recent report on conducted energy weapons conducted by the Canadian Academy of Health Sciences, the committee found that there has been, to date, no demonstration of a clear causal relationship between conducted energy weapons use. That report also stated that it is clear that sudden in-custody death occurs in a complicated, multifaceted scenario that involves individuals in a state of psychomotor distress. Conducted energy weapons have been cited by coroners and medical examiners as contributing to cause of death while also being recommended for their potentially protective roles in limiting escalating agitation and physical struggles (Council of Canadian Academies and Canadian Academy of Health Sciences, 2013) mere inquiry Modern medical interest in excited delirium began nearly two decades before conducted energy weapons arrived in law enforcement.

Pathophysiology

In persons who have died in a state of excited delirium, documentable changes in neurotransmitter receptor function underpin cellular process changes that result in unopposed sympathetic stimulation resulting in metabolic stress. Dopamine is the neurotransmitter that regulates movement, hypothalamic function, higher cognitive function, and positive behavior responses. Mash *et al.* have found that dopamine processing is altered significantly in persons who die in excited delirium (Mash *et al.*, 2003, 2002, 2000, 2009; Mash and Staley, 1999). To summarize, chronic cocaine abusers have a compensatory increase in dopamine transporters which would decrease the amount of free dopamine available to stimulate the post synaptic receptors but victims of excited delirium are lacking this adaptive change and its neuron protective effect (Mash *et al.*, 2003, 2002, 2000; Mash and Staley, 1999; Wetli *et al.*, 1996; **Figure 2**). Serotonin is also an independent modulator of dopaminergic neurotransmissions and Mash *et al.* have found that ExD victims do not display an upregulation of serotonin transporters within the substantia nigra to manage excess dopamine transmission. Furthermore, Mash *et al.* also documented that heat shock proteins were significantly elevated in persons who died in ExDs, while dopamine transporters were decreased in postmortem brain tissues in those persons as compared to age-matched controls (Mash *et al.*, 2009). These findings in concert give much credence to the theory that aberrant dopamine signaling is a very important issue in the generation of a state of ExD that results in victim death. Lastly, Mash *et al.* have found that compared to nonpsychotic cocaine users, fatal ExD victims have been shown to have alterations in

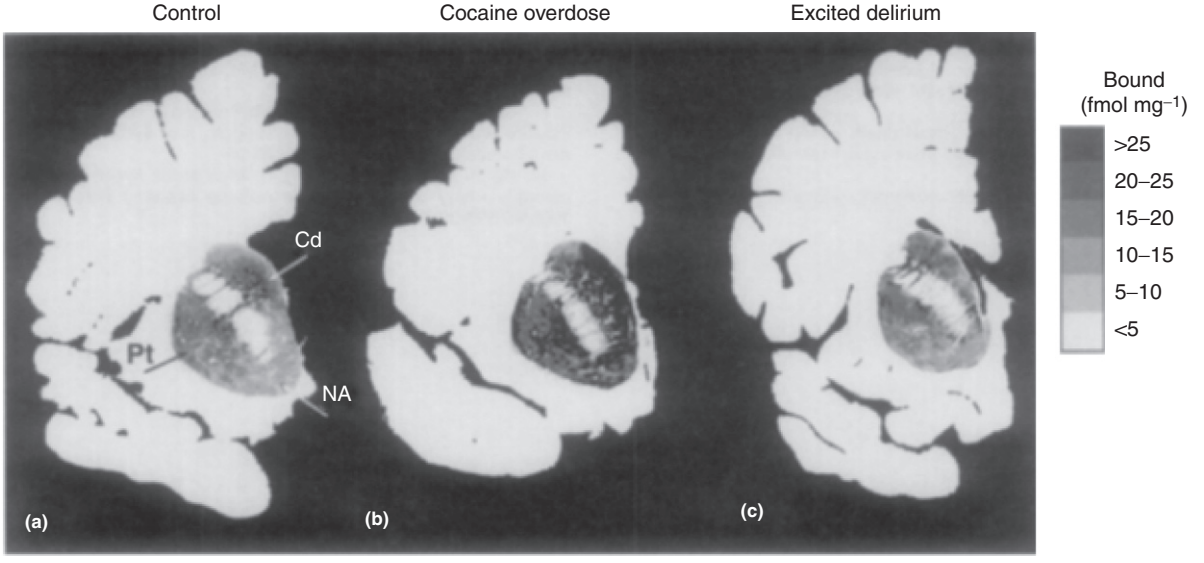

Figure 2 Abnormal dopamine uptake in the brain in ExDS. *In vitro* autoradiographic maps of [^{3}H]WIN 35 428 labeling of the dopamine transporter in coronal sections of the human brain from a representative (a) age-matched and drug free subject, (b) a cocaine overdose victim, and (c) a cocaine-related agitated delirium victim. The brain maps illustrate the adaptive increase in dopamine transporter density over the striatum in the cocaine overdose victims and the lack of any apparent elevation in the victim presenting with agitated delirium. Since the dopamine transporter regulates the synaptic concentration of neurotransmitter, the lack of compensatory upregulation may result in a dopamine overflow following a cocaine 'binge.' Elevated synaptic dopamine with repeated exposures may kindle the emergence of agitated delirium syndrome. Gray scale codes are presented at the right and are matched for the range of density values across the groups (black = high densities; gray = intermediate; and light gray to white = low to background densities). Cd, caudate; NA, nucleus accumbens, Pt, putamen. Reproduced from Wetli, C., Mash, D., Karch, S., 1996. Cocaine-associated delirium and the neuroleptic malignant syndrome. American Journal of Emergency Medicine 14 (4), 425–428.

both neuroanatomy and neurophysiology that suggest a subtype of genotype and phenotype that results in high dopamine levels and overactive autonomic function.

It is unknown whether live individuals who do not succumb to ExD have these variances, since brain tissue sampling cannot be completed in live individuals. However, research studies to examine levels of heat shock proteins and other biomarkers in live individuals with agitation have begun.

The biochemical changes as a result of unchecked sympathetic nervous system stimulation drive the increased heart rate, increased respiratory rate, increased temperature, and increased mental and physical activities of the individual. While some of these, like increased respiratory rate and psychomotor agitation may be appreciated by bystanders, until the individual is able to be physically assessed by medical practitioners, increases in blood pressure, heart rate, and temperature may not be recognized and the details of the physiologic derangement are not appreciated by bystanders. If these processes are allowed to continue unharnessed and unabating, which is the state as long as excess dopamine exists within the synapses, significant physiologic catastrophe will ensue.

Metabolic Acidosis

Precipitant States/Differential Diagnoses for Excited Delirium

The precipitant for the uncoupling of dopamine and serotonin function that generates excited delirium cannot be reliably determined in the field since information available is frequently either absent or incorrect. Once the individual has been conveyed for medical care, the task of uncovering which of a variety of potential underlying diagnoses is at play can begin. Thus, the important first step in the treatment of persons with excited delirium, like other delirious states in hospital settings, rests with recognizing its presence so that investigation and treatment can begin (Brice *et al.*, 2003; Hall *et al.*, 2013; Lonergan *et al.*, 2009; Ross, 1998; Takeuchi *et al.*, 2011; Vilke *et al.*, 2012a,b,c).

It is not surprising that the list of underlying diagnoses implicated in excited delirium are those for which sympathetic stimulation and disorders of dopamine transport are the underlying theme with the common pathway of increased sympathetic nervous system activity/autonomic arousal (Ross, 1998; Takeuchi *et al.*, 2011; Vilke and Chan, 2002; Vilke *et al.*, 2012a,b,c; **Table 2**).

Psychiatric diseases with associated psychoses such as schizophrenia and bipolar disorder, stimulant syndromes such as intoxication with cocaine, methamphetamine, bath salts (2,3 methylenedioxypyrovalerone), PCP, or 'magic' mushrooms, LSD use, drug and alcohol withdrawal, and even anticholinergic syndromes have all been implicated as underlying condition from which a state of excited delirium has evolved (Detweiler *et al.*, 2009; Fishbain and Wetli, 1981; Grant *et al.*, 2009; Hall *et al.*, 2013; Karch and Stephens, 1999; Kesha *et al.*, 2013; Lusthof *et al.*, 2011; Maher *et al.*, 2013; Mirchandani *et al.*, 1994; Morrison and Sadler, 2001; Murray *et al.*, 2012; Penders, 2013; Penders *et al.*, 2012a,b; Plush *et al.*, 2015; Ruttenber *et al.*, 1997; Stratton *et al.*, 2001; Sztajnkrycer and Baez, 2005; Takeuchi *et al.*, 2011). Other underlying diagnoses well known to generate an agitated and delirious state include whole brain processes like encephalitis, sepsis (especially in the elderly), and metabolic crises such as hypoglycemia as a result of insulin shock, toxidromes. Common toxidromes that, to the nonmedical eye, present as agitation with delirium are the anticholinergic toxidromes including the over ingestion of either prescription or nonprescription drugs.

In the prehospital environment, including police interactions, no history is either available or reliably available, subjects simply present in a state of agitation with delirium in a situational crisis that requires management without the benefit of knowing the underlying cause of the disturbance. Thus delirium with agitation that presents in the field as an agitated, incoherent, delirious person acting in a bizarre, irrational, or uncontrollable manner gains the label 'excited delirium' rather than a diagnostic one.

It is unknown whether there are specific clinical features that can define when simple agitation and physical hyperactivity have transitioned from simple drug intoxication or acute psychosis to the malignant process of ExDS with an increased risk of sudden death. It is unknown whether any specific symptoms or symptom clusters clearly predict morbidity or mortality for persons who display ExDS characteristics.

Presenting Signs and Symptoms

Until recently, the bulk of the medical literature on the topic of ExDS has come from forensic scientists and medical examiner offices, reviewing postmortem presentations. There are a number of other cohort reviews and case series that try to define presenting features of ExDS but are limited by the retrospective review process that relies on charting and documenting for the information. Being retrospective in design, they outline reported clinical characteristics of ExDS but do not describe the frequency with which clinical features are encountered by police officers interacting with subjects.

In 2010, the ACEP convened a task force to better review this topic and their findings were published confirming that ACEP has joined the NAME in recognizing ExDS as a diagnostic entity (Vilke *et al.*, 2012a,b,c). However, there is not a succinct case definition of ExDS at the present time, in part because of varying underlying pathologies that can generate an agitated and delirious state and in part because, until recently, there

Table 3 Characteristics of excited delirium, recognizable in the prehospital environment

Characteristic of excited delirium	*Common sense definition*
Naked/partially clothed	Self-explanatory
No response to police presence	Self-explanatory
Constant/near constant physical activity	Seems to never stop moving, pacing
Impervious to pain	Pain-mediated techniques ineffective
Does not fatigue	Fails to tire despite heavy physical exertion
Superhuman strength	Strength out of keeping with physical traits
Rapid breathing	Self-explanatory
Profuse sweating	Self-explanatory
Hot	Hot to the touch, does not rely on measurement
Glass attraction	Destruction of glass, mirrored surfaces

has not been prospective documentation of the frequency and profile of which features occur alone and in combination in the varying cohort of individuals who interact with police.

From the previous literature, there are specific clinical characteristics that have been previously described as being suggestive of ExDS at the time of police–public interaction and include: violent behavior, tolerance to pain, constant or near constant physical activity, subject not responding to police presence, superhuman strength, rapid breathing, does not tire despite heavy physical exertion, naked or inappropriately clothed for the environment, sweating profusely, hot to the touch, and attraction to or destruction of glass or reflective surfaces. Each of these characteristics relies on a common sense definition of its presence or absence, not reliant on intensive police training or specific scientific measure (**Table 3**).

The frequency with which these signs are seen in police use of force events has begun to be described, but it remains unknown whether any symptom or cluster of symptoms specifically predicts mortality (Hall *et al.*, 2013; Ross, 1998; Sztajnkrycer and Baez, 2005; Takeuchi *et al.*, 2011; Vilke *et al.*, 2012a,b,c; Vilke and Chan, 2002; **Tables 3** and **4**).

Intervention/Management

Recognition of excited delirium is the first step in gaining medical care to interrupt the escalated pathophysiology if there is hope of mitigating morbidity and mortality. However, medical care cannot be safely or appropriately administered in an individual who is violent, aggressive, incoherent, uncooperative, and who cannot understand or comply with direction. Thus, the first step in treating the individual once recognized is to gain and maintain physical control of the individual.

Table 4 Frequency of individual characteristics of excited delirium in a prospective law enforcement cohort of 1269 consecutive police–public interactions

	N	*% Cohort (95% CI)*
Age (mean)	31	IQR 22, 39
Male	1114	87.9 (85.9, 89.5)
Gender not recorded	2	
Comorbidities assessed at the scene	1101	86.8 (84.8, 88.6)
Emotionally disturbed person only	116	9.1 (7.6, 10.9)
Alcohol only	505	39.8 (37.1, 42.5)
Drugs only	133	10.5 (8.8, 12.3)
Any combination of comorbidities	347	27.3 (24.9, 29.9)
No ExDS characteristics	282	22.2 (20, 24.6)
Violent behavior	837	66.0 (63.3, 68.6)
Pain tolerance	264	20.8 (18.6, 23.1)
Constant/near constant activity	313	24.7 (22.3, 27.1)
Not responsive to police presence	275	21.7 (19.4, 24.0)
Superhuman strength	137	10.8 (9.1, 12.6)
Rapid breathing	123	9.7 (8.1,11.5)
Doesn't fatigue	112	8.8 (7.3, 10.5)
Naked/inappropriately clothed	94	7.4 (6.0, 9.0)
Sweating profusely	62	4.9 (3.8, 6.2)
Hot to touch	44	3.5 (2.5, 4.6)
Glass attraction/destruction	36	2.8 (2.0, 3.9)

Source: Reproduced from Hall, C.A., Kader, A.S., Danielle McHale, A.M., *et al.*, 2013. 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 20 (2), 102–107.

Despite the misconception that paramedics and medical personnel can 'capture' an individual and force them to undergo care, in North America the act of gaining physical control of an individual against his/her will or the use of force to alter a person's behavior are activities restricted to law enforcement personnel.

The most effective management of individuals in a state of excited delirium in the prehospital setting is achieved through a collaborative approach between attending law enforcement and paramedic personnel. To this end, through the combined efforts of Pennsylvania State University and the National Institute of Justice of the United States, a pocket card outlining the dual roles of law enforcement and paramedics was developed in a meeting of international experts in excited delirium and sudden in-custody death that included physicians, pathologists, law enforcement officers, paramedics, and policy makers (Hughes, 2011). The pocket card lists all known indicators of possible excited delirium on one side and the principle of Identify/Control/Sedate/Transport on the other to facilitate the recognition of the condition on the street and to document the divided yet concomitant responsibilities at the scene (**Figures 3(a)** and **(b)**).

Even with cooperative approaches, it is intuitive that physical control of the individual is unlikely to be reliably gained voluntarily in the delirious person. However, attempts at verbal command and direction are appropriate in case the individual in a moment of clarity

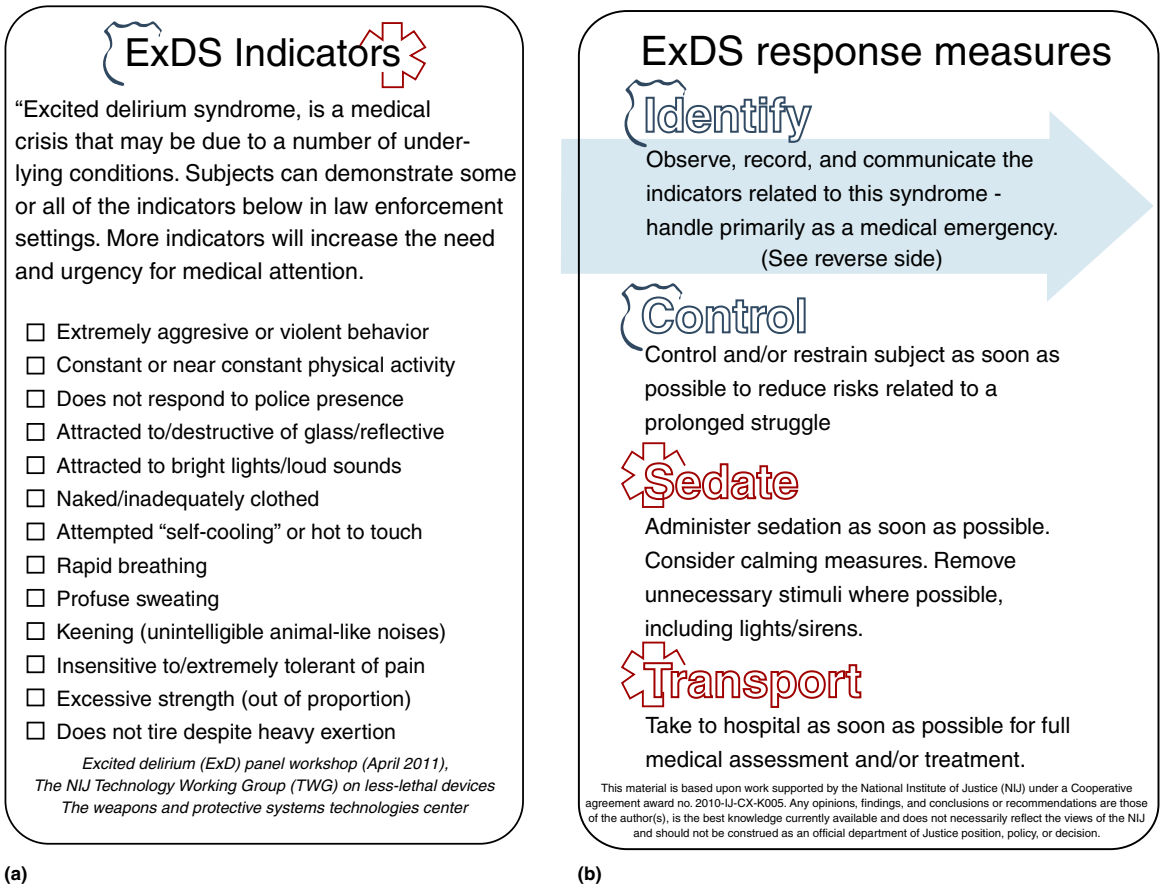

Figure 3 (a) ExDS indicators on one side of pocket card. (b) Collaborative approach between law enforcement officers and paramedics. Second side of pocket card.

is able to comply. Whether complacency will be maintained is highly questionable and a previously cooperative individual with delirium can well be expected to fail to comply suddenly and without notice (Brice *et al.*, 2003). Delirium by definition carries with it a variable interaction with the environment and the lability of cognition may prohibit a reliable cooperation from the patient despite previous short-term success.

Much criticism has been placed on the shoulders of prehospital personnel including police, fire, and paramedic staff for their application of physical control techniques in agitated individuals in a state of excited delirium. Such comments specifically demonstrate naivete to the great difficulty presented in the care and treatment of the acutely agitated patient. Perhaps it is not commonly known that patients in agitated and violent states are routinely physically restrained in hospital and while there is mortality and morbidity associated with the use of physical restraint (Abdon-Beckman, 1997; Emson, 1994; Evans *et al.*, 2003; Koiwai, 1987; Lewin, 1995; Marks, 1992; Morrison and Sadler, 2001; Paterson *et al.*, 2003; Pollanen *et al.*, 1998; Stratton *et al.*, 2001; Sullivan-Marx, 1994), physical control is necessary both to protect health and safety of healthcare practitioners – while enabling direct access to the person suffering the disorder.

Utility and approach to different de-escalation techniques are outside the scope of this chapter, save for the notation that crisis de-escalation techniques that employ a give and take approach using a cause and effect relationship to gain a common goal are unlikely to be effective in persons who, by virtue of being delirious, are unable to make appropriate cognitive decisions let alone interpret an 'if this/then that' discussion. Cognitive-based approaches are as likely to fail in these scenarios as pain-mediated control techniques.

Pain-mediated control techniques base their effectiveness in the individual receiving the painful stimulus making an appropriate cognitive decision to change his/her behavior in order to avoid further pain or discomfort. It follows that individuals who are unable to perceive pain normally or to subsequently make rational decisions through intact cognition are mentally and physically unable to comply with pain-mediated techniques in the traditional sense. Persons in a state of delirium may well interpret pain differently (impaired input from the senses including touch), and even if a painful stimulus is recognized are not able to make a

good cognitive decision to modify behavior to avoid more discomfort. Compounding the issue, persons under the influence of central dopamine agonists and/or stimulants may interpret pain differently or not feel pain at all. Those under the influence of stimulants such as cocaine may not feel pain specifically because of the central analgesic effects of the drug. Cocaine is used medically for specific painful procedures because of its ability to provide analgesic effects.

There is evidence, and intuitive plausibility, that the act of gaining and maintaining physical control during and after a struggle causes metabolic load for the patient (Ho *et al.*, 2010). The concerted struggle involved in gaining physical control of an individual carries with it a physical toll on an individual potentially already in a state of physiologic compromise particularly if the individual has been in a highly physically active state for an unknown period of time prior to a focussed physical struggle. Veterinary literature addresses the issue of capture myopathy in animals chased and cornered or simply trapped. It must also be understood that while distance measures to sedate and fell animals are geared toward limiting the struggle it must be understood that there is significant mortality following these measures.

Sedation

In medical settings, the management of delirium once it is recognized is targeted toward reducing autonomic hyperarousal in order to reduce metabolic stress and its negative effects. Anatagonists of sympathomimetic activity involve activation of GABA mediated processes to lessen psychomotor activity, tachycardia, hyperpnea, and hyptertension and the metabolic consequences of physiological excitation (Clinton *et al.*, 1987; Hick *et al.*, 1991; Peitz *et al.*, 2013; Tolonen *et al.*, 2013; Bathula and Gonzales, 2013; Carlson *et al.*, 2012; Detweiler *et al.*, 2009; Lonergan *et al.*, 2009; Maneeton *et al.*, 2013).

Vilke *et al.* (2012a,b,c) recently reviewed strategies for the prehospital management of ExD subjects (**Table 5**). While sedation is the mainstay of therapy in and out of hospital, it should be understood that sedation is the beginning of therapy not the end of it. It also must be understood that prehospital sedation is not a guarantee of survival for persons with excited delirium and diligent monitoring for signs of decompensation must continue throughout transport of the individual to the hospital.

One of the benefits of appropriate sedation is the maintenance of physical control such that the patient can be cared for; however, the primary goal of sedation is to arrest the overwhelming physiologic excitation that is present. Appropriate medication regimes for patients with ExDS consist of the benzodiazepines, the antipsychotics (including atypical antipsychotics), and newer dissociative agents such as ketamine (Nobay *et al.*, 2004; Richards *et al.*, 1998; Thomas *et al.*, 1992; TREC Collaborative Group, 2003; Burnett *et al.*, 2012; Ho *et al.*, 2013).

Thus, treatment regimes are intuitive, based in the prescribing habits of the clinician involved and the risk profile is based on an understanding of the treatment profile for individuals in other clinical scenarios.

Patients suffering from ExDS rarely self-present to medical facilities although some individuals may be

Table 5 Medication treatment options

Medication (trade name)	*Administration routes*	*Typical dosing (mg)*	*Onset (min)*	*Duration (min)*
Benzodiazapines				
Midazolam (Versed)	IN	5	3–5	30–60
	IM	5	10–15	120–360
	IV	2–5	1–5	30–60
Lorazepam (Ativan)	IM	4	15–30	60–120
	IV	2–4	2–5	60–120
Diazepam (Valium)	IM	10	15–30	15–60
	IV	5–10	2–5	15–60
Antipsychotics				
Haloperidol (Haldol)	IM	10–20	15–30	180–360
	IV	5–10	10	180–360
Droperiodol (Inapsine)	IM	5	10–30	120–240
	IV	2.5	10	120–240
Ziprasidone (Geodon)[a]	IM	10–20	15–30	240
Olanzapine (Zyprexa)[a]	IM	10	15–30	24 h
NMDA receptor antagonist/dissociative				
Ketamine (Ketaset, Ketalar)	IM	4–5 mg kg^{-1}	3–5	60–90
	IV	2–4 mg kg^{-1}	1	20–30

[a]A typical antipsychotic medications.

Source: Reproduced from Vilke, G.M., Bozeman, W.P., Dawes, D.M., Demers, G., Wilson, M.P., 2012c. Excited delirium syndrome (ExDS): Treatment options and considerations. Journal of Forensic and Legal Medicine 19, 117–121.