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Emergency Department Management of the Agitated Pediatric Patient

Identifying and managing agitated pediatric patients in the emergency department (ED) can be stressful and challenging for patients, families, and providers. ED visits for psychiatric or behavioral complaints constitute approximately 3-4% of the more than 30 million annual pediatric ED visits.[1] Encounters likely will rise as the availability of community mental health resources remains low or even decreases, insurance providers limit reimbursement for mental health services, and recognition of mental health disorders increases in the general population. In a study evaluating psychiatric-related visits to the ED, children with psychiatric-related complaints used disproportionately more hospital resources. These patients had higher rates of recidivism, longer ED stays, and higher probability of admission or transfer compared to their counterparts with non-psychiatric-related complaints.[2] Yet, many providers may be uncomfortable with treating agitation in the pediatric population. Often, ED providers have less experience using antipsychotics and benzodiazepines in children and adolescents as compared to adults, and have less data to rely on for the efficacy and safety of these agents in the pediatric population.

— Ann M. Dietrich, MD, Editor

Introduction

Understanding the agitated pediatric patient in the ED requires looking beyond simply psychiatric chief complaints. While agitation can be a manifestation of psychiatric disease, agitation may be a symptom of other disorders and even a byproduct of the ED environment itself. It is the ED physician's task to recognize patients at risk for agitation or violence, to treat or to exclude reversible etiologies, and finally, if possible, to de-escalate or to institute measures along a continuum to manage agitation when it arises.

Predicting Risk and Assessing Agitation

Agitation may be part of the initial presentation, occur as a natural progression of an underlying condition, or occur as a reaction to the ED experience itself. Manifestations of agitation may be wide-ranging in pediatric patients, and ED physicians must have a high index of suspicion for at-risk patients in order to intervene early.

Agitation is "a state of behavioral dyscontrol that will likely result in harm to the patient or health care workers without intervention."[3] In children, agitation often progresses in a stepwise fashion, providing clues and opportunities for early intervention. First is the verbal stage, in which the child will use general threats and/or abusive language. In the second stage, the motor stage, children will remain in near constant motion, such as pacing. In the third, or property damage stage, the patient becomes destructive, attempting to break nearby objects.

Executive Summary

- Agitation may be part of the initial presentation, occur as a natural progression of an underlying condition, or occur as a reaction to the ED experience itself. Manifestations of agitation may be wide-ranging in pediatric patients, and ED physicians must have a high index of suspicion for at-risk patients in order to intervene early.
- Some of these conditions are easily reversible and should be considered early. The "GOT IVS" mnemonic is a helpful reminder of commonly encountered conditions, including hypoglycemia, hypoxia, infection, and trauma.
- Primary psychiatric disorders, such as major depression or bipolar disorder, should only be diagnosed after other causes of agitation have been excluded.
- If a provider encounters agitation, a state that will likely result in harm without further intervention, interventions should progress from least restrictive to most restrictive.
- Calming interventions and space modifications should occur first. If pharmacologic intervention is needed, appropriate symptom-focused treatment should be used. Only if necessary, medications such as benzodiazepines and antipsychotics may be used as chemical restraints. Additionally, for patient and staff safety, physical restraints may also be indicated. If restraints are necessary, providers must be familiar with The Joint Commission and hospital-specific requirements and protocols.

Finally in the attack stage, children may attempt to harm themselves or others. Although each individual's course may be different, it remains imperative that providers recognize the early signs and states of agitation in order to avert or de-escalate the behavior.[4]

While it is an important diagnosis on the differential, agitation is not confined to those children with a primary psychiatric disorder. Children must be thoroughly evaluated, including a detailed history and physical exam, to determine if there is any underlying condition that either may be causing agitation or exacerbating a chronic condition. Medical conditions such as head injuries, intracranial infections such as meningitis, metabolic abnormalities, and ingestions may cause psychosis or put patients at greater risk for agitation. Some of these conditions are easily reversible and should be considered early. The "GOT IVS" mnemonic is a helpful reminder of commonly encountered conditions, including hypoglycemia, hypoxia, infection, and trauma.[5] (*See Table 1.*) Primary psychiatric disorders, such as major depression or bipolar disorder, should only be diagnosed after other causes of agitation have been excluded.

In addition, certain neurodevelopmental conditions may place children at a greater risk for agitation in an unfamiliar, unstructured, and sometimes chaotic environment. More vulnerable populations include children with intellectual disabilities, attention deficit hyperactivity disorder, and autism spectrum disorders (ASD). Beyond the difficulty these populations may have with the new and uncertain environment of an ED, comorbid psychiatric disorders exist at higher rates; as high as 80% of children with ASD may have a comorbid psychiatric disorder such as bipolar disorder.[6] A recent study found that 13% of ED visits among children with ASD were due to psychiatric emergencies as opposed to 2% in the general population.[7] In part, this may be due to a lack of access to mental health services, a paucity of mental health professionals trained for these special populations, and poor insurance coverage of mental health services for individuals with an ASD diagnosis.[7]

Finally, intoxication or ingestion, intentional or accidental, may present as a behavioral emergency. (*See Table 2.*) A review of data from National Hospital Ambulatory Medical Care Survey from 1993-1999 shows that substance-related disorder was the most common diagnosis for ED mental health visits at almost 25%.[8] The differential of substances that can cause agitation is broad. Common classes include alcohol, anticholinergics, sympathomimetics, hallucinogens, cannabis and its derivatives, and new agents are constantly emerging. Anticholinergic ingestion may present as new-onset psychosis. Other signs and symptoms include urinary retention, hyperthermia, hypertension, tachycardia, diaphoresis, and quiet or absent bowel sounds.[9] Agents responsible for anticholinergic syndrome include diphenhydramine, scopolamine, and jimson weed. Sympathomimetics may exacerbate underlying psychiatric disorders or place patients in a heightened state of irritability.[5] Offending agents include over-the-counter medications such as pseudoephedrine, dextroamphetamine, as well as drugs of abuse such as

Table 1. GOT IVS

G	Glucose: hypoglycemia
O	Oxygen: hypoxia
T	Trauma: head injury
	Temperature: hyper- or hypothermia
I	Infection: meningitis, encephalitis, brain abscess or sepsis
V	Vascular: stroke, subarachnoid hemorrhage
S	Seizure: postictal or status epilepticus

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MDMA, methamphetamine, and cocaine. Patients using hallucinogens such as phencyclidine (PCP), dextromethorphan, and ketamine may present in a dissociative state. But the most commonly used drug in the United States is marijuana. Activation of cannabinoid receptors can cause panic and anxiety.[10] Another increasingly popular drug among adolescents is salvia divinorum. Salvia has the hallmark of dysphoria, synesthesia, and visual hallucinations.[11] As the universe of potential intoxications and ingestions grows, ED providers should have a low threshold to consult their toxicologist or Poison Control in behavioral emergencies.

Table 2. Ingestions and Toxidromes

Ingestion	Presentation
Sympathomimetics	Hyperthermia, tachycardia, mydriasis, diaphoretic, hyperalert, hallucinations, paranoia
Anticholinergics	Hyperthermia, tachycardia, mydriasis, dry skin, visual hallucinations, psychosis, delirium, urinary retention, decreased bowel sounds
Opioids	Hypothermia, bradycardia, miosis, depressed mental status, confusion, hyporeflexia
Hallucinogens	Tachycardia, nystagmus, hallucinations, depersonalization, euphoria
Ethanol	Hypothermia, bradycardia, altered mental status, ataxia, slurred speech
Serotonin syndrome	Hyperthermia, tachycardia, mydriasis, diaphoretic, confusion, agitation, or coma, tremor, hyperreflexia, clonus

Agitation Management

As with many ED protocols, the ED provider must remember that agitation interventions progress from the least restrictive (environmental alterations) to most restrictive (chemical or physical restraints), unless safety is immediately at risk. (*See Figure 1.*) Evaluation for possible reversible etiologies and appropriate treatment based on these diagnostics should continue as soon as it is safe for the patient and staff.

Optimizing the Environment

First, the ED staff may simply begin with the room. If space provides, your ED can designate a room to be a "safe room." This room will have less equipment, fewer breakable items, and provide a quiet space. However, any room can be adjusted quickly to provide greater safety. Staff can remove objects that are distracting or agitating (e.g., monitors, computers), that create a risk to the patient (e.g., tubing or wires), or that clutter the space (e.g., unnecessary stands or chairs). Less stimulation in the form of objects, people, noise, and even light may be helpful. If family or friends are heightening rather than alleviating agitation and anxiety, then the ED provider should ask them to leave temporarily. In the end, creating a safe space may provide a greater sense of control. For example, allowing the patient room to pace can alleviate anxiety or psychomotor agitation.

Second, the ED staff must be mindful of their own reaction to the situation. Listening, empathizing, and being mindful of your own responses to the child's actions are key. Both the content and style of language are important to creating a calm environment. Using a calm, soft voice at a slow pace will be reassuring. ED providers can minimize uncertainty by clearly introducing themselves and explaining procedures and time course to patients and to their families. Reassure the patient that your goal is to keep him or her safe. Further, clarify the patient's goals as well. Understanding the patient's concerns and goals will help you connect these to actions, rewards, and an improved sense of control. ED providers must not take the patient's anger personally. Other calming interventions may include:

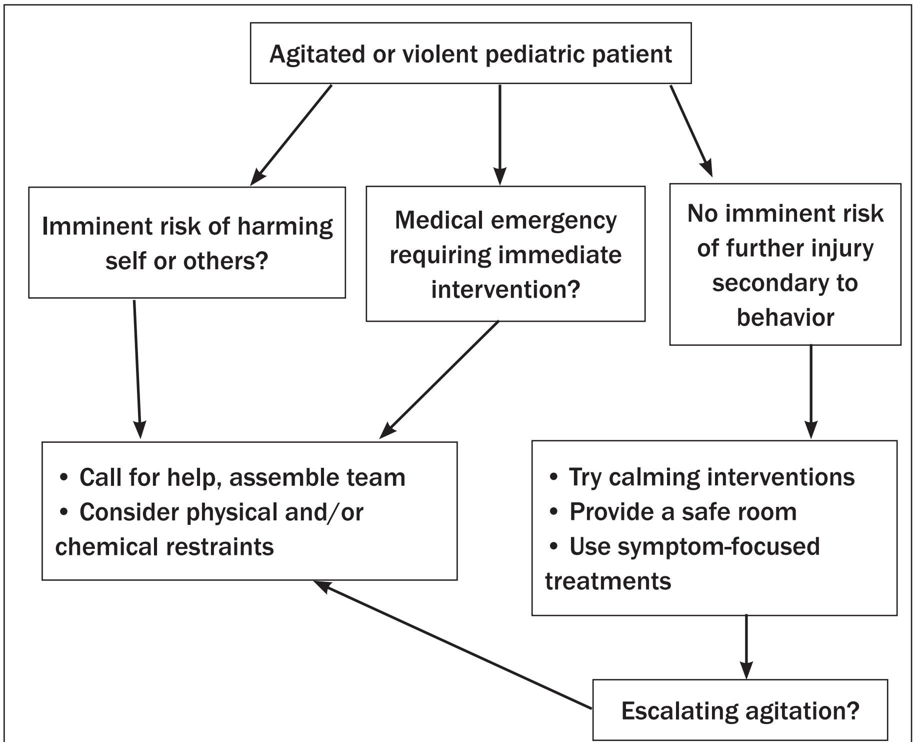
- offering a warm blanket, food, or drink if possible;
- offering discrete choices, such as choosing to have lights on or off, choosing to have parents in or out of the room, choosing what type of juice or snack to have;
- offering toys or similar items that may distract the patient from uncomfortable procedures;
- engaging consultants, such as social work, psychiatry, child life, and security as needed.[12]

Psychopharmacology: Symptom-Focused Treatment versus Restraint

If the de-escalation efforts described fail to work, ED providers will need to turn to pharmacologic interventions. The same classes of medications are used to prevent further agitation as well as to restrain. As described by the Centers for Medicare and Medicaid Services, a drug or a medication becomes a restraint "when it is used as a restriction to manage the patient's behavior or restrict the patient's freedom of movement and is not a standard treatment or dosage for the patient's condition."[13] Use of these medications short of this would be considered symptom-focused treatment.

Therefore, when possible ED providers should start with symptom-focused treatment. It is imperative to know as much as possible about the patient's other medical conditions, current medications, allergies or other adverse reactions, and any possible ingestions (medication, alcohol, or otherwise).[1] There are at least three scenarios in which an

Figure 1. Management of an Agitated Patient



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ED provider should use symptom-focused treatment. First, the patient may have missed doses of his usual medications because he had to come to the ED. If so, providing a patient with his usual medication regimen is an appropriate first step, unless there is a concern for overdose or other complicating ingestion. Second, even if the patient has not missed a dose but is taking an antipsychotic or a benzodiazepine for anxiety, one-quarter to one-half of his daily dose in between scheduled doses may help to treat agitation or anxiety.[12] Third, patients without an underlying psychiatric diagnosis may benefit from symptom-specific treatment for anxiety, agitation, or psychosis to prevent escalation. Oral medications are preferred to intramuscular administration, and while not as rapid in onset, are as effective.[14] In this setting, the ED provider should use doses in the lower therapeutic range to achieve symptom-focused treatment rather than a level of sedation that restricts the patient's freedom of movement. Table 3 provides recommendations based on symptoms.[12]

Ultimately, chemical restraints may be necessary for the safety of the patient and the staff. According to The Joint Commission, restraints may be used only when clinically justified or if the patient's behavior may endanger the patient or staff.[15] Thus, the same medications used for symptom-focused treatment become chemical restraints when used specifically to prevent imminent injury to the patient and others, and when the medication is not part of the patient's usual or expected treatment plan. Clinical justifications for restraints must be documented and may include decreasing a patient's anxiety and, thus, preventing an escalation of behavior, minimizing disruptive behavior, or providing urgent medical aid. Restraints must not be used as a form of punishment or for convenience. The Joint Commission requires that hospitals have written policies and procedures guiding the use of restraints.[15] Providers must be familiar with their hospital's policies, including requirements regarding initiation and application of restraints, patient monitoring while restrained, reassessment of necessity, documentation, and reporting complications.

Medications

Due to the lack of studies in pediatric patients, ED providers must be familiar with a range of medications that can be used to treat varying levels of agitation, taking into consideration patient characteristics and the particular features of his or her agitation. (*See Table 4.*)

Antihistamines. Diphenhydramine is commonly used in the pediatric ED for other chief complaints; thus, providers and even families are well familiar with its sedating properties. It is most appropriate for milder agitation as symptom-focused treatment.[1] Some patients, particularly children with intellectual disabilities or developmental disorders, may have a paradoxical reaction to diphenhydramine, leading to disinhibition and possibly worsened agitation.[3] As with any antihistamine, there is a risk of anticholinergic side effects such as dry mouth, tachycardia, dizziness, constipation, urinary retention, delirium, and seizures or arrhythmias. If this occurs, most patients need only supportive care; the use of physostigmine is controversial.[16]

Benzodiazepines. Benzodiazepines are an appropriate pharmacologic intervention for several etiologies causing agitation, including panic disorders, anxiety, alcohol withdrawal, and cocaine use.[1] Further, they are often the drugs of choice for sedation because of their rapid onset and variety of available routes of delivery. While there are no data in children showing superiority of any particular benzodiazepine, lorazepam is frequently used for its rapid onset, short half-life, multiple routes of administration (sublingual, IM, rectal, oral, IV), and lack of active metabolites.[17] In a study by Dorfman, 82% of emergency medicine residencies and 82% of pediatric emergency medicine fellowships used benzodiazepines for chemical restraint of children age 12 years old and younger, with lorazepam being the most commonly cited.[18] The rates of benzodiazepine use were similarly high in children age 13 years or older.

The most common complication of benzodiazepines is respiratory depression, which underscores the need for close respiratory monitoring. Benzodiazepines may also cause confusion, ataxia, and nausea. Similar to diphenhydramine, some patients may have a paradoxical response to the medication and become increasing agitated. In particular, patients with developmental delay or organic brain disorders may be at increased risk for this side effect.[17]

Neuroleptics. The use of neuroleptics in the ED relies less on their antipsychotic effects and more so on their sedative properties. Typical antipsychotics such as haloperidol are dopamine receptor antagonists. Atypical antipsychotics, such as risperidone, olanzapine, and ziprasidone, are serotonin-dopamine receptor antagonists. Most ED providers are familiar with using haloperidol across a range of agitation presentations. However, risperidone has been studied in pediatric patients, specifically with autism or pervasive developmental disorders, demonstrating effectiveness at controlling aggression and self-injury.[3] Ziprasidone may be best for patients with underlying schizophrenia, and is used routinely in patients with Tourette syndrome.[17] Olanzapine is recommended for patients with underlying schizophrenia, bipolar mania, and dementia.

Of note, the use of droperidol remains controversial. In 1995, the Food and Drug Administration issued a black box warning for droperidol for QT prolongation. In one retrospective chart review, 79 pediatric ED patients ages 13 to 21 years old who were given droperidol reported no cardiac arrhythmias while on continuous monitoring.[19] Similarly, a review of 2,468 ED patients ages 20 months to 98 years who receive droperidol showed only one case of cardiac arrest.[20] The cardiac arrest occurred 11 hours after administration in a cocaine-intoxicated patient. At this time, the use of droperidol remains controversial.

Table 3. Symptom-based Treatments

Symptom	Medication
Anxiety	Lorazepam, diazepam, or diphenhydramine
Psychosis or mania	Risperidone, olanzapine, ziprasidone or haloperidol
Impulsivity, maladaptive aggression	Risperidone or olanzapine

Unlike their newer counterparts, typical antipsychotics have a greater risk of complications. In addition to oversedation, side effects include extrapyramidal symptoms (EPS), neuroleptic malignant syndrome (NMS), lowering of seizure threshold, and QT prolongation.[3] EPS may manifest as akathisia or dystonic reactions such as oculogyric crisis, torticollis, or opisthotonos. The incidence of EPS is low, approximately 1%.[17] The treatment of EPS is diphenhydramine IV or IM, or benztropine IV or IM.[21] NMS is a potentially fatal side effect that may occur at any point with antipsychotic treatment. It manifests as autonomic instability with hyperthermia, altered mental status, and muscle rigidity. There have been no reports in the literature of pediatric fatalities from NMS since 1986.[3] If NMS is suspected, however, stop the offending agent and provide supportive care, including hydration and fever control. Benzodiazepines can be given to treat muscle rigidity and prevent rhabdomyolysis. Consider bromocriptine, a dopamine agonist, as a reversal agent in critically ill patients only.[22]

QT prolongation may occur with both typical and atypical antipsychotics, which may lead to torsades de pointes. Of the atypical antipsychotics, ziprasidone causes the most significant QT prolongation.[23] Patients should be assessed for signs or symptoms that signal that the patient is at increased risk of developing torsades, such as known long QT interval, syncope, palpitations, congenital deafness, or early sudden death in the family. Ziprasidone poses the most significant risk of QT prolongation, but there have been no reported cases of QT prolongation with olanzapine.[17]

Combination Therapy. There are no studies comparing a single agent to combination therapy in children. In adults, haloperidol and lorazepam are a common combination, as both can be delivered in the same syringe. A double-blind, randomized, prospective study in adults demonstrated more rapid improvement in agitation and less EPS with haloperidol given with lorazepam as compared to haloperidol alone.[24]

Physical Restraints

Finally, physical restraints may be necessary with some adolescent patients. Physical restraints ultimately are needed to limit mobility to administer chemical restraints, initiate treatment for reversible etiologies, and/or to keep the patient safe as the medication begins to take effect. If an ED provider decides physical restraints are necessary, a team approach should be used, with one person to restrain each limb and one person to protect the patient's head. Only devices approved as restraints should be used, with the least restrictive means used at all times. A patient should be restrained in the supine position, with arms at the side, and the restraints tied to the gurney frame, rather than the side rails. The prone position should be avoided, as it may put the patient at greater risk for asphyxiation. Once restrained, patients will continue to require close monitoring, including

Table 4. Chemical Restraints

Medication	Dose	Route	Max	Onset	Half-life
Diphenhydramine	1 mg/kg/dose	PO/IM/IV	50 mg	15-30 min	2-8 h
Diazepam	0.04-0.2 mg/kg/dose	PO/IM/IV	0.6 mg/kg/8 h IM/IV	1-2 h PO 20-30 min IM	30-60 h
Lorazepam	0.05 mg/kg/dose	PO/IM/IV	2 mg/dose	16 h PO 20-30 min IM 5-20 min IV	14 h
Midazolam	0.025-0.05 mg/kg/dose if 6-12 years	IV/IM	10 mg	3-5 min IV 15 min IM	2-6 h
	0.5 mg/kg/dose	PO	20 mg	20-30 min	
Haloperidol	0.025-0.075 mg/kg/dose	IM	5 mg/dose	2-6 h PO 30-60 min IM/IV	12-18 h
Risperidone	0.25 mg (school age) to 0.5 mg (late adolescent)	PO	—	30-60 min	20 h
Olanzapine	2.5 mg (school age) to 10 mg (late adolescent)	PO	—	5-8 h PO	20-50 h
Ziprasidone	10 mg if 12-16 Y 10-20 mg if > 16 Y	IM	—	4-5 h PO 60 min IM	14 h PO 4-10 h IM

vital signs and reassessment to determine the earliest possible removal of restraints. Once the patient no longer poses a danger to him- or herself or to staff, then restraints may be released. Depending on the patient's improvement, a provider may be able to release all restraints at once or in a stepwise fashion.

Physical restraints pose certain risks. In addition to monitoring a patient's mental and respiratory status, it is important to look for signs of skin breakdown or even neurovascular damage if a restraint is too tight.[18] If a patient's agitation is not well controlled, the patient may be at risk of developing rhabdomyolysis while constantly fighting against the restraint. Again, as soon as is safe for the patient and staff, restraints should be removed.

Conclusions

Pediatric behavioral emergencies are increasingly common in the ED. Providers must complete a thorough history and physical exam to quickly identify reversible causes of agitation or conditions that are the etiology for or that may exacerbate underlying disorders leading to behavioral disinhibition. If a provider encounters agitation, a state that will likely result in harm without further intervention, interventions should progress from least restrictive to most restrictive. Calming interventions and space modifications should occur first. If pharmacologic intervention is needed, appropriate symptom-focused treatment should be used. Only if necessary, medications such as benzodiazepines and antipsychotics may be used as chemical restraints. Additionally, for patient and staff safety, physical restraints may also be indicated. If restraints are necessary, providers must be familiar with The Joint Commission and hospital-specific requirements and protocols. The selection of medication will depend on patient history, exam, and presentation. At the earliest point possible based on treatment efficacy and patient safety, physical restraints should be removed. Finally, providers should be mindful that successful handling of behavioral emergencies requires a team approach involving different levels of staff, patients, and families.

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CME Questions

1. An 11-year-old male presents as a helmeted bicyclist who was struck by a car at 10 mph. His past medical history is notable for insulin-dependent diabetes mellitus. Further review of the history is negative for ingestions. He is confused and combative. Which of the following are possible causes for his agitation?

   A. hypoglycemia
   B. hypoxia
   C. head injury
   D. all of the above

2. It is imperative that providers recognize the early stages of agitation. Which of the following most accurately describes the progression of agitation?

   A. property damage stage, motor stage, verbal stage, attack stage
   B. motor stage, verbal stage, attack stage, property damage stage
   C. verbal stage, motor stage, property damage stage, attack stage
   D. motor stage, property damage stage, verbal stage, attack stage

3. A 6-year-old female with Down syndrome presents for a URI; pulse oximetry is 98% in room air and vital signs are within normal limits. She is becoming agitated. Which of the following are appropriate calming interventions?

   A. Offer a choice of snacks or juice.
   B. Turn off all monitors, remove O2 saturation probe.
   C. Provide distractions, if available, including movies, toys, or books.
   D. Include parents in physical exam process and explain each part to parent and patient.
   E. All of the above.

4. A 15-year-old male is brought in by his brother who reports he has been drinking alcohol and smoked "something." He is combative and confused. Other than tachycardia, vital signs are within normal limits. The patient is yelling at staff, beginning to throw objects, and threatening to harm himself. Despite your best efforts at calming him, the patient remains violent, and you must initiate restraints. According to The Joint Commission, all of the following must be in place *except*:

   A. an order initiating restraints
   B. patient consent
   C. monitoring and documentation while the patient is in restraints
   D. institution-specific written procedures and policies for the use of restraints

5. Your team is able to successfully place the above patient in physical restraints and he is given haloperidol and lorazepam IM. He is now sleeping. In addition to cardiac and respiratory monitoring, what else should nursing monitor while the patient is in restraints?

   A. skin breakdown
   B. neurovascular exam in extremities restrained
   C. serial abdominal exams
   D. A and B
   E. none of the above

6. A 16-year-old female with a history of bipolar disorder presents with mania. She is pacing around the room and is difficult to redirect as she becomes increasingly agitated. Vital signs are stable and she weighs 55 kg. Per her parents, she normally takes lithium and olanzapine. Which of the following is the most appropriate first pharmacologic intervention?

   A. haloperidol 5 mg IM x 1
   B. lorazepam 0.5 mg PO x 1
   C. olanzapine 2.5 mg PO
   D. diphenhydramine 25 mg PO x 1

7. You have just given a 13 year-old male patient haloperidol 2.5 mg IM x 1. Initially he appeared more sedate, but the nurse has called you to the bedside and you note that he has a temperature of 38.9°C, blood pressure of 162/90, a pulse of 110, and a respiratory rate of 18. On exam, he is confused, with increased tone throughout. What is the next best treatment?

   A. supportive measures including fluid bolus and anti-pyretic
   B. Benadryl 50 mg IV x 1
   C. dantrolene 0.5 mg/kg/dose
   D. A and C
   E. none of the above

8. You have just given a 17-year-old female patient, who presented with combativeness and hallucinations, haloperidol 5 mg IM x 1. Her agitation improves, but she is complaining of neck pain and is now holding her head turned to the right. On arrival and now, vital signs have been within normal limits; she has suffered no trauma.What might this be an example of?

   A. malingering
   B. extrapyramidal symptoms
   C. paradoxical response
   D. neuroleptic malignant syndrome

9. A 7-year-old female patient with a history of autism and attention deficit hyperactivity disorder presents with urticaria after eating strawberries, a known allergen for her. You give her Benadryl, and she begins running around the room, is difficult to control, and is becoming increasingly agitated. What is this an example of?

   A. extrapyramidal symptoms
   B. paradoxical response
   C. intoxication
   D. neuroleptic malignant syndrome

10. A 15-year-old female presents to your ED with psychosis. She has a history of bipolar disorder with psychotic features. According to her parents, the patient syncoped last month and was told her ECG was abnormal, but they cannot recall why. If the patient requires treatment for her agitation, which of the following would be the safest?

    A. ziprasidone
    B. olanzapine
    C. droperidol
    D. haloperidol

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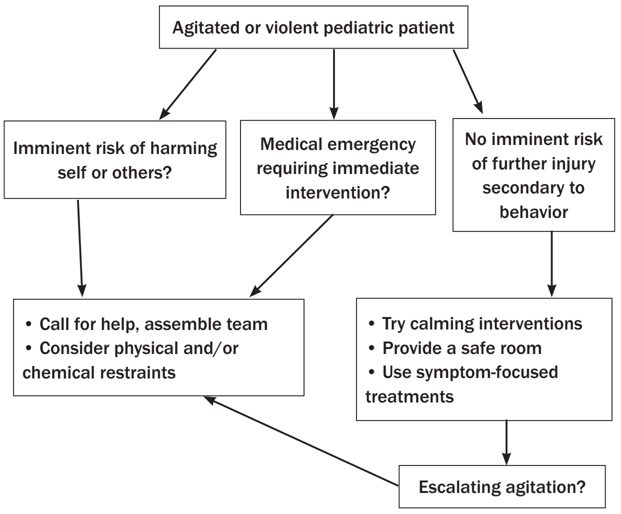
Pediatric Emergency Medicine Reports

The Practical Journal of Pediatric Emergency Medicine

Emergency Department Management of the Agitated Pediatric Patient

Management of an Agitated Patient



Adapted with permission from: Hilt RJ, Woodward TA. Agitation treatment for pediatric emergency patients. *J Am Acad Child Adolesc Psychiatry* 2008;47:132-138.

Ingestions and Toxidromes

Ingestion	Presentation
Sympathomimetics	Hyperthermia, tachycardia, mydriasis, diaphoretic, hyperalert, hallucinations, paranoia
Anticholinergics	Hyperthermia, tachycardia, mydriasis, dry skin, visual hallucinations, psychosis, delirium, urinary retention, decreased bowel sounds
Opioids	Hypothermia, bradycardia, miosis, depressed mental status, confusion, hyporeflexia
Hallucinogens	Tachycardia, nystagmus, hallucinations, depersonalization, euphoria
Ethanol	Hypothermia, bradycardia, altered mental status, ataxia, slurred speech
Serotonin syndrome	Hyperthermia, tachycardia, mydriasis, diaphoretic, confusion, agitation, or coma, tremor, hyperreflexia, clonus

Symptom-based Treatments

Symptom	Medication
Anxiety	Lorazepam, diazepam, or diphenhydramine
Psychosis or mania	Risperidone, olanzapine, ziprasidone or haloperidol
Impulsivity, maladaptive aggression	Risperidone or olanzapine

GOT IVS

G	Glucose: hypoglycemia
O	Oxygen: hypoxia
T	Trauma: head injury Temperature: hyper- or hypothermia
I	Infection: meningitis, encephalitis, brain abscess or sepsis
V	Vascular: stroke, subarachnoid hemorrhage
S	Seizure: postictal or status epilepticus

Used with permission from: Rossi J, Swan MC, Isaacs ED. The violent or agitated patient. *Emerg Med Clin North Am* 2010;28:235-256.

Chemical Restraints

Medication	Dose	Route	Max	Onset	Half-life
Diphenhydramine	1 mg/kg/dose	PO/IM/IV	50 mg	15-30 min	2-8 h
Diazepam	0.04-0.2 mg/kg/dose	PO/IM/IV	0.6 mg/kg/8 h IM/IV	1-2 h PO 20-30 min IM	30-60 h
Lorazepam	0.05 mg/kg/dose	PO/IM/IV	2 mg/dose	16 h PO 20-30 min IM 5-20 min IV	14 h
Midazolam	0.025-0.05 mg/kg/dose if 6-12 years	IV/IM	10 mg	3-5 min IV 15 min IM	2-6 h
	0.5 mg/kg/dose	PO	20 mg	20-30 min	
Haloperidol	0.025-0.075 mg/kg/dose	IM	5 mg/dose	2-6 h PO 30-60 min IM/IV	12-18 h
Risperidone	0.25 mg (school age) to 0.5 mg (late adolescent)	PO	—	30-60 min	20 h
Olanzapine	2.5 mg (school age) to 10 mg (late adolescent)	PO	—	5-8 h PO	20-50 h
Ziprasidone	10 mg if 12-16 Y 10-20 mg if > 16 Y	IM	—	4-5 h PO 60 min IM	14 h PO 4-10 h IM

Common Pediatric Psychiatric and Behavioral Disorders

Type of Disorder	Examples
Mood disorders	Major depression, bipolar disorder, dysthymic disorder
Anxiety disorders	Generalized anxiety disorder, obsessive-compulsive disorder, post-traumatic stress disorder, panic disorder, phobias
Psychosis in childhood	Schizophrenia, psychosis associated with epilepsy
Pervasive developmental disorders	Autism spectrum, Asperger's syndrome, Rett's syndrome, childhood disintegrative disorder, pervasive developmental disorder
Disruptive behavioral disorders	Oppositional defiant disorder, conduct disorder

Supplement to *Pediatric Emergency Medicine Reports*, September 2013: "Emergency Department Management of the Agitated Pediatric Patient." *Authors:* **Jennifer A. Newberry, MD, JD**, Clinical Instructor, Division of Emergency Medicine, Stanford University School of Medicine, Stanford, CA; and **N. Ewen Wang, MD**, Division of Emergency Medicine, Stanford University School of Medicine, Stanford, CA.
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Trauma Reports

PRACTICAL, EVIDENCE-BASED REVIEWS IN TRAUMA CARE

Volume 14, Number 5 **Sept/Oct 2013**

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Civilian Blast Injury

Blast injuries are commonly thought of as incidents that occur in other countries, not here in the United States. The majority of clinicians are not prepared to deal with the devastation of a civilian blast incident and the resulting injury patterns. The author reviews expected injury patterns, triage decisions, and current therapies.

— Ann M. Dietrich, MD, Editor

Introduction

With more than 54 local wars and armed conflicts in the first decade of this century[1] and notable terrorist activity in Afghanistan, Great Britain, India, Iraq, Pakistan, and Spain, it is easy to see why many American clinicians view terrorist blast injuries as an overseas issue. However, in the two decades from 1983 to 2002, more than 36,110 criminal bombing incidents occurred in the United States. During the decade between 1992 and 2002, more individuals were injured or killed by bombs on U.S. soil than all of the U.S. citizens killed during this same period in terrorist events overseas.[2]

High-profile terrorist bombings leading to mass casualties have occurred on American soil. In the past two decades, the 1995 bombing of the Murrah Federal Building in Oklahoma resulted in 759 injuries and 168 deaths;[3] the 2001 World Trade Center bombing in New York led to nearly 4,000 casualties and 3,000 deaths;[4,5] and most recently, in 2013, three people were killed and 264 injured when two improvised explosive devices (IEDs) were detonated at the Boston Marathon.[6]

Conventional explosive devices, either traditional or improvised, remain the terrorists' weapon of primary choice,[7] and blast injury is the common result. Despite these facts, we are relatively unprepared as emergency providers and systems to treat mass casualties as a result of blast injuries.[2,3,7,8] This review provides a primer on the physics, common injury patterns, and triage for blast injury.

Physics

Terrorist explosive devices are often weapons of convenience. These devices are categorized as high-energy or low-energy.[9] Examples of high-energy explosives include trinitrotoluene (TNT), plastic explosives such as C-4, and fertilizer-based explosives. The Oklahoma City bombing was the result of a high-energy explosive: the combination of nitrate fertilizer and fuel oil configured for maximum explosive effect as fuel-air explosive.[10]

Low-energy explosives include black powder and petroleum products. The World Trade Center explosions were the result of fuel-filled commercial aircraft. The Boston Marathon bombers probably used a combination of black powder, nails, and ball bearings packed into pressure cookers detonated with standard egg timers.[11]

High-energy explosives create a blast effect as the result of transient overpressurization. A brief period of high pressure is followed by a transient low pressure of longer duration, which can suck debris into the scene of injury. The

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Executive Summary

- Secondary to the multiple simultaneous mechanisms of injury, the blast patient is often more seriously injured than his multiple trauma cohort, and there is a "multidimensionality of injury" in these patients, largely because primary, secondary, tertiary, and quaternary blast effects may impact the victim simultaneously.
- Delayed injury, although uncommon, is really a delay of injury presentation. These injuries are typically primary blast injuries to the hollow organs, which do not manifest on initial roentgenogram, but should be anticipated based on clinical presentation and mitigated by careful observation and reassessment.
- The mid-facial skeleton contains large air-filled cavities and is susceptible to the spalling effects of the blast wave and implosion, resulting in "crushed egg shell" fractures of the sinus walls.
- Tympanic membrane (TM) rupture is common because of the relatively low pressure needed to perforate an eardrum.
- Research has shown that patients with skull fracture, burns greater than 10% of the body surface, and penetrating injuries to the head or torso were more likely to suffer a blast lung injury, and require early critical intervention at a level 1 trauma center.

Table 1. Classification of Blast Injuries[13,50]

Classification	Type	Mechanism	Typical Injuries
Primary	Blast wave		
	Implosion	Air-filled structures rupture from over-pressurization	Tympanic membrane rupture, blast lung, GI rupture
	Spalling	Explosive energy transfer in tissue interfaces of differing density	Lung, liver, brain contusion
	Inertia	Acceleration and deceleration forces lead to shearing injury	Mesenteric tears, axonal injury
Secondary	Blast wind	Bomb fragments, displaced foreign bodies	Penetrating or blunt multi-system injury
Tertiary	Blast wave and wind	Individual or structure thrown or crushed	Blunt or penetrating multi-system injury
Quaternary	By-products of explosion	Fireball and toxic agents	Burns and inhalation injuries

Classification of blast injury based on the mechanism of blast effect, based on the Zukerman classification developed during WWII[13] and modified from Plurad[50]

result is a shock wave that travels at supersonic speeds and a blast wind. The leading edge of this shock wave can injure tissue in its path (primary blast injury) by implosion, spalling, and inertia. The blast wind can move objects in its path, resulting in secondary blast injuries from flying debris and projectiles, or tertiary blast injuries from victims or objects that are hurled or structures that collapse. Quaternary blast injury results from the by-products of combustion such as burns and inhalation injuries.[8,9,12] (*See Table 1.*)

In the open air, blast energy rapidly dissipates with distance in inverse relation to the cube of the distance from the blast. For this reason, the distance from the blast is important in predicting injury and subsequent survival.[8,13] The Department of Homeland Security published a Bomb Stand-off Chart, which provides estimated safe distances from ground zero for a given TNT equivalent. (*See Table 2.*) The blast effect is magnified in water by an estimate of three times, and because water is less compressible than air, the wave travels for a greater distance.[8] Table 3 provides an estimate of the effect of blast over-pressurization.

Table 2. Bomb Threat Stand-off Chart

Threat Description Improvised Explosive Device (IED)	Explosives Capacity[1] (TNT Equivalent)	Building Evacuation Distance[2]	Outdoor Evacuation Distance[3]
Pipe bomb	5 lbs	70 ft	1200 ft
Suicide bomber	20 lbs	110 ft	1700 ft
Briefcase/suitcase	50 lbs	150 ft	1850 ft
Car	500 lbs	320 ft	1500 ft
SUV/van	1000 lbs	400 ft	2400 ft
Small moving van/ delivery truck	4000 lbs	640 ft	3800 ft
Moving van/water truck	10,000 lbs	860 ft	5100 ft
Semi-trailer	60,000 lbs	1570 ft	9300 ft

1. These capacities are based on the maximum weight of explosive material that could reasonably fit in a container of similar size.
2. Personnel in buildings are provided a high degree of protection from death or serious injury; however, glass breakage and building debris cause some injuries. Unstrengthened buildings can be expected to sustain damage that approximates five percent of their replacement cost.
3. If personnel cannot enter a building to seek shelter, they must evacuate to the minimum distance recommended by Outdoor Evacuation Distance. This distance is governed by the greater hazard of fragmentation distance, glass breakage, or threshold for ear drum rupture.

Source: Department of Homeland Security

Department of Homeland Security Bomb Threat Stand-off Chart. This chart provides an estimate of safe distance from the blast epicenter for a given charge of TNT. Note that 5 pounds of TNT is dangerous at up to a quarter mile. Further detail is available from dhs.gov.

Closed spaces significantly modify and amplify the blast effect. Walls and other hard surfaces reflect the wave and extend its duration, leading to a greater transfer of energy to susceptible organ systems.[13,14] Low-energy explosives can also have a primary blast effect, which is quickly mitigated by distance, and injuries are usually due to secondary and tertiary blast effects. In the Centennial Olympic Park bombing in 1996, advance warning and a low-energy explosive allowed for an orderly evacuation, which minimized casualties.[15]

Initial Assessment

The initial assessment and management of patients with blast injuries does not differ from the management of any multiple-injury trauma victim, and should follow standard Advanced Trauma Life Support (ATLS) principles.[16] There are, however, some differences in injury patterns and potential pitfalls specific to the organ systems involved, which will be discussed below. (*See Table 4.*) An understanding of the mechanism of injury is especially critical to understanding and managing the patient who has sustained a blast injury. The explosive agent used, the medium of wave propagation (air vs. water), the presence of flying debris and shrapnel, distance from the blast, open vs. closed environment, building collapse, and fire all provide different wounding mechanisms and morbidity and mortality rates.[8,12-14]

Additionally, because of the multiple simultaneous mechanisms of injury, the blast patient is often more seriously injured than his or her multiple trauma cohort, and there is a "multidimensionality of injury" in these patients, largely because primary, secondary, tertiary, and quaternary blast effects may impact the victim simultaneously.[8] This means time is of the essence and the opportunity for missed injury is magnified. Delayed injury, although uncommon, is really a delay of injury presentation. These injuries are typically primary blast injuries to the hollow organs, which do not manifest on initial roentgenogram, but should be anticipated based on clinical presentation and mitigated by careful observation and reassessment.[12] (*See Tables 4 and 5.*)

Maxillofacial Skeleton

The most common injuries are blunt and penetrating trauma as a result of secondary and tertiary blast effects. However, the blast wave can cause differential acceleration/

Table 3. Blast Pressure Effects

Pressure (kPa)	Effect
30	Shatters glass
100	50% chance of tympanic membrane rupture
200	100% tympanic membrane rupture, minimum pressure for lung injury
500	50% chance of lung injury
900	50% chance of death
2000	Lethal

Adapted from Boffard and MacFarlane.[31] The blast effects are governed by the size and type of charge, distance from the blast, and the medium of propagation. For example, 25 kg of TNT produces 1500 kPa (150 psi) of over pressure for 2 milliseconds at the epicenter and travels at up to 8,000 meters/second.[51] The resultant blast wind can be of hurricane proportions.[31]

Figure 1. Subdural Hematoma

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deceleration forces (inertia), which can lead to transverse shearing fractures of the mandible.[1] The mid-facial skeleton contains large air-filled cavities and is susceptible to the spalling effects of the blast wave and implosion. This may result in "crushed egg shell" fractures of the sinus walls.[1] Isolated maxillofacial injury rarely leads to death, and should be managed as appropriate within the context of the victim's other injuries.

Ear

Injury to the external ear is usually the result of secondary, tertiary, or quaternary blast injury.[17] The blunt and penetrating injuries require appropriate wound care, debridement, and repair. Injuries to the middle and inner ear are often the result of primary blast injury. Hearing loss, tinnitus, and ear pain are common and often temporary. Vertigo is unusual and should suggest the possibility of concomitant head injury.[17,18] Tympanic membrane (TM) rupture is common because of the relatively low pressure needed to perforate an eardrum. (*See Table 3.*) Ossicular injury is uncommon and suggests significant trauma.[17] The physical examination should include a hearing evaluation.[17] Most TM ruptures will heal spontaneously; however, referral to an otolaryngologist is appropriate.[17,18]

The common wisdom has been that a TM injury is a harbinger of potential occult primary blast injury. Recent evidence, however, suggests that while TM injury is common, its presence or absence does not include or exclude other injuries.[9,17,19,20] In the survivors of the 2005 London bombings, TM rupture as a biomarker of concealed primary blast injuries had a sensitivity and specificity of 50%, and a low positive predictive value. External evidence of injury may be a more appropriate triage tool.[21]

Eye

Primary blast injury to the eye can lead to globe disruption, retinal injury, and hyphema.[14] However, penetrating injury from flying debris and shrapnel as the result of secondary blast injury is the more common cause of eye injury.[22,23] One major receiving center from the 2004 Madrid bombing reported an incidence of ocular injury in 16% of their patients with minor injuries and 15% of their patients with critical injuries.[24] Ocular injury was the second most common injury (26%) in the injured survivors of the 2001 World Trade Center bombing.[4] Symptoms include loss of visual acuity, eye pain, and foreign body sensation. Appropriate wound care should be provided for external injuries. Emergency management for injury to the globe (evaluation, irrigation, topical antibiotics, and patching) should be followed with specialty evaluation and management for complex injuries.

Brain

Brain injury is a common cause of death in blast injury. One hundred sixty-seven people died as a result of the Oklahoma City bombing in 1995. Head injury was the second most common cause of death (14%), with multiple trauma the leading cause (73%). Fifty-two percent of critically injured patients treated at the closest hospital during the 2004 Madrid bombings sustained head injuries.[24] Blunt and penetrating injury can result from primary, secondary, and tertiary blast effects.[23] Blunt injury can range from concussion to diffuse axonal injury;

Table 4. Overview of Explosion-related Injuries

System	Injury or Condition
Auditory system	Tympanic membrane rupture, ossicular disruption, cochlear damage, foreign body
Cardiovascular	Cardiac contusion, myocardial infarction from air embolism, shock, vasovagal hypotension, peripheral vascular injury, air embolism-induced injury
Extremity injuries	Traumatic amputation, fractures, crush injuries, compartment syndrome, burns, cuts, lacerations, acute arterial occlusion, air embolism-induced injury
Gastrointestinal	Bowel perforation, hemorrhage, ruptured liver or spleen, sepsis, mesenteric ischemia from air embolism
Neurologic system	Concussion, closed and open brain injury, stroke, spinal cord injury, air embolism-induced injury
Ocular injury	Perforated globe, foreign body, air embolism, fracture
Renal injury	Renal contusion, laceration, acute renal failure due to rhabdomyolysis, hypotension, and hypovolemia
Respiratory system	Blast lung, hemothorax, pneumothorax, pulmonary contusion and hemorrhage, A-V fistulas (source of air embolism), airway epithelial damage, aspiration pneumonitis, sepsis

Adapted from Centers for Disease Control and Prevention. Explosions and blast injuries: A primer for clinicians. Available at http://www.bt.cdc.gov/masscasualties/explosions.asp.

however, subarachnoid hemorrhage and subdural hemorrhages occur most frequently in fatalities.[23] (*See Figure 1.*)

Recent studies using diffusion tensor imaging suggest there is a component of axonal injury in military personnel with blast-related mild traumatic brain injury.[25] However, it is not clear if isolated primary blast injury at a distance leads to mild traumatic brain injury in the absence of a direct blow.[25-27]

Evaluation and management of head injury should follow the basics of emergency management as outlined in ATLS.[16] Because patients sustaining injury as a result of a blast are more critically injured than their multisystem trauma cohort,[8] avoidance of hypoxia and hypotension are essential, and the early involvement of neurosurgical specialists in the initial management is appropriate.

Chest

Blast lung injury (BLI) implies proximity to the blast and is a common cause of mortality at the scene of bomb blasts.[9] (*See Figure 2.*) It is also a frequent cause of morbidity for survivors.[28,29] Avidan et al analyzed a two-decade experience with BLI and found a 71% incidence of BLI in blast victims admitted to the intensive care unit (ICU) of their Israeli trauma center.[28]

The blast wave causes a combination of implosion, inertia, and spalling, which can lead to bronchoalveolar disruption, pulmonary contusion, and arterial air emboli.[9,21,28,29] Survivors generally present with hypoxemia and respiratory distress.[29] Bloody sputum and evidence of barotrauma (pneumothorax) are not uncommon, even though radiologic evidence may lag by 12 to 24 hours.[12] Secondary injury may also lag presentation by several hours. Eckert et al advocate observation for at least 18 hours and selected bronchoscopy to evaluate quaternary airway injury based on their experience at a combat support hospital in Iraq.[30]

Penetrating injuries as a result of flying debris and projectiles, blunt injuries as a result of falls and crush injuries, and inhalation injury and burns should be managed emergently with ATLS principles.[16] Definitive management should be provided with advanced pulmonary and surgical critical care.

Abdomen

Abdominal blast injury can be both blunt and penetrating. In the 2004 Madrid bombings, 12 of the 243 patients (5%) and 10 of 27 (37%) critically injured patients treated at the nearest hospital sustained abdominal injuries.[24] The

Table 5. Clinical Signs and Symptoms of Explosion-related Injuries

System	Injury or Condition
Auditory system	• Blood oozing from the mouth, nose, or ears* • Eardrum hyperemia, hemorrhage, or rupture* • Deafness* • Tinnitus* • Earache*
Cardiovascular	• Tachycardia • Fall of mean arterial blood pressure
Gastrointestinal	• Nausea* • Abdominal tenderness* • Abdominal rigidity*
Neurologic system	• Vertigo • Retrograde amnesia
Ocular injury	• Eye irritation** • Hyphema** • Distorted pupil** • Decreased vision** • Blindness** • Funduscopic findings of retinal artery air embolism**
Respiratory system	• Cyanosis* • Ecchymosis or petechiae in hypopharynx* • Cough (often dry)* • Tachypnea (often preceded by a short period of apnea)* • Dyspnea* • Hemoptysis* • Rales or moist crepitation in lung fields* • Chest pain*

* Most common findings

** Common findings

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experience in the Oklahoma City bombing in 1995 was quite different. Four of a total of 759 patients sustained life-threatening abdominal injuries: one bowel transection, two splenic lacerations, one kidney laceration, and one liver laceration.[10] (*See Figures 3 and 4.*) Both of these bombings involved high-energy explosives, but the mechanisms were quite different. In Madrid, suicide bombers used military explosives and shrapnel to detonate in a closed space at close proximity.[24] As expected, the injuries included a combination of blunt and penetrating trauma, and were relatively common in seriously injured survivors. In Oklahoma City, the explosive was an improvised fuel-oil bomb,[10] and the abdominal injuries were blunt and uncommon.

Primary blast injury is relatively uncommon in survivors. Implosion can rupture air-filled bowel wall, spalling forces can disrupt viscera at tissue interfaces (i.e., lung/liver, or spleen), and shearing forces associated with inertia typically affect mesentery.[8,12,31] These effects are significantly increased under water.[32] The emergency management of these patients does not change from the management of the multiple-injury patient, except as mentioned above.

Extremities

Soft-tissue injury accounts for the majority of trauma in civilian blast events.[33] In the Oklahoma City bombing, soft-tissue trauma was the most common injury, followed by fractures and dislocations.[3] Primary blast injury has been shown to cause fractures and amputations;[34] however, the most common cause of extremity injury is related to secondary and tertiary blast effects. Injuries can range from simple soft-tissue trauma to amputations. In the military experience in Afghanistan and Iraq, extremity fractures accounted for 82% of combat injuries, and the majority of the fractures were open.[35] In the civilian bombings, the incidence of extremity injury is much lower. In the Madrid bombings of 2004, only 17% of survivors sustained extremity fractures, and only one patient sustained a traumatic lower extremity amputation.[24] Civilian traumatic amputations as a result of blast injury, however, are often lethal because the blast energy required to amputate a limb is usually lethal to other organ systems.[12,36,37] This was illustrated in the 2005 London bombing in which six of seven patients with traumatic amputations of the upper extremity died at the scene.[36] Almogy et al reported a three-year experience with 15 suicide bombings in Israel.[21] In their series, 63 of 74 (85%) patients with traumatic amputations died at the scene. The Boston bombings of 2013 resulted in several amputations, but few fatalities. These blasts were low-energy and in the open air.

Evaluation should proceed by standard ATLS protocols.[16] Special consideration should be given to

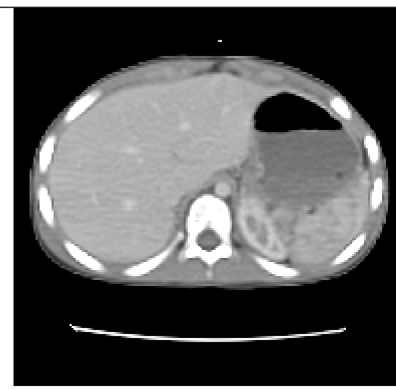
Figure 2. Blast Lung

Reprinted with permission from: Wolf YG. Vascular trauma in high-velocity gunshot wounds and shrapnel-blast injuries in Israel. *Surg Clin North Am* 2002;82:237-244.

Figure 3. Liver Laceration

Reprinted with permission from: Wang NE, Blankenburg RL. Pediatric abdominal trauma. *Trauma Rep* 2007;8:7.

Figure 4. Spleen Fracture



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the surrounding environment and the potential for contamination and secondary infection.[23] One unique aspect of terrorist bombing is the potential for biologic foreign bodies and the risk of blood-borne disease these biologic fragments may carry.[38] Tourniquets are often discouraged in civilian practice, but can be life-saving in traumatic amputations. Surgical completion of the amputation is often a difficult decision, but should be made based on the potential viability and projected functionality of the injured extremity.[35,36]

Pregnancy

Blast injury in pregnancy is uncommon. Mallonee et al reported three of the 167 deaths (1.8%) in the Oklahoma City bombing were pregnant women.[10] Marti et al reported one maternal fetal death from massive hemoperitoneum in 36 patients (3%) treated at their institution following the 2004 Madrid bombings.[39] The fetus may be cushioned by amniotic fluid; however, the placenta would be subject to implosion and shearing forces,[14,40] and the mother would be subject to all of the other mechanisms and injuries described above. Initial assessment and management should follow standard ATLS protocols.[16] Postmortem cesarean section for blast injury is rare, but should be considered for the viable fetus in the case of sudden maternal death.[41]

Burns

The primary blast is associated with a brief fireball at detonation.[23] These burns are often lethal[14] because they herald a close proximity to the blast[12] and are associated with the other primary blast injuries discussed above. Burns and inhalation injuries make up the majority of quaternary blast injuries.[14] Among the 790 injured survivors in the World Trade Center attack in 2001, 386 (49%) were treated for smoke inhalation.[4] The combination of burns and inhalation injuries with the constellation of other injuries sustained by these patients can make them difficult to manage.[12]

Triage

Mass casualty events as the result of a blast are dramatic events and muster an immediate and often less than coordinated response. Despite this, the vast majority of survivors are walking wounded (*see Table 6*), and the over-triage rate is typically 50-90%.[43] Additionally, the receiving

Table 6. Civilian Blast Event Severity of Injury

Event and Year (Reference)	Casualties	Dead	Treated		Explosive
			Hospitalized	Released	
Oklahoma City 1995[10]	759	168 (22%)	83 (14%)	508 (86%)	Fertilizer and fuel oil
Atlanta Olympics 1996[15]	111	1 (<1%)	24 (22%)	87 (78%)	Pipe bomb and shrapnel
World Trade Center 2001[4,5]	3922	2819 (72%)	181 (17%)	810 (73%)	Jet fuel
Madrid 2004[24]	2000	191 (10%)	91 (29%)	221 (71%)	Military explosives and shrapnel
London 2005[42]	775	56 (7%)	27 (14%)	167 (86%)	Military explosives and shrapnel
Boston Marathon 2013[6]	267	3 (1%)	20 (8%)	244 (92%)	Black powder and shrapnel

Casualties for the World Trade Center attack and the Madrid bombings are estimates based on the available literature. Disposition of casualties at the hospital for Madrid and London are based on single institutional experience. The differences in mortality are related to the explosive agent used (high-energy vs. low-energy), open vs. closed space, and high-rise vs. ground level.

facility for the largest influx of patients is often the nearest facility and not necessarily the facility most capable of handling the injuries.[42] Triage at the scene should be performed by experienced personnel, and patient distribution allocated based on available resources and patient need.[42] Triage at the receiving facility should also be done by experienced clinicians and with the understanding that most of the patients seen will not be critically injured.[42,43]

Although injury to the tympanic membrane is the most common blast injury and has been heralded as a harbinger of more serious blast injury, the correlation doesn't hold. It is a poor diagnostic tool for triage. Serious injury is usually obvious. The mechanism is a more important predictor for occult injury, and history becomes an important indicator of blast exposure. The combination of mechanism and evidence of external injury can often help to identify those patients in need of critical resources. (*See Table 7.*) Almogy et al described a retrospective analysis of 15 suicide bomb attacks treated over a three-year period (1994-1997) in Israeli hospitals.[21] These authors found that patients with skull fracture, burns greater than 10% of the body surface, and penetrating injuries to the head or torso were more likely to suffer a blast lung injury, and would require early critical intervention at a level 1 trauma center.

The initial management of these patients should follow damage-control principles to allow for the greatest good to the largest number of victims.[13,42,43] Resource allocation for definitive management will typically mirror those resources used in trauma; however, the seriously injured are a magnitude of several times more severely injured than their typical multi-system counterparts. Experience from military conflicts can help to guide their management.[44] In the Oklahoma City bombing, general surgery, ophthalmology, orthopedics, neurosurgery, and vascular surgery were utilized (in decreasing order) for patients needing operative intervention.[3]

Although it is counter-intuitive, blood usage is not out of proportion to the injury and does not exceed local resources.[42,44] Predictive models from military[44] and civilian experience[13,39,42,43,45-49] may help with disaster planning and allocation of resources.

There are a number of excellent courses to help emergency providers better understand the basics of mass casualty and its management. These include: The National Disaster Life Support™ (NDLS™) course from the National Disaster Life Support Foundation (formerly a collaboration with the AMA); Collaborative Disaster Planning Processes from the American College of Emergency Physicians (ACEP) and the Federal Emergency Management Agency (FEMA); and Disaster Management and Emergency Preparedness (DMEP) from the American College of Surgeons Committee on Trauma Disaster and Mass Casualty Management Committee.

Table 7. Considerations for Injury Severity

Blast Force
- Explosive energy (high-energy vs. low-energy)
- Distance from ground zero
- Energy dissipates by the cube of the distance from the blast

Environment
- Building collapse (high-rise vs. low-rise)
- Confined space vs. open air explosions
- Urban vs. rural (less population dense) settings

Projectiles
- Environmental debris vs. intentional shrapnel

By-products of explosion
- Fire
- Smoke
- Toxins

Anatomic Markers of Severe Injury
- Traumatic amputation
- Blast lung injury
- Severe head injury
- Torso trauma
- Multidimensional injury

Adapted from Ciraulo DL, Frykberg ER. The surgeon and acts of civilian terrorism: Blast injuries. *J Am Coll Surg* 2006;203:942-950.

Conclusion

Blast injury provides a unique challenge in management. There are often multiple survivors, and many of them have minor injuries. This can lead to a dramatic surge in patient inflow to the facility closest to the event. The seriously injured patient can be subjected to a multitude of mechanisms, including primary, secondary, tertiary, and quaternary blast. They are often more severely injured and more complex in their presentation than their multiple trauma counterparts. The emergency provider should understand and anticipate the basics of blast injury to provide optimum care. Continuing education in mass casualty and disaster management is strongly encouraged.

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CNE/CME Objectives

Upon completing this program, the participants will be able to:

- discuss conditions that should increase suspicion for traumatic injuries;
- describe the various modalities used to identify different traumatic conditions;
- cite methods of quickly stabilizing and managing patients; and
- identify possible complications that may occur with traumatic injuries.

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CME/CNE Questions

1. Blast-injured patients are more seriously injured than their multiple-trauma counterparts because of:

    A. primary blast injury
    B. secondary blast injury
    C. tertiary blast injury
    D. quaternary blast injury
    E. all of the above

2. Tympanic membrane rupture is an accurate predictor of occult primary blast injury.

    A. true
    B. false

3. Which of the following is (are) the best predictor(s) of occult primary blast injury?

    A. tympanic membrane rupture
    B. external injury
    C. mechanism of injury
    D. B and C
    E. none of the above

4. The most common cause of blast injury is:

    A. primary blast injury
    B. secondary blast injury
    C. tertiary blast injury
    D. quaternary blast injury
    E. quintenary blast injury

5. Explosions are dramatic events, but over-triage is uncommon.
   A. true
   B. false

6. Blast-injured patients at the scene of a mass casualty event should be routinely transferred to a trauma center.
   A. true
   B. false

7. In a blast injury mass casualty situation, management should include:
   A. scene transfer to a trauma center
   B. damage-control principles
   C. hospitalization and observation of all victims evaluated in the ED to exclude occult blast injury
   D. all of the above
   E. none of the above

8. Traumatic amputation is often:
   A. a lethal injury in civilian experience
   B. survivable in the recent military experience
   C. associated with other severe blast injuries
   D. best managed with damage-control principles
   E. all of the above

9. Which of the following is true?
   A. Quaternary blast injury is uncommon in survivors.
   B. Infectious risk does not differ from other multiple-injury patients.
   C. Burn patients are managed identically to non-blast counterparts.
   D. All of the above are true.
   E. None of the above are true.

10. Which of the following is true of primary blast lung injury?
    A. It is usually evidenced on admission with clinical findings.
    B. It uniformly occurs with blast over pressures of 300 psi.
    C. It may be associated with quaternary blast effect on the airways.
    D. A and C are true.
    E. None of the above are true.

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