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PEDIATRIC

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The recent terrorist attacks on New York City and Washington have left all emergency medicine physicians feeling a little closer to our colleagues who were confronted with the unthinkable. Our hearts are heavy with sorrow, strength, and support for those individuals directly affected by the tragedy. From these events, each emergency physician realizes the importance of preparing ourselves, families, and departments for future terrorist attacks. The children we serve are a vulnerable group with special needs. Future terrorist attacks or other disasters may result in a patient with significant muscle injuries and subsequent distant metabolic effects.

Crush injuries and the metabolic complications of these injuries are seen infrequently in pediatric emergency medicine. However, incidents like the 1995 Oklahoma City bombing demonstrated how this type of injury may affect children. The term "crush injury" encompasses several major syndromes: rhabdomyolysis, the crush syndrome, acute atraumatic compartment syndrome, traumatic compartment syndrome, and traumatic asphyxia.

The first four crushing injuries comprise a spectrum of muscle trauma associated with extremity injuries, while the last reflects direct pressure trauma to the thorax. The prolonged pressure that produces crush syndrome in an extremity muscle is lethal if applied to the torso. The duration of pressure that causes traumatic asphyxia is much shorter than that which causes crush syndrome. This article reviews in detail the spectrum of crush injuries in pediatrics.

— The Editor

Current Approaches to Pediatric Crush Injury

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History

The association of a crushing injury with kidney damage was first identified in the German military literature around World War I and was based on experience from the earthquake of Messina in 1909.[1] The consequences of a crushing injury and its distant metabolic effects were called "crush syndrome." The crush syndrome consists of acidosis and acute renal failure followed by hypovolemic shock, hyperkalemia, and ultimately, death. Dr. Bywaters' later research led to the demonstration that the nephrotoxic agent released by damaged muscle was myoglobin.[2] He went on to demonstrate that myoglobin is nephrotoxic in acid but not in alkaline urine, and described the initial treatment that is still used today. In 1975, Mubarak and Owen suggested that rhabdomyolysis, compartment syndrome, and crush syndrome are interrelated and that, if any one of these exists, the other two should be considered.[3]

This type of injury usually is associated with multicasualty disaster operations such as terrorist bombings, earthquakes, building collapse, mine accidents, and train accidents. The incidence of

crushing injuries in these disasters varies widely. Earthquakes are estimated to have a 3-20% incidence of crushing injuries.[4-6] A collapse of a multistory building may cause crush injury in up to 40% of the extricated survivors.[7] The bombing in Oklahoma City resulted in a large number of crush injury casualties. Crush injuries also may be seen in isolated individuals who work under cars or are trapped in collapsed excavations or ditches. In disasters and excavations, extrication often is prolonged when specialized equipment and personnel are required. Such catastrophes may present the emergency physician with patients who have similar injuries and who present simultaneously.

A wringer-type injury also may result in this syndrome. This injury was much more common when wringer-type washing machines were prevalent. Since most washers today use a spin cycle to partially dry the clothing, the wringer injury is now seen in occupational accidents. Common sources include industrial gears, rollers, punches, or presses.[8]

Initial Findings in Crush Injuries

Following extrication from the rubble or the machinery, the patient may feel no pain and have few physical complaints.[9] Most patients are conscious and lucid.[10] Emotional complaints dominate the history, as might be expected from a patient who has been buried.[11] After extrication the patient may appear normal and be categorized as having only a minimal injury. This lack of complaints frequently is misleading to the rescuers and may be lethal to the patient.[12]

On examination, the patient may have flaccid paralysis of the injured extremity. Underlying the skin, the muscles often are pale and do not contract to pinch or electrical stimulation. They do not feel elastic and may resemble cooked meat. The muscle tissue may bleed quite readily.

The skin often is relatively undamaged, but may have bullae and discoloration. There may be a patchy lack of sensation.[9] Multiple skin lacerations and tears are more common when a direct pressure trauma occurs — such as when a car runs over an extremity.

There often is little edema of the affected limb on initial examination. After a few hours, a striking edema may develop. Pulses usually are present, even in the presence of edema, but may be difficult to palpate.

Hypovolemia

The model that Dr. Bywaters used for his research conclusively demonstrated that crushed muscle could absorb large amounts of edema fluid. With extensive injury, this fluid sequestration will result in hypovolemic shock and renal failure from acute tubular necrosis. It is rarely appreciated how important hypovolemia is in production of renal failure with the crush syndrome.[1,13,14] In Dr. Bywaters' rabbit model, up to one-third of the body water may be trapped in the injured muscle. This corresponds to human clinical experience. Better and associates estimated that the amount of fluid that is "third-spaced" in extensive crush syndrome may be equal to the total extracellular volume in a 75-kg adult.[15] Hypovolemic shock and acute tubular necrosis develop if the volume of fluid lost is not replaced within six hours.[16] Since this disease often is seen as a part of disaster medicine, ED physicians must be aware of the possible need for massive fluid replacement.

Rhabdomyolysis

Causes. Rhabdomyolysis is an integral part of the crush syndrome but there are multiple other "nontraumatic" etiologies.[17] In the crushing injury, rhabdomyolysis results from both continued direct pressure on muscles and direct crushing injuries of the muscle. As a consequence of this pressure, the muscle is destroyed and releases myoglobin, potassium, and phosphorus into the tissues.[18]

In a continuous crushing injury, muscle breakdown products are not released into the circulation until the trapped extremities are freed and decompressed. There are many anecdotal reports of patients who are freed from debris, only to deteriorate rapidly after being rescued. This "rescue death" may be explained by

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sudden hypovolemia, as described above, combined with acidosis and the release of trapped myoglobin, potassium, and phosphorus as circulation is restored.

Rhabdomyolysis may occur after severe exercise, particularly in untrained individuals. The most common occurrence is in young, unconditioned military recruits who train in hot and humid climates. Training in heat with insufficient water leads to an environment ideal for disruption of muscle fibers, with high muscle temperatures, hypovolemia from sweating, shunting of circulation from muscle to skin for cooling, and degradation of enzymes by heat. The subsequent hypovolemia, coupled with the release of myoglobin from damaged muscle, leads to a decreased glomerular filtration rate, metabolic acidosis, an acidic urine pH, and subsequent myoglobin-induced renal failure. Similar situations may occur with amphetamines and other drugs of abuse.[19-20] These patients may be agitated, violent, and require physical restraint.

Muscle tissue is quite sensitive to direct, continually applied pressure. Regional ischemia may occlude the circulation and cause ischemic muscle cell death. After the ischemic cell is reperfused, restoration of blood flow causes an infusion of calcium, generation of free radicals, production of mitochondrial superoxide and hydrogen peroxide, and release of the cytotoxic free radicals and proteases into the area surrounding the cell.[21]

A variety of disparate clinical situations are associated with rhabdomyolysis that do not involve trauma, increased activity, or direct pressure. These have included amphetamine and cocaine use, neuroleptic malignant syndrome, hypothermia, chronic and acute ethanol intake, electrolyte abnormalities, several infectious organisms, heatstroke, and hypothermia.[22]

Myoglobin. Myoglobin is found in both cardiac and skeletal muscle and released when the sarcoplasm is destroyed. Myoglobin is an oxygen-binding, single peptide molecule containing an iron-porphyrin (heme) group and a folded peptide (globin) group. Myoglobin is related to hemoglobin and the single heme group in myoglobin is the same as that found in hemoglobin. Myoglobin is about one-fourth of the molecular weight of hemoglobin, which has four such heme/globin moieties. The molecular weight of myoglobin is 17,500 daltons, and the molecule is a folded 153-amino acid polypeptide chain.[23]

Myoglobinuria is an abnormal pathologic state in which an excess of myoglobin is found in the urine. Myoglobin is normally excreted in minute amounts in the urine. The term "myoglobinuria" has been used interchangeably with rhabdomyolysis in some literature, which underscores the muscle necrosis that causes the excess release of myoglobin.

Normal serum myoglobin concentration is 85 nanograms/mL or less. If more than 200 g of muscle are injured, serum myoglobin levels will rise to greater than 1.5 mg/dL, and detectable myoglobinuria will result.[24] Myoglobin rapidly is cleared from the urine, so the period of myoglobinuria is short and may be missed.

Myoglobin has a toxic effect on tubular epithelium if the urine pH is less than 5.4 (acidic urine). The myoglobin will dissociate into globin and ferrihemate. The ferrihemate moiety is toxic to the renal tubular cells. It does not appear to be nephrotoxic in alkaline urine.[13, 25,26] Myoglobinuria is estimated to cause between 5% and 25% of all cases of acute renal failure.

Detection of Myoglobinuria. Myoglobinuria should be suspected whenever the patient's urine turns dark red or brown. A urinalysis may reveal a muddy brown color. Microscopic examination may show a few red blood cells (RBCs) and pigmented urinary casts.[27]

Myoglobin is detectable with the same dipstick tests that detect hemoglobin. The ortho toluidine tests detect the presence of the heme moiety and do not differentiate between hemoglobin and myoglobin.

Laboratory testing for myoglobin in both urine and serum should be obtained. A fresh specimen of urine should be checked for the presence of RBCs. If no (or only a few) RBCs are found, then myoglobinuria should be suspected. Intravascular hemolysis also can cause hemoglobinuria without RBCs, but the serum will be pinkish in color and react for hemoglobin with the benzidine test.

Unfortunately, a negative dipstick for myoglobin does not always rule out rhabdomyolysis. As noted above, myoglobin is freely filtered in the glomerulus and rapidly cleared from the blood stream. If the urine isn't checked a few hours after the injury, and if the patient has received sufficient fluids, then the urine will be negative by dipstick or another similar test.

Creatinine phosphokinases. Creatinine phosphokinase (CPK) is released from damaged muscle. Elevation of this enzyme is a very sensitive indicator of damaged muscle from any cause. Since CPK is not an enzyme found in RBCs, it is not released in hemoglobinuria.

Serum CPK levels are more likely to be elevated than serum myoglobin levels because of the slower plasma clearance of CPK. The serum half-life of CPK is about 1.5 days.[28] A normal CPK level is less than 200, while 10,000 or higher is frequently found in rhabdomyolysis. Elevation of the CPK is the most sensitive diagnostic finding for rhabdomyolysis. A fivefold or greater increase in serum CPK in a patient without cardiac or brain injury can be used as a diagnostic criterion for rhabdomyolysis. No other disease or medical condition will produce this magnitude of CPK elevation.

The CPK elevation will parallel the amount of muscle damage. With appropriate treatment, there is not a significant correlation between CPK levels and morbidity and mortality.

Uric Acid. Hyperuricemia is found in most patients with crushing injuries. It results from release of muscle adenosine nucleotides and subsequent hepatic conversion to uric acid. The part that uric acid plays in renal failure is not well understood. High uric acid concentrations in the presence of urinary acidosis can cause nephropathy. The tremendous volume of uric acid produced in crush syndrome and rhabdomyolysis may help precipitate renal failure by direct action on the kidney in an acid environment.

The hyperuricemia that accompanies muscle destruction may respond to saline diuresis if the urine output is adequate. Xanthine oxidase inhibitors will not prevent elevation of uric acid levels when it is due to ongoing muscle damage.

Potassium. The skeletal muscles contain the largest amount of potassium of any organ in the body.[29] Necrosis of about 150 grams of muscle will acutely elevate the serum potassium.[28] Rhabdomyolysis is associated with severe hyperkalemia caused by release of potassium from crushed muscles and later from

renal failure. Since the kidney is not able to easily excrete high loads of potassium, early hyperkalemia may be seen despite entirely normal renal function.[30]

Hyperkalemia is a dangerous complication of rhabdomyolysis, particularly if anuria or oliguria is present. With dehydration, the "third-spacing" of fluid, and the release of potassium from injured muscles, the rise in potassium may cause cardiac arrest within an hour of extrication.[29] Given the amount of potassium contained in muscle cells, it is surprising that more patients do not develop complications from hyperkalemia.

The hypokalemic effects of insulin and glucose may be impaired in rhabdomyolysis. This means that the usual emergency therapy of insulin and glucose may not be appropriate in the presence of rhabdomyolysis.

Calcium. Hypocalcemia is a frequent early finding in the course of rhabdomyolysis. Calcium is absorbed into hypoxic tissues or tissues reoxygenated after hypoxia. Hyperphosphatemia may contribute to this decrease in serum calcium by causing decrease in $1,25(OH)_2$ vitamin D, skeletal resistance to parathyroid hormone, and exacerbation of calcium deposition. This calcium uptake in damaged tissue is not blocked by calcium channel-blocking agents.[31]

Hypocalcemia in rhabdomyolysis usually is asymptomatic and self-correcting. Cardiotoxic and symptomatic patients will require treatment.

There usually is little necessity to treat the patient acutely for hypocalcemia in the emergency department.

During the recovery phase of acute renal failure, some patients may liberate calcium from damaged muscles and become hypercalcemic. Development of severe hypercalcemia during the early recovery phase may be due to elevated parathyroid hormone levels and increased vitamin D-3 synthesis in the recovering kidney. Unlike early hypocalcemia, hypercalcemia frequently is symptomatic. Treatment of this hypercalcemia can include volume expansion, diuresis with furosemide, and possibly dialysis.

Hyperphosphatemia. Hyperphosphatemia also is seen frequently and is thought to be caused by leakage of phosphorus from injured muscle. Phosphate levels usually remain lower than 7 mg/dL. If the phosphate level rises above this, then the use of phosphate binding agents is appropriate. This modest hyperphosphatemia may contribute to early hypocalcemia.

Hypophosphatemia is a later finding in rhabdomyolysis. This should not be treated unless the phosphate level falls below 1 mg /dL.

Acute Renal Failure Due to Rhabdomyolysis

Early references to renal failure induced by trauma all were associated with crush injuries.[32] In 1941 Bywaters and Beall described severe degenerative changes in the kidneys of victims of crush syndrome.[33] In their series, acute renal failure was uniformly fatal. Even with modern techniques, mortality of acute renal failure may approach 60%.[32] Fortunately, not all patients with rhabdomyolysis will develop renal failure.

Renal failure in these cases often is multifactorial. The development of acute renal failure in crush victims initially relates to the underlying hypovolemic shock. Hypovolemia may be quite severe and precipitate acute tubular necrosis.

Superimposed on this setting of decreased renal perfusion is the presence of potentially toxic circulating myoglobin. Recent studies provide strong evidence to the direct cytotoxic effect of the hemeproteins on the renal tubular cell.[21] Vasoactive peptides may result in renal vasoconstriction and enhance the toxic effects of myoglobin.

These same studies have provided insight into the role of the reactive oxygen molecule in this renal failure. Hydrogen peroxide and superoxide are formed and catalytically react with free iron to form the hydroxyl radical (OH). In rhabdomyolysis, very large quantities of the porphyrin (heme) ring are present in the proximal tubular cell. Normally, the renal tubular cell is able to metabolize the porphyrin into ferritin (the storage form of free iron). Since iron is a transitional metal, it is able to generate oxygen free radicals and lead to damage of the renal cell.[34-35]

The actual free radical that causes the damage is not yet known. The site of the damage also is not known. Given the complexities of the interactions between free-iron generation and its cytotoxicity, it is quite possible that several interlinking factors may act in concert to cause cell death.

Glutathione has been found to be protective, and there is accumulating evidence that renal glutathione is depleted by large amounts of circulating myoglobin.[36] This depletion may be prevented by the administration of glutathione.

Finally, myohemoglobin combines with the renal tubular secretory protein (Tamm-Horsfall protein) to form a cast. Acid pH of the tubular fluid greatly favors the formation of these protein casts.[37] Cast formation obstructs the tubule and increases the intratubular pressure. This leads to an acute reduction of glomerular filtration. Alkalization of the urine prevents this cast formation.

Many patients will maintain a relatively normal urine output for the first 24-36 hours after the insult. Over the next 2-3 days, the patient will develop progressive oliguria. As the output drops, the blood urea nitrogen, potassium, and the creatinine will rise. Appropriate rehydration and supportive measures may prevent this oliguria.

Some patients may develop nonoliguric renal failure. This has a much better prognosis. The patient with non-oliguric renal failure is unable to concentrate the urine, so the urine sodium will remain greater than 40 mEq/L, the urine osmolality will be nearly the same as the serum, and the urine to plasma creatinine ratio will be less than 10.

Therapeutic Considerations in Crushing Injuries

During Extrication. Victims of crush injury have been saved even after more than 24 hours of entrapment.[38] Initial treatment should be geared toward prevention of hypovolemia and acute renal failure. In these patients, massive fluid replacement during and shortly after extrication likely will be required. Large quantities of intravenous fluids should be available at the site of a disaster where buildings have collapsed.

During extrication of the patient, an intravenous (IV) line of normal saline should be started as soon as possible; 50 mEq/L of sodium bicarbonate should be added to this line. This IV solution should be infused at a rate of 1.5 L/h and the rate adjusted for the age and weight of the child. In a recent Israeli study of seven victims trapped in a building, this pre-extrication

volume loading appeared to prevent acute renal failure in all patients.[13] Mannitol may provide temporizing protection against renal failure, but IV fluids will be required.[39]

Crush injury may be an indication for field amputation. This most frequently occurs when the building remnants are immobile, sufficient to destroy the limb, and the patient is otherwise healthy. It also may be indicted when the danger to life from moving the building remnants is greater than the possible loss of limb.[40]

There are no studies that address the pregnant patient with a crush injury. These patients should be treated as noted above.[41] An obstetrician should be consulted to ensure that the unborn child gets the optimum possible care.

Specific treatment of the compartment syndrome component is discussed below.

Following Extrication. Close observation of every patient who has a possible crush syndrome is essential. Emergency medical service (EMS) providers and rescuers should be warned that these patients might initially look well and then crash suddenly. IV lines should be started in situ if at all possible.[42] Close monitoring of blood pressure and pulse should be maintained for at least 12 hours after extrication in all patients. Continuous cardiac monitoring is essential.

Massive amounts of fluid may be used in these patients. All patients should have a Foley catheter and input and output should be monitored quite carefully. Patients who have risk factors for congestive heart failure may benefit from Swan-Ganz pulmonary artery monitoring if available. It is easy to see that administration of large amounts of fluid to prevent renal failure also can lead to massive tissue swelling and compartment syndrome. Monitoring of compartment pressures as described below is important.

Laboratory studies should include CPK, urinalysis with micro and dipstick examination, complete blood count, electrolytes, blood urea nitrogen (BUN), creatinine, glucose, calcium, phosphorus, uric acid, albumin, and serum protein. Clotting studies including a prothrombin time and partial thromboplastin time also should be drawn. The CPK should be repeated every 12 hours. Electrolytes, bicarbonate, BUN, creatinine, glucose, calcium, and phosphorus should be repeated every 6-12 hours depending on the clinical severity of the patient. Urine and serum may be sent for myoglobin, but clinical judgments should not depend on this procedure.

Indications for emergent dialysis include persistent hyperkalemia and resistant metabolic acidosis. If the patient develops fluid overload, pulmonary edema and congestive heart failure may be relieved by acute dialysis. Dialysis will not remove myoglobin from the circulation because of the size of the molecule. Similarly, plasma exchange is ineffective for clearing myoglobin. Continuous arteriovenous hemofiltration and continuous venous hemofiltration have been shown to remove myoglobin in animal models.

Compartment Syndrome

In 1881, Richard Von Volkmann described paralysis and subsequent contracture resulting from the application of tightly wrapped splinting bandages used for fractures. He suggested that the bandages interrupted the arterial blood supply and that subsequent ischemia led to the paralysis and contractures. Later, authors reported that the contractures developed without the tight bandages. Still later, authors reported that surgical decompression of the limb would prevent the sequelae of paralysis and contractures. With better anatomic definition of the compartments of the extremities and the development of devices to measure the pressure within these compartments, we have come to realize that an untreated compartment syndrome causes Volkmann's ischemic contracture. An arterial injury is not required. Despite a century of investigation, this syndrome remains elusive in diagnosis.

As discussed above, skeletal muscle appears to be exquisitely sensitive to pressure.[43] A compartment syndrome is caused by a higher than normal pressure within a closed fascial space (muscle compartment). Other names have been used, including Volkmann's ischemia, tension ischemia, march gangrene, anterior tibial syndrome, calf hypertension, impending ischemic contracture, and exercise myopathy.[44-46]

A compartment syndrome occurs when there is either a decrease in the compartment size or an increase in the contents of the compartment. An increase or swelling in the compartment contents may be caused by bleeding, IV or intraosseous infiltration, or by posttraumatic or ischemic swelling. The compartment size may be decreased by crush injury, a compression bandage, or constrictive device, such as a cast. The pathophysiology becomes simple: too much stuff in too little space. If the pressure rises too high, then the microcirculation within the compartment will fail.

When the perfusion ceases, ischemia occurs. It is followed by tissue edema and more swelling. This feedback loop will continue to increase whenever the compartment pressures exceed the capillary pressures. The tissue ischemia is followed by necrosis within the compartment. Death of the muscle may result in myoglobinuria, acidosis, and renal complications. Muscle necrosis may lead to amputation of the extremity, sepsis, or death.

Full recovery is probable when the treatment is started within four hours of the onset of symptoms. If treatment is delayed more than eight hours, then chances of recovery are markedly diminished. The higher the compartment pressures, the shorter the time until irreversible damage is sustained.

Causes

Most compartment syndromes result from trauma to an extremity, with the forearm and lower leg being the most commonly involved extremities.[47-48] Compartment syndrome also may develop in the extraocular eye muscles, gluteal muscles, thigh, and lumbar paraspinous muscles. Inciting events may range from direct pressure for an extended period of time to excessive exercise of a muscle group, surgical procedures including closure of fascial defects, major vascular surgery, bleeding disorders, snakebites, burns, lightning strikes, electrical injuries, infiltration of infusions into deep veins, intravenous drug abuse, burns, pneumatic tourniquets, weight lifting, and post-ischemic swelling.[48-52] *(See Table 1.)* Indeed, any of the causes of rhabdomyolysis discussed above can cause the development of a compartment syndrome.

Drugs and Coma. Although compartment syndrome and subsequent crush syndrome most often is seen after trauma, it also can occur after a drug overdose or even intoxication with

alcohol.[53-57] This mechanism may surpass or rival limb trauma as a major cause of compartment syndrome. The victim falls into a coma and part of his or her body is compressed by the body's weight. The unconscious patient lies on a hard surface or on top of a limb, exerting prolonged pressure on the limb.

An alternative mechanism for compartment syndrome associated with stimulant drugs is found in the agitated and violent patient who requires either physical or chemical restraint (or both). Methamphetamine often is implicated in this form of rhabdomyolysis and the victims are younger, less sick, and have a shorter hospital stay.[58] Since the IV drug user often has other problems or an altered sensorium, the diagnosis of a compartment syndrome frequently is delayed.

Anesthetic agents also may predispose to a compartment syndrome. In these situations, the normal protective mechanism of pain is circumvented. Coma induced for anesthesia in prolonged surgical procedures, particularly in the "knee-tuck" or lithotomy position, can iatrogenically cause compartment syndrome.[59-62] The prolonged pressure, subsequent swelling, and decreased perfusion of the area will damage dependent tissues.[63] This mechanism is quite similar to that found in crush syndrome.

Prolonged Pressure. Indeed, the compression mechanism is not necessarily limited to coma from drug abuse or systemic intoxication. A similar presentation has been reported in carbon monoxide poisoning, and after being trapped in confined spaces.[64] The patient is unable to roll or shift weight and relieve pressure on the dependent areas.

Pneumatic anti-shock garments that are inflated improperly or left in place for extended periods of time have produced compartment syndromes.[65] Tight casts and compressive dressings are common causes of compartment syndrome and have been since Volkmann's time. The compression effects of the pressure garment or constrictive cast/dressing cause the crush injury or compartment syndrome.

Local Effects of Injections. Local effects of drugs also may cause crush syndrome. Intra-arterial injection of barbiturates and intravenous diazepam have been known to cause vascular damage. Iatrogenic hypertonic saline during Bier block regional anesthesia also has been implicated as a cause.

Other drug-related causes include bleeding due to anticoagulation, injection of medications or fluids through an improperly placed intraosseous line, IV use of drugs, and infiltrated IV sites.

Fractures. Fractures of the tibia and fibula, usually in the middle or distal third of the leg, can cause compartment syndrome of the leg. Upper extremity compartment syndromes may accompany supracondylar fractures of the elbow. Other fractures that can be associated with this injury include fractures of the femur and tarsus. Tight casts and dressings also have been implicated.

Diagnosis

The onset of a compartment syndrome usually occurs about 6-8 hours after the injury occurs. Left unchecked, the cycle of edema and ischemia will result in muscle infarction, nerve injury, and permanent loss of function in the extremity.

Remember that a compartment syndrome can occur in an open wound if the skin laceration is not sufficient to decompress the edema or hemorrhage. Even if the wound does not involve the compartmental fascia, compartment syndrome still is possible.

Table 1. Etiologies of Compartment Syndrome: Traumatic and Atraumatic

DECREASED COMPARTMENT SIZE
• Crush syndrome
• Closure of fascial defects
• Tight dressings or casts
• External pressure
• Anesthesia (iatrogenic external pressure)
• Coma
INCREASED COMPARTMENT CONTENTS
• Bleeding
• Edema
• Post ischemic swelling
• Exercise
• Trauma
• Burns
• Orthopedic surgery or trauma
• Venous obstruction
LOCAL (DIRECT VASOTOXICITY)
• Intra-arterial drugs
• Vasotoxic drugs

Physical Examination. The early diagnosis of compartment syndrome is made on clinical grounds. On examination, the patient may have a tense and tender extremity. This finding may be hard to elicit if there is isolated involvement of the deep posterior compartment in the calf. In the patient with a crush syndrome, the limb may be flaccid with widespread sensory loss in the overlying skin. Of course, falling debris may cause extensive external damage.

Pain. The initial symptom often is pain that seems to be out of proportion to the initial injury. This pain may increase at the time, when the examiner and patient expected that it would decrease. A classic finding is pain that increases after reduction of a fracture. An extremity should be more comfortable after the fracture has been reduced. Ischemic muscle is very painful.

The patient also may complain of pain when the affected muscles are compressed or stretched. The muscle will be painful after almost any injury, so this finding is not a great help.

Pulselessness and Pallor. Although a major feature of the pathology of a compartment syndrome is ischemia, only rarely will the pressures be sufficient to occlude arterial flow. This means that capillary refill and distal pulses do not change. Pallor, poor capillary refill, and pulselessness are not early signs of compartment syndrome. If present, they are signs of severe involvement.

Paresthesia. Nerves that traverse the area of decreased flow will not work properly. Paresthesias will develop in the distribution of those nerves. This may be an early complaint, but if allowed to persist, may not be reversible. Unfortunately, paresthesias also are seen after contusions that affect the nerve and after vascular damage.

The most reliable physical finding is a sensory deficit.[66] Sen-

Table 2. Common Locations of Compartment Syndrome[99]

The most common locations are the fascial compartments of the leg and forearm.

EXTREMITY	COMPARTMENT INVOLVED	SENSATION DECREASED	WEAKNESS	PASSIVE MOVEMENT	PAIN
Hand	Intraosseous	None	Finger adduction and abduction	Finger adduction and abduction	Between metacarpals on dorsum of hand
Forearm	Volar (anterior)	Palmar aspect of fingers (median and ulnar nerve)	Wrist and finger flexion	Wrist and finger extension	Volar forearm
	Lateral	Radial nerve	Wrist and finger flexion	Wrist and finger extension	Lateral forearm
	Dorsal (posterior)	None (deep branch of the radial nerve)	Wrist and finger extension	Wrist and finger flexion	Dorsal forearm
Leg	Deep posterior	Sole of foot (tibial nerve)	Toe flexion	Toe extension	Deep calf — between the Achilles tendon and medial malleolus
	Superficial posterior	Posterior lateral cutaneous (sural nerve)	Plantar flexion	Dorsiflexion of the foot	Calf
	Lateral	Top of foot (superficial peroneal nerve)	Eversion of foot	Inversion of foot	Lateral lower leg
	Anterior	First web space (deep peroneal nerve)	Extension of toes	Flexion of toes	Lateral anterior tibia

sation may be assessed by sharp/dull discrimination, two-point discrimination, and vibratory sensation testing. Of these, loss of vibratory sensation is the earliest and most sensitive test available.[67] Two-point discrimination is normally 3.5-5.0 mm. This discrimination will fail more quickly than sharp/dull discrimination. In the patient with crush syndrome, sensation may be impaired by direct pressure on the skin. *(See Table 2.)*

Poor Function. An ischemic muscle is unable to contract effectively. Unfortunately, this also is a very nonspecific finding, and a fracture or contusion may damage the muscle and cause splinting with any attempt to elicit motion. In the patient with crush syndrome, this is an expected finding and may not correlate with the extent of injury.

Paralysis due to crush injury may mimic spinal trauma. Since spinal trauma also occurs in this setting, it is important to differentiate the two. Neurologic examination may show a normal anal sphincter tone and urinary bladder function. It is unlikely that, with these two findings, there will be a cord injury.[68]

The most common site of compartment syndrome is the lower leg.[69] The anterior compartment is the most frequent site in the leg, followed by the lateral compartment and the deep posterior compartment. Compartment syndrome in the deep posterior compartment is associated with a second compartment syndrome about one-third of the time.

In the upper extremity, the most common site of compartment syndrome is the volar forearm with median nerve involvement followed (if untreated) by the classic Volkmann's contracture.

Compartment Pressure Measurement

The standard of care in management of a suspected compartment syndrome should be a measurement of the compartment pressure. The compartment pressure should be measured whenever there is a suspicion of compartment pressure or if the patient has an injury commonly associated with compartment syndrome and is unable to speak (if the patient is too young, unresponsive, intoxicated, unreliable, or uncooperative). Pressures start to rise early in the course of the injury since tissue fluids are not compressible. A normal compartment pressure will effectively rule out a compartment syndrome. Unfortunately, even a mild elevation should be re-evaluated frequently, since the pressure rapidly rises in the non-distensible fascial compartment.

A compartment pressure of 10 mmHg or less is normal. If the patient has a consistent pressure in excess of 30 mmHg, and there are any findings compatible with a compartment syndrome, then the patient needs prompt therapy to decrease the compartment pressure. If the pressure is above 40 mmHg, emergency treatment is needed. A gray area exists between 10 and 30 mmHg. In this window, the patient may be admitted and closely followed with repeated pressure measurements. If the pressure continues to rise, treatment is indicated.

The difference between the mean arterial pressure and the

compartment pressure may be more important than the absolute compartment pressure. This change in pressure (MAP-compartment pressure) represents the perfusion pressure of the capillary. The lowest change in pressure that keeps tissue viable is about 30 mmHg in normal tissue and 40 mmHg in traumatized tissues.[70]

Laboratory Diagnosis

Laboratory testing is not particularly helpful in the diagnosis of a compartment syndrome. It is more helpful in suggesting an appropriate differential diagnosis. Although CPK measurements may be elevated with a compartment syndrome, they also will be elevated with muscle trauma from any cause. Likewise, myoglobinuria may be present from muscle damage.

Differential Diagnosis

Conditions that may be mistaken for compartment syndrome include an isolated arterial lesion, isolated nerve damage, cellulitis, osteomyelitis, tenosynovitis, synovitis, and thrombophlebitis. Arterial occlusion or nerve damage may coexist with compartment syndrome.

Therapeutic Considerations

As mentioned earlier, an increased pressure in a closed fascial compartment causes a compartment syndrome. You can either open the compartment, or decrease the size of the contents. Adjunctive therapy should decrease the distant complications of the compartment syndrome.

First Things First. The very first therapy that should be done is to remove all restrictive dressings. The most common constrictive dressing is a plaster or fiberglass cast. This should be bivalved, even if the reduction was difficult. If the pain is promptly relieved, then further therapy may not be needed. Air splints and automatic blood pressure cuffs should be deflated or removed. Air splints have caused compartment syndrome when applied in cold weather or at low altitude and then allowed to remain when the patient is moved to a warm area or a higher altitude.

Alkaline Diuresis. A vigorous alkaline diuresis is, perhaps, the best way to protect the kidney from the effects of rhabdomyolysis.[7]

Fluids. Prevention of the complications of rhabdomyolysis and crush injury dictates early and vigorous replacement of the volume losses. An IV line should be started at the earliest chance in the extrication process. Normal saline is infused during the extrication. Following the extrication, the urine output and systemic hemodynamics may be measured. Correction of volume deficit may require as much as 12 L per day. This should be given until the myoglobin disappears from the urine and may require as long as three days.

Bicarbonate. The urine should be alkaline with a pH higher than 6.5. Add 50 mEq of bicarbonate to each liter of saline infused. This usually will keep the urine alkalotic and prevent myoglobin nephropathy.

Theoretically, bicarbonate may precipitate metastatic calcification within traumatized muscles.

Mannitol. The diuresis can be further forced by addition of 20% mannitol 0.25 g-0.5 g/kg/dose to a total of 120 grams per day. Mannitol also protects against muscular cellular swelling. Some evidence exists that mannitol protects the kidney by direct intrarenal action as well as secondarily by expanding the extra- cellular fluid, improving the myocardial contractility, and stimulating the release of atrial natriuretic factor.[71] Mannitol should not be used if there is no urine flow. Mannitol may have some direct protection by decompression of the muscle compartments and subsequent decrease of tamponade.[72]

Furosemide. The use of loop diuretics may not be necessary for prevention of acute renal failure in traumatic rhabdomyolysis. They have a theoretical disadvantage of causing urinary acidification.

Acetazolamide. The use of acetazolamide to correct metabolic alkalosis may be indicated when the arterial pH is above 7.45. Acetazolamide also will help to alkalinize the urine and decrease the serum potassium.

Fasciotomy. Fasciotomy is a surgical operation to open the compartment and release the pressure. If the compartment pressure is not relieved by other means, then timely fasciotomy will decompress the compartment and allow reperfusion of the muscle. Levels of greater than 40 mmHg from the estimated arterial perfusion pressure or 30 mmHg from the estimated systemic diastolic pressure are indications for fasciotomy.[73]

The fascia is slit the entire length of the fascial compartment. All compartments of the affected extremity usually are opened at one time to prevent a recurrent increase in another compartment. The skin often is left open and covered with moist dressings for 3-5 days. A delayed primary closure is then attempted. A grafting technique may be needed at this time. Some surgeons advocate skin closure at the same time as the original procedure.[74]

The current literature recommends a fasciotomy with delayed closure of the wounds.[75] It is interesting to note that there is a controversy about the utility of fasciotomy in compartment syndrome associated with crush syndrome. Fasciotomy has a high complication rate because it transforms a closed lesion into an open wound. The open wound, together with necrotic muscle in the wound, may provoke life-threatening infection.

Based on a small group of patients who were successfully treated without fasciotomy, some believe that, in crush injury, fasciotomy should be reserved for patients who have imminent gangrene.[9,29] These researchers also believe that if a fasciotomy is required, then a radical muscle debridement should be performed. Although they feel that muscle should be radically debrided, the skin should not be removed, if at all possible. In their opinion, a closed crush injury is not benefited by fasciotomy because necrotic muscle is not salvaged by decompression.

Hemodialysis

In patients who get post-traumatic acute renal failure (ARF), vigorous daily hemodialysis usually will tide the patient over the anuric period, which may last several weeks.[71]

When hemodialysis is not available, peritoneal dialysis or continuous arteriovenous hemofiltration may salvage the patient. Both of these methods are less effective in elimination of nitrogenous waste products and phosphates. Both require large volumes of sterile replacement fluid.

Other Treatments. If the clinician tries to decrease the contents of the compartment by using pressure dressings, the risk of raising the pressure within the compartment is increased.

Table 3. Reported Causes of Traumatic Asphyxia[79]

- Direct compression or crush injury
- Deceleration injuries
- Asthma
- Seizures
- Paroxysmal coughing
- Paroxysmal vomiting
- Extended Valsalva maneuver
- Barotrauma
- Blast injury
- Strangulation
- Acute superior vena cava syndrome
- Acute jugular vein obstruction

Likewise elevation will both decrease the contents of the compartment and decrease the pressure head available at the capillary level. This may make the muscles more ischemic and thus cause more swelling rather than less.

Ice and elevation have a place in the prevention of compartment syndrome by decreasing the swelling after an injury. They aren't appropriate for treatment after a compartment syndrome develops.

Hyperbaric oxygen has been advocated, with animal studies, for treatment of crush injuries and compartment syndrome.[76] These animal studies seem promising. There are few human studies on which to base any conclusions, but it seems to be effective.[77] What remains to be seen and studied is the utility in mass casualty situations as found in earthquake or building collapse.

Traumatic Asphyxia

The effects of heavy weight crushing a victim have been known for centuries; the first autopsy descriptions were of victims of mobs in Paris in 1837.[78] Dr. Ollivier described the syndrome of subconjunctival and subcutaneous hemorrhages, bluish discoloration of the skin of the face and neck, and edema. He coined the term "Masque Ecchymotique" to describe these effects. Other terms for this condition include "Ollivier's syndrome," traumatic cyanosis, compression cyanosis, Perthe's symptom complex, and cervicofacial static cyanosis. Most subsequent case reports describe injuries of direct compression. Similar manifestations can be seen due to an increase in intrathoracic pressure from other causes.[79]

Traumatic asphyxia is a rare condition. In a series of more than 100,000 hospitalizations, there were only seven cases of traumatic asphyxia identified.

The differential diagnosis of traumatic asphyxia must include basilar fractures of the skull, periorbital hematomas, subconjunctival hemorrhage, and hemotympanum from other causes. *(See Table 3.)*

The exact pathology of this syndrome is not yet known. One hypothesis is that the crushing force drives blood out of the right atrium. This blood moves through the innominate and jugular veins and toward the head. The resulting rapid dilation of the capillaries and venules will cause rupture of these small vessels and subsequent formation of petechiae.

Another hypothesis requires a "fear response." This response includes deep inspiration, glottic closure, and constriction of the abdominal muscles.[80] The closed epiglottis increases the intrathoracic pressure during the compression.[81] Traumatic asphyxia has been induced experimentally, but the induction requires that the endotracheal tube is occluded before classic findings occur.[82]

The magnitude of the clinical manifestations of traumatic asphyxia will reflect the duration of the compression and the magnitude of the force applied. One researcher found that no animal would survive pressure equal to more than five times its body weight for longer than 10 minutes. The mechanism of death was thought to be chest wall constriction, hypoxia, and apnea.[83]

Dermatologic Manifestations. The very striking physical characteristics of traumatic asphyxia include multiple small petechiae extending over the face, neck, and the upper chest. If a hat is worn, the hatband often will be free of petechiae. Stasis of the blood in the small vessels will lead to the purplish discoloration of the skin about the eyelids, nose, and lips. The tympanic membrane can be stained blue from the blood engorgement. Rarely do petechiae extend below the nipple line. These petechiae blanch on pressure. They will increase in intensity over the first few hours and then gradually fade. Pronounced facial edema often is present.

Ocular Injuries. The orbital contents also are affected. The sudden elevation in venous pressure is thought to cause the ocular injuries.[84] The high intrathoracic pressure is transmitted to the retinal vessels, leading to leakage and arteriolar spasm. The patient may have periorbital edema, proptosis, diplopia, and ecchymosis together with subconjunctival hemorrhages.[85] Loss of vision has been reported due to retinal edema.[86] This vision loss usually is transient and is uncommon. Permanent impairment usually is associated with retinal hemorrhage and involvement of the macula or optic disk.

Pulmonary Injuries. Most patients with traumatic asphyxia will have a significant pulmonary contusion. Other thoracic injuries that have been associated include multiple fractured ribs, flail chest, and tension pneumothorax.[87-88] Pneumonia, empyema, pleural effusion, and pulmonary edema can further complicate the course. The chest x-ray may be normal on initial examination despite quite serious injury.

Cardiac Abnormalities. Cardiac abnormalities are rare in patients who have traumatic asphyxia. Myocardial contusion has been reported, but is distinctly unusual.[89-90]

Neurovascular Injuries. Neurologic symptoms frequently are associated with traumatic asphyxia. About 85% of survivors will have transient loss of consciousness or prolonged confusion following the episode.[91] Many of the patients will be agitated, restless, and disoriented. Most of these effects will resolve within 24 hours. It is thought that minor intracranial hemorrhage or cerebral edema causes these transient manifestations.[92]

Skeletal Injuries. Although the "pure" syndrome of traumatic asphyxia has no skeletal injuries, the compression of a heavy weight carries a high probability of such injuries. Associated thoracic injuries are found in 58% of patients, fractures in 34% of patients, and abdominal injuries in only 11% of patients.[93]

Diagnosis. This syndrome should be easy to recognize. Petechiae that develop after cough or vomiting do not have the cyanosis, edema, or complications of traumatic asphyxia. Supe-

rior vena cava syndrome will give a similar clinical picture, but the history and mechanism of injury will be quite different. Basilar skull fractures may cause subconjunctival hemorrhages, epistaxis, and periorbital ecchymosis. Skull fractures are usually rare in the setting that leads to a traumatic asphyxia. History and mechanism of injury, again, should be able to rule out basilar skull fractures. If there is any doubt, a computed tomography scan of the skull will provide a definitive diagnosis.

Treatment. Treatment of traumatic asphyxia is completely supportive. The petechiae should resolve within 2-3 weeks. The head of the bed can be elevated 30° and the patient placed on high flow oxygen. Cardiac monitoring is essential. Associated injuries may require that the patient be intubated and placed on a ventilator. Following basic resuscitation, evaluation of the pulmonary injury is needed. The patient's abdomen and extremities should be assessed.

The prognosis is either dismal or excellent, with little middle ground.[94-95] All survivors in a large series were extricated within 15 minutes of entrapment.[96] Those patients who survive for more than an hour will uniformly do well, with 90% or higher survival rates without sequelae or complications.[97] Late deaths usually are due to an infection or associated injuries.[98]

Summary

Although infrequently seen in field emergency medicine, the crushing injuries are associated with major complications, mortality, and morbidity. Three major syndromes discussed include the crush syndrome, traumatic asphyxia, and compartment syndrome.

These injuries are found in disasters such as bombings, earthquakes, building collapse, mine accidents, and train accidents. In disasters and excavations, extrication often is prolonged, resulting in increased injuries. Such catastrophes may present the emergency practitioner with multiple similar patients simultaneously. Immediate airway and fluid resuscitation will provide the most salvage of patients in these disasters. The emergency practitioner should be aware of the rapid development of shock and renal failure from these injuries. Management of compartment syndrome and crush syndrome are similar and overlap. Management of traumatic asphyxia is supportive.

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Physician CME Questions

51. What is the initial concern in a patient who has just been extricated following a crush injury?
    A. Compartment syndrome
    B. Muscle necrosis
    C. Hypovolemia
    D. Hypocalcium
    E. Acute renal failure

52. Which of the following is *not* a nontraumatic cause of rhabdomyolysis?
    A. Cocaine
    B. Barbiturates
    C. Colchicine
    D. Acetaminophen
    E. Inhalational anesthetic

53. Which of the following is correct regarding myoglobinuria?
    A. The patient's urine may be brown.
    B. Myoglobin has a toxic effect if urine pH is less than 5.4.
    C. Myoglobin may dissociate into globin and ferrihemate.
    D. Ferrihemate is toxic to renal tubular cells.
    E. All of the above.

54. A negative dipstick for myoglobin excludes rhabdomyolysis.
    A. True
    B. False

55. Which of the following CPK values is suspicious for rhabdomyolysis?
    A. 200
    B. 400
    C. 10,000
    D. 6000
    E. 800

56. Which of the following is a frequent early finding in rhabdomyolysis?
    A. Hypocalcemia
    B. Hypernatremia
    C. Hypokalemia
    D. Hyponatremia
    E. Hypouricemia

57. Which of the following may be sites where a compartment syndrome may develop?
    A. Extraocular eye muscles
    B. Gluteal muscles
    C. Forearm
    D. Lumbar paraspinous muscles
    E. All of the above

58. Which of the following is true regarding a compartment syndrome?
    A. It usually is found immediately after injury.
    B. Pulselessness and pallor are found early in the disease.
    C. Early diagnosis is based on clinical findings.
    D. The most common site for compartment syndrome is the thigh.
    E. The first therapy should consist of mannitol.

59. Which of the following compartment pressures requires careful monitoring and repeat compartment pressures but not emergency treatment?
    A. 65 mmHg
    B. 70 mmHg
    C. 20 mmHg
    D. 42 mmHg
    E. 50 mmHg

60. Which of the following physical findings is associated acutely with traumatic asphyxia?
    A. Liver hematomes
    B. Splenic rupture
    C. Myocardial infarction
    D. Blue tympanic membrane
    E. Empyema

CME Objectives

The CME objectives for *Pediatric Emergency Medicine Reports* are to help physicians:

a.) Quickly recognize or increase index of suspicion for specific conditions;

b.) Understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;

c.) Be educated about how to correctly perform necessary diagnostic tests;

d.) Take a meaningful patient history that will reveal the most important details about the particular medical problem discussed;

e.) Apply state-of-the-art therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;

f.) Understand the differential diagnosis of the entity discussed;

g.) Understand both likely and rare complications that may occur; and

h.) Provide patients with any necessary discharge instructions.

In Future Issues: Inborn Metabolic Disorders

PEDIATRIC
Emergency Medicine Reports
The Practical Journal of Pediatric Emergency Medicine

Crush Injuries

Common Locations of Compartment Syndrome

The most common locations are the fascial compartments of the leg and forearm.

EXTREMITY	COMPARTMENT INVOLVED	SENSATION DECREASED	WEAKNESS	PASSIVE MOVEMENT	PAIN
Hand	Intraosseous	None	Finger adduction and abduction	Finger adduction and abduction	Between metacarpals on dorsum of hand
Forearm	Volar (anterior)	Palmar aspect of fingers (median and ulnar nerve)	Wrist and finger flexion	Wrist and finger extension	Volar forearm
	Lateral	Radial nerve	Wrist and finger flexion	Wrist and finger extension	Lateral forearm
	Dorsal (posterior)	None (deep branch of the radial nerve)	Wrist and finger extension	Wrist and finger flexion	Dorsal forearm
Leg	Deep posterior	Sole of foot (tibial nerve)	Toe flexion	Toe extension	Deep calf — between the Achilles tendon and medial malleolus
	Superficial posterior	Posterior lateral cutaneous (sural nerve)	Plantar flexion	Dorsiflexion of the foot	Calf
	Lateral	Top of foot (superficial peroneal nerve)	Eversion of foot	Inversion of foot	Lateral lower leg
	Anterior	First web space (deep peroneal nerve)	Extension of toes	Flexion of toes	Lateral anterior tibia

Reported Causes of Traumatic Asphyxia

- Direct compression or crush injury
- Deceleration injuries
- Asthma
- Seizures
- Paroxysmal coughing
- Paroxysmal vomiting
- Extended Valsalva maneuver
- Barotrauma
- Blast injury
- Strangulation
- Acute superior vena cava syndrome
- Acute jugular vein obstruction

Etiologies of Compartment Syndrome: Traumatic and Atraumatic

DECREASED COMPARTMENT SIZE

- Crush syndrome
- Closure of fascial defects
- Tight dressings or casts
- External pressure
- Anesthesia (iatrogenic external pressure)
- Coma

INCREASED COMPARTMENT CONTENTS

- Bleeding
- Edema
- Post ischemic swelling
- Exercise
- Trauma
- Burns
- Orthopedic surgery or trauma
- Venous obstruction

LOCAL (DIRECT VASOTOXICITY)

- Intra-arterial drugs
- Vasotoxic drugs

Supplement to *Pediatric Emergency Medicine Reports*, December 2001: "Current Approaches to Pediatric Crush Injury."
Author: Charles Stewart, MD, Emergency Medicine Physician, Colorado Springs, CO.

BIOTERRORISM WATCH

Preparing for and responding to biological, chemical and nuclear disasters

Flu or anthrax? First inhalational cases yield clues for clinicians to make the critical call

Use case history, blood work, X-rays, rapid tests

There is a postal worker in your emergency department (ED) with flulike symptoms.

That once insignificant observation about occupation and illness now triggers a detailed algorithm created by the Centers for Disease Control and Prevention (CDC) in Atlanta. **(See algorithm, p. 2.)** Is it flu or inhalational anthrax? Whether a realistic question or not, it is what many of your incoming patients may be asking — particularly if another wave of anthrax scares coincides with a nasty influenza season. Many of the initial symptoms are similar, but investigators dealing with the first inhalational anthrax cases have gleaned out key indicators that will help clinicians make the call.

"It is important to take a careful history from the [patients] when they present," says **Julie Gerberding**, MD, acting deputy director of CDC's National Center for Infectious Diseases. "If the [patients are] mail handlers in a professional environment — where they're dealing with large amounts of mail that is not their own — then the index of suspicion should be raised and more testing should be done to be sure there aren't additional clues to suggest that it is not a common viral infection."

Using the first 10 cases of inhalational anthrax as a baseline patient profile, the CDC reports that the median age of the patients was 56 years (range: 43-73 years), and seven were men.[1]

The incubation period from the time of exposure to onset of symptoms when known (seven cases) was seven days (range: five to 11 days).

The initial illness in the patients included fever (nine) and / or sweats / chills (six). Severe fatigue or malaise was present in eight, and minimal or nonproductive cough in nine. One had blood-tinged sputum. Eight patients reported chest discomfort or pleuritic pain. Abdominal pain or nausea or vomiting occurred in five, and five reported chest heaviness. Other symptoms included shortness of breath (seven), headache (five), myalgias (four), and sore throat (two). The mortality rate was 40% for the 10 patients, much lower than historical data indicated. Indeed, one of the critical reasons to recognize inhalational anthrax early is that it is far more treatable than originally thought.

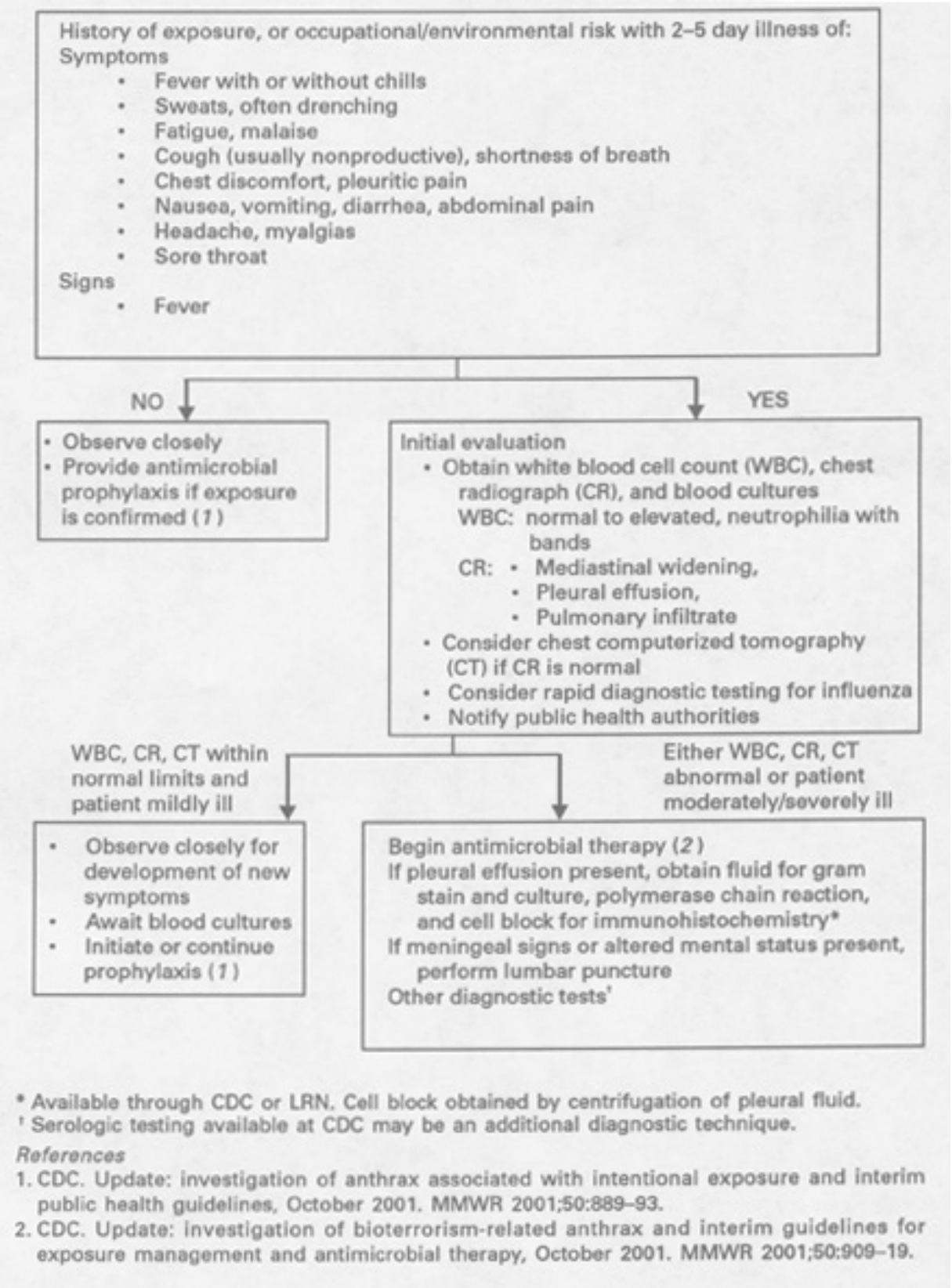
The CDC gathered comparative data on the symptoms and signs of anthrax and influenza, finding, for example, that only 20% of the anthrax patients reported sore throat.[2] Flu sufferers report a sore throat in 64% to 84% of cases. Likewise, 80% of the anthrax cases reported symptoms of nausea and vomiting. That symptom is reported in only 12% of flu cases. Shortness of breath appears to be another key distinguishing symptom, affecting 80% of the anthrax patients but seen in only 6% of flu patients.

"One of the other clues that we are noticing is that the patients with inhalation anthrax actually do not have nasal congestion or a runny nose,"

(Continued on page 3)

This supplement was prepared by Gary Evans, editor of *Hospital Infection Control*. Telephone: (706) 742-2515. E-mail: gary.evans@ahcpub.com.

Clinical Evaluation of People with Possible Inhalational Anthrax



Source: Centers for Disease Control and Prevention. Update: Investigation of bioterrorism-related anthrax and interim guidelines for clinical evaluation of persons with possible anthrax. *MMWR* 2001; 50:945.

Gerberding says. "They don't have the symptoms of an upper-respiratory tract infection. They have a more systemic chest presentation, and that may be another distinguishing characteristic."

Another finding on initial blood work is that none of the inhalational anthrax patients had a low white blood cell count (WBC) or lymphocytosis when initially evaluated. Given that, CDC officials note that future suspect cases with low WBC counts may have viral infections such as influenza. Chest X-rays were abnormal in all patients, but in two an initial reading was interpreted as within normal limits. Mediastinal changes including mediastinal widening were noted in all eight patients who had CT scans. Mediastinal widening may be subtle, and careful review of the chest radiograph by a radiologist may be necessary, the CDC advises.

Complementing the CDC's effort, are the observations of the few clinicians who have actually seen inhalational anthrax cases come into their hospital systems. Two inhalational anthrax cases, both of which survived, were admitted to the Inova Healthcare System in Fairfax, VA (near Washington, DC).

"Clinically, I think the history of the people who presented here is useful," says **Allan J. Morrison Jr.**, MD, MSc, FACP, health care epidemiologist for the Inova system. "They stutter-stepped toward their pulmonary symptoms. That had some mild symptoms and then they were sort of 'meta-stable.' They were not relentlessly progressing. Then they progressed with symptoms more aggressively. Whereas with influenza — in our experience — once you start to get sick, it just keeps on progressing with very high fevers, chills, muscle aches, and pains. As a consequence, we feel there should be a good way to differentiate the two."

Since anthrax is a realistic concern in the Washington, DC, area, what about the aforementioned scenario of symptomatic postal workers in the ED?

"We would take a very aggressive history, not only of occupation but physically where they have been," Morrison says. "If they are symptomatic and have been in or work around a 'hot zone' — a location from which anthrax has either been cultured environmentally or patients have come from there — we will err on the side of being very aggressive about working up anthrax. By that I mean chest X-rays, chemistry profile, [etc.]"

In addition, the hospital system pushed early flu vaccination programs for staff and the surrounding community. "We want to move toward herd immunity," he says. "We are also working with our local hospitals to make sure that they have access to the rapid influenza tests. So for diagnosis — for obvious reasons — it is very helpful to make that distinction early."

One such rapid test is ZstatFlu (ZymeTX Inc., Oklahoma City), which the company claims can yield a diagnosis of influenza A or B some 20 minutes after a throat swab. The test detects neuraminidase, an influenza viral enzyme. However, Gerberding cautions clinicians not to rely solely on such tests. Rather, they should use the results of tests in combination with the patient history and clinical presentation, she says.

"So it is a constellation of history, clinical findings, and laboratory tests," she says. "Hopefully, when we get these all together, we'll be able to at least reduce the anxiety among some people and help clinicians diagnose those patients who really do require the antibiotic treatment. What we don't want to have happen is for everybody coming in with the flu to get an antibiotic because that undermines a whole other set of public health issues relating to antimicrobial resistance and proper management of influenza."

Even the vaccinated can still have flu

Complicating the issue is the fact that the flu vaccine efficacy can vary annually, but is usually 70% to 90% protective, says **Keiji Fukuda**, MD, a medical epidemiologist in the CDC influenza branch. Thus, depending on how well the vaccine matches the circulating strain, a certain portion of flu patients will tell clinicians they have been immunized. But in addition to vaccine breakthrough infections, there is a plethora of other viral and respiratory pathogens that will be creating similar symptoms, he says. In a somewhat sobering reminder — given that at this writing, the total anthrax cases remained in the double digits — Fukuda notes that a typical flu season will send 114,000 people to the hospital and 20,000 to their graves.

"There has been an awful lot of attention on the [anthrax] cases, but the bottom line is that there have been few cases, and these cases generally have occurred in a limited number of communities within a limited number of groups," he says. "And so the epidemiologic message is that anthrax really has not been diagnosed in most parts of the country, whereas we expect to see millions and millions of flu cases all over the place."

If facilities are faced with an onslaught of patients with respiratory illness there are several measures they can take, he notes. Those include:

- Reduce or eliminate elective surgery.
- Relax staff-to-patient ratios within the limits of your licensing agency.
- Emphasize immunizing staff so more staff are available.
- Identify ways to bring in extra staff to help out with the patients.
- Set up walk-in flu clinics to triage the patients.

Reference

1. Centers for Disease Control and Prevention. Update: Investigation of bioterrorism-related anthrax and interim guidelines for clinical evaluation of persons with possible anthrax. *MMWR* 2001; 50:941-948.

2. Centers for Disease Control and Prevention. Consideration for distinguishing influenza-like illness from inhalational anthrax. *MMWR* 2001; 50:984-986. ■

CDC moving quickly on smallpox front

Immunizations, training, vaccine dilution studied

Though officially stating it has no knowledge of any impending use of smallpox as a bioweapon, the Centers for Disease Control and Prevention (CDC) is scrambling with conspicuous speed to be ready for just such an event.

CDC workers from a variety of specialties are not only receiving smallpox vaccinations, they are being trained to give them to others using the old bifurcated needle scarification technique. And, even as creation of a new vaccine is fast-tracked, researchers are trying to determine if the current stockpile of 15.4 million doses can be expanded fivefold by simply diluting the vaccine.

Based on such actions, it is fair to say the agency is at least highly suspicious that the known stocks of smallpox virus are not safely ensconced in their official repositories in Russia and the United States.

"CDC is putting together a number of teams, which will probably total [more than] 100 employees, that could be quickly dispatched in a moment's notice to assist state and local health departments and frontline clinicians investigate suspect cases of smallpox," **Tom Skinner**, a spokesman for the CDC, tells *Bioterrorism Watch.*

"They are Epidemic Intelligence Service (EIS) officers, laboratorians, and others. Part of this includes vaccinating them against smallpox," he explains.

But while confirming that the CDC teams are being trained to administer the vaccine, Skinner would not specify who would be vaccinated following a smallpox bioterror event. "We have a smallpox readiness plan," he says. "Issues around vaccination are covered in that plan. That plan is being finalized. It is considered an operational plan. If we have a case tomorrow, it could be implemented. It covers who should be vaccinated and when."

The general consensus among bioterrorism experts is that those exposed would be vaccinated because the vaccine can prevent infection and possibly death even if given several days out. Likewise, health care workers and their family members would want vaccine if they were expected to care for the infected. Some aspect of quarantine would no doubt come into play because, unlike anthrax, it will be critical to separate the first smallpox cases and their contacts from the susceptible population.

Another aspect of CDC preparations includes the smallpox vaccine dilution study, which is being headed up by **Sharon E. Frey**, MD, associate professor of infectious diseases and immunology at Saint Louis University School of Medicine.

The vaccine, known as Dryvax, is no longer produced, but there are 15.4 million doses left. Frey and colleagues are looking at dilution studies that could maintain vaccine efficacy while increasing the available stock by millions of doses. In a study last year, Frey tried a one to 10 vaccine dilution, which would create a stockpile of more than 150 million doses. However, the resulting vaccine had only a 70% effective rate.

"The undiluted vaccine has about a 95% take rate," she tells *BW*. "It is not perfect, but we would like to be as close to that as we could be."

The new study will include a one to five dilution, which should show greater efficacy while increasing the stockpile to more than 75 million doses.

"We are looking at a 'take' rate for the vaccine, in other words how many people actually develop a typical lesion and whether they have a strong neutralizing antibody response to the vaccine," Frey says. "We know that the vaccine is still good. We actually titered the vaccine and it is very similar to its original titer," she adds. ■

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PEDIATRIC

Special Supplement

Emergency Medicine Reports

The Practical Journal of Pediatric Emergency Medicine

Supplement, December 2001

Emergency department (ED) physicians often are confronted with a problem (i.e., vomiting, diarrhea, rash), not a diagnosis. The challenge to the ED physician is to develop a rational approach to the problem based on the child's history and physical examination.

Based on the clinical information, the ED physician must formulate a cost effective diagnostic evaluation. Fortunately, the majority of children who present with vomiting will have a benign disease and clinical course. Identifying the child with an atypical history, risk factors that point to a serious disease (i.e., significant weight loss), or suspicious findings on physical examination (i.e., split sutures, murmur, etc.) are critical for the patient and rewarding to the physician. This issue provides an opportunity for ED physicians to expand their differential diagnosis and enhance their diagnostic skills for the child who presents with vomiting.

— The Editor

Occult Vomiting in Pediatric Patients

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Nausea

Nausea is a prodrome of vomiting. It is the conscious recognition of subconscious excitation in an area of the medulla that is linked to the vomiting center. Stimuli may originate from irritative impulses from the gastrointestinal tract, a portion of the brain associated with motion sickness, or from the cortex to initiate vomiting. A common cause of nausea is distention or irritation of the duodenum or lower small intestine. As a preliminary step to vomiting, the intestine contracts forcefully while the stomach relaxes. Intestinal contents reflux into the stomach. Nausea is not always experienced prior to the act of vomiting.

The Vomiting Act

Vomiting is the means by which the upper gastrointestinal tract rids itself of its contents when the gut becomes excessively irritated, over-distended, or even over-excited. Distention or irritation of the stomach or duodenum provides a strong stimulus resulting in the act of vomiting. The "vomiting center" for both vagal and sympathetic afferents lies in the medulla at the level of the dorsal motor nucleus of the vagus. Motor reactions initiated to cause the vomiting act are transmitted from the vomiting center through the fifth, seventh, ninth, 10th, and 12th cranial nerves to the upper gastrointestinal tract and through the spinal nerves to the diaphragm and abdominal muscles. Once there is sufficient stimulus, the subsequent sequence of events is: a deep breath, raising of the hyoid bone and the larynx to pull the cricoesophageal sphincter open, closing of the glottis and lifting of the soft palate to close the posterior nares, and a strong downward contraction of the diaphragm with simultaneous contraction of all the abdominal muscles. This force squeezes the stomach between the abdominal muscles, building intragastric pressure. At the same time, reverse peristalsis begins in the antral region of the stomach and passes over the body of the stomach, forcing the contents

toward the esophagus. The pathway ends with gastroesophageal sphincter relaxation, allowing for expulsion of the gastric contents through the esophagus.

Apart from direct stimuli of the gastrointestinal tract, vomiting may be caused by impulses arising in areas of the brain outside the vomiting center. The chemoreceptor trigger zone is located bilaterally on the floor of the fourth ventricle. Electrical stimulation and some pharmaceutical agents can stimulate the chemoreceptor trigger zone directly.

Many people experience the sensation of nausea or the act of vomiting as a result of rapid change in motion. Motion stimulates the receptors of the labyrinth, and impulses are transmitted via the vestibular nuclei into the cerebellum. Passing through the uvula and nodule of the cerebellum, signals are transmitted to the chemoreceptor trigger zone and then to the vomiting center.[1,2]

Table 1. Bilious vs. Nonbilious Causes of Vomiting

BILIOUS
Distal obstructive lesion
• Appendicitis
• Compressing or obstructing mass lesion
• Ileus from any cause
• Incarcerated inguinal hernia
• Intestinal atresia and stenosis
• Intestinal duplication
• Intussusception
• Malrotation with or without volvulus
• Peritoneal adhesions
• Pseudo-obstruction
• Superior mesenteric artery syndrome
NONBILIOUS
• General considerations
• Infectious disease
• Inflammation and irritation
• Metabolic/endocrine abnormalities
• Neurologic disorder
• Obstructive lesion
• Psychological disorder

True vomiting can be characterized by the sequence of nausea, retching, and emesis, and can be categorized as bilious or nonbilious. Bilious vomiting occurs when bile is purged along with the gastric contents. In nonbilious vomiting, antegrade intestinal flow is preserved, and the majority of the bile drains into the distal portions of the intestine. Conditions leading to bilious vomiting involve either a disorder of motility or physical blockage. Obstruction of the gastrointestinal tract can occur at almost any point along its course. Abnormal consequences of obstruction depend on the site of the gastrointestinal tract affected. Table 1 lists the causes of bilious vs. nonbilious vomiting.

Etiologies

Vomiting is a common symptom in childhood. Vomiting may be the chief complaint, or it may be a symptom associated with a multitude of diagnostic entities.[3,4] *(See Table 2.)* In most cases it is merely a manifestation of a benign, self-limited illness. Chronic vomiting always should be considered abnormal. In general, the most important aspect of caring for the child is to exclude a surgical emergency.

Though often self-limited, the act of vomiting is anxiety-provoking for the parent who seeks medical attention for a child. Vomiting is a prominent feature of many disorders of infancy and childhood. The act of vomiting may be a defense mechanism to expel ingested toxins, an abnormality of the vomiting center related to increased intracranial pressure, a result of intestinal obstruction or anatomic/mucosal abnormalities, or the result of generalized metabolic disease. Regurgitation is the passive movement of gastric contents into the esophagus and out through the mouth. It is important to ascertain that the symptom complaint is true vomiting and not regurgitation. Spitting or regurgitation in the small infant usually is caused by gastroesophageal reflux, a self-limited

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Table 2. Diagnostic Entities Associated with Vomiting

GENERAL CONSIDERATIONS

- — Poor feeding technique
- — Postnasal drip
- — Activity of child
- — Immobilization
- — Coughing

INBORN ERRORS OF METABOLISM

- — Amino acid/organic acid metabolism defects
  - • Glutaric acidemia
  - • Isovaleric acidemia
  - • Maple syrup urine disease
  - • Phenylketonuria
  - • Propionic acidemia
  - • Tyrosinemia type I
  - • Urea cycle defects
- — Carbohydrate metabolism defects
  - • Glycogen storage disease II
  - • Galactosemia
  - • Hereditary fructose intolerance
  - • Pyruvate carboxylase deficiency
  - • Pyruvate dehydrogenase complex deficiency
- — Fatty acid oxidation disorders
  - • Carnitine deficiency
  - • MCAD (medium-chain acyl-COA dehydrogenase deficiency)
  - • LCAD (long-chain acyl-COA dehydrogenase deficiency)
- — Lysosomal storage diseases
  - • Mucopolysaccharidoses
  - • Mucolipidoses
  - • Niemann-Pick diseases
  - • Wolman disease
- — Peroxisomal disorders
  - • Zellweger disease
  - • Adrenal leukodystrophy
- — Metabolic and endocrine disorders
  - • Congenital adrenal hyperplasia (adrenogenital syndrome)
  - • Diabetic ketoacidosis
  - • Diabetes insipidus
  - • Hypoparathyroidism

STRUCTURAL OR DISORDERS OF MOTILITY

- — Structural
  - • Annular pancreas
  - • Duplication esophagus/ diverticulum/choledochal cyst
  - • Esophageal/gastric atresia
  - • Esophageal/gastric stenosis
  - • Foreign body
  - • Gastric bezoars
  - • Gastric tumors
  - • Intestinal atresia, duplication, and stenosis
  - • Peptic disease
  - • Pyloric stenosis
  - • Stricture
  - • Tracheoesophageal fistula
  - • Traumatic pancreatitis
  - • Web
- — Disorders of motility
  - • Achalasia
  - • Appendicitis
  - • Gastroesophageal reflux
  - • Gastroparesis
  - • Ileus
  - • Pseudo-obstruction
  - • Scleroderma
- — Gastrointestinal bleeding
  - • Bleeding varices
  - • Dieulafoy lesion
  - • Esophagitis
  - • Gastritis
  - • Mallory-Weiss tear
  - • Peptic ulcer disease (gastric/duodenal)
- — Genitourinary tract disorders
  - • Hydrometrocolpos
  - • Urinary tract obstruction
  - • Pregnancy
  - • Pylonephritis

NEUROLOGIC CONDITIONS

- — Cyclic vomiting
  - • Cyclic vomiting syndrome
  - • Abdominal migraine
  - • Epilepsy
- — Structural
  - • Congenital malformations
  - • Hydrocephalus
  - • Intracranial hemorrhage
  - • Intracranial mass lesions
  - • Vestibular injury or inflammation
- — Infectious diseases
  - • Congenital infections
  - • Encephalitis and meningitis
  - • Neurocysticercosis
- — Toxicology
  - • Acidosis and metabolic byproducts
  - • Kernicterus
  - • Lead encephalopathy
- — Psychological conditions
  - • Anxiety or stress
  - • Bulimia
  - • Rumination

INFLAMMATION AND IRRITATION

- — Regional enteritis
- — Ileocecal inflammation in leukemia (Typhlitis)
- — Necrotizing enterocolitis
- — Ulcerative colitis
- — Food poisoning

INFECTIOUS AGENTS (MOST COMMON PATHOGENS)

- — Bacterial
  - • Campylobacter
  - • *Escherichia coli*
  - • *Giardia lamblia*
  - • Salmonella
  - • Shigella
  - • Streptococcal infection
- — Viral
  - • Rotavirus
- — Parasites
  - • *Ascaris lumbricoides*

problem that typically resolves around age 1. In the healthy, thriving child there is no need for a complex evaluation. Vomiting may be a sign of formula protein intolerance or allergy, especially in association with irritability, loose stools, or blood in the stool. Nearly 7% of infants suffer from cow milk protein intolerance; of these infants, approximately 20% are sensitive to soy protein as well.[3,5]

However, the child who has other symptoms or fails to thrive should receive an extensive evaluation. Infectious, endocrine, neurologic, and urinary disorders, as well as inborn errors of metabolism should be considered in the evaluation of an infant who fails to thrive. Inborn errors of metabolism generally present

Table 3. Evaluating the Child with Vomiting: Important Physical Exam Considerations

APPEARANCE
- Well-nourished?
- Lethargic?
- Toxic? Non-toxic?

VITAL SIGNS
- Temperature
- Heart rate
- Blood pressure
- Respiratory rate
- Pulse oximetry

ASSOCIATED FINDINGS
- Abnormal pelvic exam
- Auscultation for bowel signs
- Discoloration of skin and sclera
- Enlarged parotid glands and hypersalivation
- Neurologic dysfunction (nystagmus, head tilt, weakness)
- Palpation for a mass effect and tenderness
- Tense anterior fontanelle
- Unusual odor
- Visible bowel loops

Table 4. Evaluating the Child with Vomiting: Important Historical Considerations

GENERAL
- Age
- Gender
- Geographic area
- Psychological or behavioral disorders
- Social
- Trauma

EXPOSURES
- Contacts (i.e., day care, home, school)
- Travel
- Pets, wildlife, insects
- Medications, toxins
- Immunizations

CHARACTERISTICS OF VOMITING
- Onset
- Progression (increase or decrease in frequency, associated triggers)
- Alleviated by meals
- Alternating vomiting and lethargy

ASSOCIATED SYMPTOMS (ACUTE, SUBACUTE, OR CHRONIC)
- Fever
- Abdominal pain and frequent, forceful, or bilious emesis
- Chronic headaches, fatigue, weakness, weight loss, or early morning vomiting
- Constipation
- Diarrhea
- Gynecologic complaints
- Nausea and epigastric pain related to meals
- Right- or left-sided abdominal pain

in early infancy, and the vomiting is associated with symptoms of lethargy, hypo- or hypertonia, seizures, and/or coma. As the same symptom complex may be associated with sepsis, it is important to maintain a high index of suspicion in the patient evaluation process. Forceful vomiting indicates a potential anatomic disorder such as pyloric stenosis. Hematemesis implies the likelihood of a proximal gastrointestinal inflammatory process. A syndrome of cyclic vomiting in children may be precipitated by psychological disorders. Acute onset of vomiting suggests infectious enteritis, ingestion, or neurologic disorder. The rapid onset differentiates these disorders from those associated with chronic or recurrent vomiting. Clues discovered while taking the patient's history and performing a physical examination should narrow the diagnostic impression.[6-8]

Evaluation

Characteristics surrounding the act of vomiting become important clues to diagnosis. The history distinguishes true vomiting from regurgitation. For example, post-tussive emesis is common in conditions such as pneumonia or reactive airway disease. Post-prandial emesis may indicate a bowel obstruction or acute gastroenteritis. The presence of bile in the emesis is never normal. Upon completion of the history (See Table 3)[9] and physical examination (See Table 4), the patient's hydration status should be assessed and treated accordingly.

Physical Examination

The physical examination of the patient with vomiting is similar to that of the patient with abdominal pain.[10] Non-gastrointestinal causes of vomiting are excluded, with particular attention paid to the central nervous system. An enlarged or full anterior fontanelle, lethargy, or splitting of the sutures indicates increased intracranial pressure in the infant.[11,12] Associated physical findings, such as discoloration of skin and sclera, may provide clues to diagnosis of hepatotoxins or metabolic disorders. Assess the abdomen for presence and character of bowel sounds, tenderness, guarding, or rebound. Differential considerations vary according to age. For example, sexually transmitted diseases or pregnancy must be considered when evaluating the adolescent female. A thorough pelvic examination and testing also are indicated.

Laboratory Testing

The laboratory evaluation depends upon the clinical presentation, and may include determination of the complete blood count (CBC), serum electrolytes, appropriate liver enzymes, blood ammonia level, metabolic studies, urinalysis, or other test suggested by the clinical presentation. Anemia and iron deficiency can occur with intestinal duplication and obstruction, gastritis or esophagitis, and ulcer disease. Electrolyte abnormalities are found in pyloric stenosis and metabolic abnormalities, while an elevated alanine aminotrans-

Table 5. Causes of Vomiting by Age

BIRTH-3 MOS.	3 MOS.-2 YRS.	2-12 YRS.	ADOLESCENTS
Anatomic	**Anatomic**	**Anatomic**	**Anatomic**
• Trachoesophageal fistula	• Incarcerated hernia	• Appendicitis	• Appendicitis
• Duodenal/jejunal stenosis	• Intussusception	• Pancreatitis	• Testicular torsion
• Esophageal web	• Abdominal neoplasm	• Traumatic pancreatitis	• Renal colic
• Hernia	• Malrotation with volvulus		
• Pyloric stenosis		**Infectious**	**Infectious**
• Annular pancreas	**Infectious**	• Gastroenteritis	• Gastroenteritis
• Gastroesophageal reflux	• Gastroenteritis	• Hepatitis	• Hepatitis
	• Hepatitis	• Otisis media	• Pelvic inflammatory disease
Infectious	• Otitis media	• Pharyngitis	• Peptic ulcer disease
• Gastroenteritis	• Pharyngitis	• Peptic ulcer disease	• Pneumonia
• Meningitis/encephalitis	• Pylonephritis	• Pylonephritis	• Sinusitis
• Pneumonia/pertussis	• Pneumonia/bronchiolitis	• Pneumonia/bronchiolitis	• Urinary tract infection
• Sepsis	• Sepsis	• Sinusitis	
• Urinary tract infection	• Urinary tract infection	• Urinary tract infection	**Metabolic/Endocrine**
			• Diabetic ketoacidosis
Metabolic/Endocrine	**Metabolic/Endocrine**	**Metabolic/Endocrine**	• Thyrotoxicosis
• Amino acid metabolism defect	• Diabetic ketoacidosis	• Diabetic ketoacidosis	• Uremia
• Congenital adrenal hyperplasia	• Uremia	• Hypoparathyroidism	
• Hypoparathyroidism		• Thyrotoxicosis	**Neurologic disorders**
• Inborn errors of metabolism	**Neurologic disorders**		• Head trauma
• Organic acidemias	• Head trauma	**Neurologic disorders**	• Intracranial tumor
• Urea cycle	• Intracranial tumor	• Head trauma	• Migraine headache
		• Intracranial tumor	
Neurologic disorders	**Miscellaneous**	• Neurocysticercosis	**Miscellaneous**
• Hydrocephalus	• Appendicitis	• Pseudotumor cerebri	• Bulimia
• Intracranial tumor	• Bezoars	• Seizure disorder	• Drugs
	• Constipation		• Ingestion/toxins/ethanol
Miscellaneous	• Ingestion/toxins	**Miscellaneous**	• Pregnancy
• Bezoars	• Reactive airway disease	• Cyclic vomiting	• Reactive airway disease
• Milk allergy	• Reye syndrome	• Ingestion/toxins	• Scleroderma
• Shaken baby syndrome	• Shaken baby syndrome	• Reactive airway disease	

ferase, total bilirubin, and glutamyl transpeptidase can indicate liver, gallbladder, or metabolic disease. In the vast majority of pediatric patients with vomiting, radiographs are not helpful. Abdominal ultrasound is the test of choice for pyloric stenosis. It also is useful when considering abdominal abscess; appendicitis; pyloric stenosis; or liver, gallbladder, renal, pancreatic, ovarian, or uterine disease. Contrast radiography is useful for the evaluation and diagnosis of intestinal anatomic abnormalities such as malrotation, intussusception, or volvulus. Not generally indicated for evaluation of vomiting, computed tomography (CT) is an effective tool when anatomic abdominal detail is required (i.e., for appendicitis or appendiceal abscess).[13-15]

Treatment

Treatment is disease-specific. Associated signs and symptoms that should lead to a pediatric subspecialist referral include weight loss, severe abdominal pain or irritability, gastrointestinal bleeding, evidence of intestinal obstruction, serum electrolyte abnormalities, an abnormal neurologic examination, dehydration, signs of an acute abdomen, or lethargy.[16-21]

In conclusion, because a wide variety of stimuli may produce vomiting, there are many diagnoses to consider. Differential considerations vary by age, and the spectrum of etiologies are more prevalent in specific age groups. *(See Table 5.)*

Case Studies

CASE 1

A 4-week-old female arrives in the emergency department (ED) with a one-day history of lethargy and vomiting. The mother reports no prenatal care but that the baby was delivered vaginally without complication. The baby and mother were discharged from the hospital two days after delivery. The baby is breastfed and has no difficulty nursing. The baby's frequent vomiting is what brings her to the ED. Mother states the last few episodes of vomiting were green in color.

Examination. The baby is lethargic, pale, and grunting.

Vital signs are: Pulse = 190/minute, respiration rate (RR) = 60/minute, blood pressure (BP) = 90/60 mmHg, rectal temperature = 36°C.

Breath sounds are clear. Extremities are mottled. Skin turgor is poor. There are decreased bowel sounds and the distended abdomen is firm to palpation.

What is the most likely diagnosis is this case: pyloric stenosis, malrotation, duodenal atresia, sepsis, or inguinal hernia?

Discussion. The history, physical examination and a cross-reference of Tables 1 and 5 support anatomic obstruction as the presumed cause of bilious vomiting in the neonate. The first step is application of supplemental oxygen and correction of the hypovolemia in this child. Although anatomic obstruction is the major concern, this child also should receive metabolic and septic evaluations.

Malrotation results from failure of normal rotation of the midgut leading to abnormal fixation of the mesentery. The majority of patients present in the neonatal period with bilious vomiting, abdominal distension, and pain. Older patients may present with a history of feeding problems and formula intolerance with vomiting or with poor growth. Abdominal radiograph findings are variable. An upper gastrointestinal study is the study of choice to make the diagnosis. The duodenal loop may appear dilated with obstruction at the third portion, or there may be narrowing of the small bowel at the site of the obstruction and spiraling of the small bowel about the mesenteric artery. In cases of malrotation and midgut volvulus, the normal mesenteric attachments are replaced by fibrous bands called Ladd bands; surgical repair involves release of the Ladd bands (Ladd procedure) and resection of necrotic bowel as needed. Surgery should not be delayed to prevent further necrosis of the bowel.

The correct diagnosis in this case is malrotation.

CASE 2

A 6-year-old boy fell 5 feet from the top of a playground slide. He rode his bike home and complained to his mother of a bad headache. Within an hour he vomited twice. The mother became concerned and brought him to the ED for evaluation.

*Physical Exam.*The boy is agitated and cannot be calmed by his mother. Vital Signs: heart rate (HR) = 110/minute; RR = 22/minute; BP = 98/70 mmHg; oral temperature = 38°C; weight = 22 kg.

The child exhibits spontaneous, unlabored respirations. His extremities are warm, with a capillary refill time of 2 seconds.

Is the management priority in this case radiologic imaging; early consultation and transfer; assessment for associated injuries; administration of dexamethasone; or stabilization of airway, breathing, and circulation?

The priority in this case is stabilization of the patient's airway, breathing, and circulation.

Discussion. Agitation and uncooperativeness may be normal behavior in a frightened toddler; this is not appropriate for a school-age child. His cardiopulmonary status is normal. The combination of history and physical examination findings are consistent with closed head trauma. The management goal of head injury in children is to prevent secondary injury to the brain. Prevention of hypoxia, ischemia, and increased intracranial pressure is essential. Relative dehydration will decrease intracellular water and thus reduce intravascular pressure and fluid leakage from blood vessels in the brain. Therefore, supplemental oxygen by facemask and establishment of vascular access are appropriate initial interventions. This child is able to maintain his airway and is ventilating normally. Though the child walked into the ED, he remains at risk for associated injuries, including spinal cord injury. Prompt consultation with a neurosurgeon will determine the need for transport and focus neuroimaging studies. Osmolar therapy may provide some benefit in acutely decreasing intracranial pressure; however, it should not precede the aforementioned management priorities. Dexamethasone is not recommended acutely for the treatment of increased intracranial pressure.

CASE 3

A previously healthy 2-year-old girl has a history of upper respiratory infection for six days. Her primary care provider diagnosed an acute otitis media two days ago, for which he prescribed oral antibiotic therapy. The child is unable to take her medicine because of frequent vomiting. Dad brings his daughter back to the physician's office complaining of persistent fever, increase in vomiting, and because she is not behaving normally.

Physical Exam. The child is listless. She moans when her body is moved. As her neck is flexed, she grimaces and brings her shoulders and knees involuntarily up off the stretcher, demonstrating nuchal rigidity. Petechiae are noted on her extremities.

Vital Signs: HR = 140/minute; RR = 40/minute; BP = 80/40 mmHg; oral temperature = 40° C; weight = 14 kg.

What is the most likely diagnosis in this case: encephalitis, Reye syndrome, meningitis, intussusception, or septic shock?

Most likely, the patient is suffering from meningitis.

Discussion. The early phases of meningitis may be confused with illnesses such as upper respiratory infection, otitis media, or simple gastroenteritis, confounding the practitioner. The younger the child, the fewer the signs and symptoms evident for the diagnosis of meningitis. The physical finding of nuchal rigidity in a febrile child suggests meningitis, but is not conclusive. The differential diagnosis for nuchal rigidity includes cervical spine trauma, myositis, retropharyngeal abscess, adenitis, pneumonia, and infection of congenital anomalies such as branchial cleft or thyroglossal duct cysts. While it is important to determine nuchal rigidity, this is difficult in infants and young children. It is reported that fewer than 15% of children younger than 18 months of age exhibit resistance to flexion of the neck. When meningitis is suspected, the diagnostic test of choice is the lumbar puncture. Common childhood bacterial pathogens include *Haemophilus influenzae* type b, *Streptococcus pneumoniae*, and *Neisseria meningitidis*. The availability of conjugated vaccines against *H. influenzae* has reduced dramatically the incidence of meningitis in childhood. In the near future, we may witness a similar reduction in cases with the widespread use of pneumococcal vaccine. Following the widespread use of *H. influenzae* type b vaccines, *S. pneumoniae* has become the most common cause of invasive bacterial infection in children in the United States. Pneumococci are the most common cause of bacteremia in young children ages 2-36 months who have fever without an identifiable source, accounting for more than 84% of recovered bacterial pathogens. Children younger than 12 months have the highest rates of pneumococcal infections. Among children younger than age 5, pneumococcal infections cause an estimated

minimum of 1400 cases of meningitis, 17,000 cases of bacteremia, 71,000 cases of pneumonia, and 5 million to 7 million cases of otitis media annually. The new American Academy of Pediatrics guidelines state that the heptavalent pneumococcal conjugate vaccine (PCV7) is recommended for use in all children 23 months of age and younger. Although other pneumococcal vaccines are available, PCV7 represents the first pneumococcal vaccine approved for use in children younger than age 2. The policy recommends that PCV7 be given concurrently with other recommended childhood vaccines at 2, 4, 6, and 12-15 months. The number of PCV7 doses required depends upon the age at which vaccination is initiated. The vaccine also is recommended for all children 24-59 months of age who are at especially high risk of invasive pneumococcal infection. This includes children with sickle cell disease, human immunodeficiency virus (HIV) infection, and other children who are immunocompromised.

Conclusion

When the ED physician is confronted with a child with a chief complaint of vomiting, a broad differential should be considered. The history (acute vs chronic, weight loss, febrile vs afebrile) and physical examination (fontanelle, nuchal rigidity, abdominal examination) should guide the diagnostic evaluation. Early recognition and appropriate subspecialty referral for a child who has a disease that requires subspecialty expertise will improve the outcome for a child who presents with vomiting.

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