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AHC Media

Emergencies in the Dialysis Patient

As a group, I find most emergency physicians skeptical of many of the "advanced" technologies used to treat chronic diseases. I attribute this to our biased experience with these patients; those who do well are not likely to come to the ED very often. We typically see those with problems and complications, which unduly influences our opinion about the benefits of such treatments. So it is with patients receiving dialysis therapy. This issue of EM Reports *will review the myriad of problems related to renal failure and its treatment.*

— J. Stephan Stapczynski, MD, Editor

Definitions

Kidney disease is classified based on glomerular filtration rate (GFR), according to the Kidney Disease Outcomes Quality Initiative Guidelines of the National Kidney Foundation. Chronic kidney disease (CKD) involves kidney damage (by pathology, imaging, or clinical tests) or GFR < 60 mL/min/1.73 m^2 for 3 months or longer. A GFR of 60 is chosen, as it defines loss of at least 50% of the normal adult kidney function. This article discusses the end stage renal disease (ESRD) or kidney failure patient (stage 5), as defined by a GFR of less than 15 mL/min/1.73 m^2 or treatment with dialysis.

There are two modalities for dialysis: hemodialysis (HD) and peritoneal dialysis (PD). The decision to do one instead the other is based on kidney function, overall health, personal preferences, home situation, and the presence of gastrointestinal conditions such as inflammatory bowel disease and diverticular disease. PD has two modalities: automated PD (APD) and continuous ambulatory PD (CAPD).

Epidemiology

The number of patients with ESRD and on dialysis has been steadily increasing. In 2009, there were 370,274 patients on HD and 27,522 patients on PD in the United States.

More than half of the ESRD cases are attributed to diabetes mellitus (DM), with hypertension (HTN) being the second most common cause.[1] African Americans, Native Americans, and Hispanics are at greater risk of developing ESRD.[1]

The costs for ESRD are staggering. This population constitutes 1.9% of Medicare patients but accounts for 8.1% of the Medicare budget spending (2009 data).[2] The cost per patient is estimated at $82,000 per year when on HD and $61,000 per patient per year when on PD. This is significantly higher than the estimated annual cost of $30,000 for a kidney transplant patient.

ED Evaluation

The ED evaluation of the dialysis patient should follow the same steps as with every acutely ill patient. However, with these patients, other important items to address include:

1. The HD/PD access site must be located and evaluated. Access points can become both clotted (HD) and infected (HD and PD). The HD access points

Executive Summary

- The dialysis access site should be assessed in ESRD patients: Inspect for redness and exudate; palpate for tenderness and swelling.
- Assess for possible hyperkalemia in ESRD patients: An ECG and serum potassium via point-of-care methodology are very useful.
- Symptoms of fluid overload in ESRD patients can be temporized with oxygen, patient positioning, BP reduction, diuretics (for patients who still make urine), and noninvasive ventilatory support, but definitive treatment requires dialysis.
- Most medications, particularly opioid analgesics, require dose adjustments in renal failure patients.

can also bleed. Rarely, the shunts cause high-output cardiac failure. In general, vital signs in the arm with a HD access point are to be avoided.

2. The last HD session: This can provide a gauge of the chances for hyperkalemia and other major electrolyte changes. Longer intervals also make the patient more prone to volume overload.

3. If known, and if the patient is not in extremis, the dry versus actual weight can help further define volume status.

4. In PD patients, any changes to the color or transparency of the dialysate fluid is an indication of infection.

5. Ask the patient if he or she still makes urine. Patients with some residual urine output are partially protected from the fluid overload and electrolyte complications described below.

6. Ask the patient about the precipitating cause of his or her renal failure. These conditions likely still exist and may contribute to the symptoms the patient is experiencing and may also influence treatment in the ED.

Electrolyte Emergencies

Hyperkalemia. Hyperkalemia is common in the dialysis patient, and is also one of the few lethal electrolyte disorders.

First, the physician should make sure the sample is not hemolyzed. Hemolysis is a common cause for falsely elevated potassium levels.

Potassium levels are of concern at a level of 6 mEq/L or above; however, the urgency of treatment is based on the absolute potassium level, the rapidity of the rise, and a multitude of other factors such as the presence of acidosis and other electrolyte disturbances.[3] In general, ECG changes mandate treatment, but a normal ECG is never reassuring, especially with potassium levels greater than 6 mEq/L.[3]

The usual teaching for the progression of the ECG in hyperkalemia is: peaked T waves, widened QRS, the P wave disappears, and, finally, a sine wave.[4] Ventricular fibrillation in some cases may be the first electrocardiographic manifestation.[3] Heart blocks can also be seen in profound hyperkalemia. It is critical to remember that the typical ECG progression is not always stepwise and is very patient-dependent.[3]

The emergency management of hyperkalemia should follow a chronological sequence: (*see Table 1*)

1. Antagonism of the effects on excitable cells, especially the myocardium;
2. Redistribution of potassium to the intracellular compartments;
3. Increasing the elimination of potassium from the body.[3]

Management of Hyperkalemia. Calcium salts work by membrane stabilization of the myocytes without altering the total potassium concentration. This should be the first intervention in a patient who has myocardial irritation from hyperkalemia.[3] The salts of calcium are available in two formulations: calcium chloride and calcium gluconate.

The gluconate is less potent, as it has less calcium per weight. Calcium gluconate cannot be pushed as a bolus in general, as it causes hypotension. It requires intact liver metabolism to release calcium and, therefore, is not the best option for patients with chronic liver disease or those patients in shock or cardiac arrest. One definite benefit of this formulation is that it does not cause vein irritation, so it can be infused via a peripheral vein.[3]

Calcium chloride is about three times more potent than the gluconate, as it contains more calcium per weight. It can be pushed, but only when using a central line, as it is very irritating to the veins and will cause tissue necrosis if it extravasates. The molecule disassociates when in plasma without any metabolism.[3]

The effects of the calcium salts are short-lived, estimated at 30-60 minutes. Frequently, re-dosing will be needed.

Albuterol and other beta adrenergics work by redistribution of the potassium into the cell. To reduce the serum potassium by 0.6-1 mEq/L, large doses should be used (10-20 mg). Terbutaline has also been used, causing a decrease of serum potassium by an average of 1.3 mEq/L within 60 minutes of dosing. Both medications will cause tachycardia. The effects of the beta adrenergics are blunted in patients using beta-blockers. Up to 40% of patients can be resistant to the hypokalemic effect of the beta adrenergic agents, even when not using beta adrenergic blockers.[5] Therefore, these agents should never be used alone.[3]

Insulin and glucose work by redistribution of potassium into the cell. The most important consideration when using insulin and glucose is that the combination often results

Table 1: Management of Hyperkalemia

Drug	Dose	Onset of Action	Duration of Action	Mechanism of Action (MOA)
Calcium chloride	10 mL of 10% solution IV (use only central line or in code situation) over 10 minutes Can repeat in 5 min	Immediate	30-60 min	Antagonizes cell excitation
Calcium gluconate	10 mL of 10% solution IV over 10 min; can repeat in 5 min	Immediate	30-60 min	Antagonizes cell excitation
Insulin	10 units regular insulin IV, with 25 g of D50W IV; should use D5W or D10W infusions thereafter	20 min	4-6 hours	Intracellular K redistribution
Albuterol	10-20 mg by nebulizer	30 min	2 hours	Intracellular K redistribution
Terbutaline	7 mcg/kg body weight, SQ	< 60 min		Intracellular K redistribution
Sodium bicarbonate	150 mEq/L at 1-2 cc/kg/hr	Hours	While infused	Intracellular K redistribution
Furosemide	40-80 mg IV	15 min	2-3 hours	Urinary K excretion
Bumetanide	2-4 mg IV	15 min	2-3 hours	Urinary K excretion
Sodium polystyrene sulfonate	30-60 g PO or enema	At least 12 hours (probably longer)	At least 24 hours	Fecal K excretion

Adapted from: Weisberg LS. *Crit Care Med* 2008;36:3246-3251.

in hypoglycemia. Therefore, use two ampules of 50% dextrose instead of one unless the patient is hyperglycemic, and closely monitor the glucose after dosing.

Since the volume of insulin is so small, dilute 10 units in 10 mL of saline so the dose does not stay in the port. Otherwise, flush the dose with 10 mL of saline. The onset of effects is about 20 minutes, and lasts for 4-6 hours.

Sodium bicarbonate works by potassium redistribution into the cell, but the efficacy of this common intervention is not well supported by the literature. A secondary mechanism of action for bicarbonate is possibly by urinary alkalinization, resulting in increased urinary potassium excretion. It should probably only be used if significant acidemia is present. The limited literature showing some benefit uses infusions rather than boluses or discrete doses.[3] The effect of the bicarbonate is very modest, at best, and lasts only while infused.[3]

Loop diuretics work by increasing potassium elimination by the kidney. Of course, these drugs will not have any effect in the anuric patient, but potassium is eliminated even in severely impaired kidney function.

Sodium polystyrene sulfonate (SPS; Kayexalate®) works by increased elimination from the gut. The cation exchange resin has a preference for binding potassium. In the gut lumen, it exchanges the sodium ion for a potassium ion, which is then fecally excreted. SPS can be administered orally or by an enema. Sorbitol is added to avoid concretions, and the premixed suspension is the only formulation available in many U.S. hospitals.[6]

Contrary to teaching, SPS will not lower the potassium until about 12-24 hours after administration, making it a poor choice when managing hyperkalemia in the ED. It is also uncomfortable and inconvenient for the patient, as it causes diarrhea.[6]

Hemodialysis (HD) is the definitive means for removing excess potassium and the preferred treatment for hyperkalemia. The serum potassium falls by about 1 mEq/L by 60 minutes of HD, and plateaus after 180 minutes with an average decrease of 2 mEq/L.[3]

Hypermagnesemia. Mild to moderate hypermagnesemia is frequently found in the patient with ESRD due to decreased renal excretion, but also from increased intake of magnesium-containing products such as laxatives, enemas, and antacids.[7] The results of

mild to moderate hypermagnesemia (2.2-5 mEq/L) include progressive flaccid muscle weakness, areflexia, drowsiness, and confusion. Findings with severe hypermagnesemia (> 5 mEq/L) include bradycardia, heart blocks on ECG, decreased level of consciousness, hypotension, and cardiac arrest. Hypermagnesemia is treated with 5-10 mL of intravenous calcium gluconate (10% solution) given over 5 minutes, prior to initiation of emergent HD.

Hypocalcemia. Hypocalcemia is a frequent occurrence in patients with renal failure. It is caused by a decrease in the conversion of 25 hydroxyvitamin D to its active form 1,25 dihydroxyvitamin D in the kidney. Hypocalcemia also results from the aggressive treatment of metabolic acidosis with intravenous sodium bicarbonate.[7]

Hypocalcemia is defined as a serum calcium level less than 8.5 mg/dL or an ionized calcium less than 4.2 mg/dL. Ionized levels are more accurate in identifying calcium disturbances in patients with ESRD because total serum calcium levels are affected by pH changes, albumin fluctuations, and hyperphosphatemia.[8] Symptoms developing when ionized calcium levels fall below 2.5 mg/dL include perioral and fingertip paresthesias, muscle cramps, and muscle weakness. Physical examination findings include hyperreflexia, positive Chvostek and Trousseau signs, and a prolonged QT interval on ECG. At ionized levels below 1.6 mg/dL, tetany, seizures, and heart failure can ensue.

Treatment for severe, symptomatic hypocalcemia begins with 10 mL of 10% calcium chloride IV, given over 10-20 minutes. This is followed by a continuous infusion of 0.02 to 0.08 mL/kg/hr of 10% calcium chloride. Alternatively, 10% calcium gluconate IV may be used. Follow the serum calcium level after the initial IV bolus and then every 4-6 hours while using the infusion. Treatment with IV calcium should be reserved only for treatment of severe or symptomatic hypocalcemia.

Hyperphosphatemia. Hyperphosphatemia is defined as a serum phosphorus concentration greater than 5 mg/dL. Elevated serum phosphorus levels occur in ESRD when the GFR falls below 30 mL/kg/1.73 m^2, and it is due to decreased renal filtration and excretion. Chronically elevated levels are associated with increased cardiovascular mortality risk and altered homeostasis of bone metabolism pathways (see below). This forms the basis for treatment of hyperphosphatemia with phosphate binding agents and dietary phosphate restriction in ESRD.[7,9]

Hyperphosphatemia is usually mild, but severe elevations (greater than 7 mg/dL) with concomitant life-threatening hypocalcemia can be seen with ingestions of large amounts of sodium phosphate laxatives used for bowel preparation. It can also be caused by the use of phosphate-containing medications such as calcium phosphate. Patients with severe hyperphosphatemia must have a rapid correction with intensive HD.[7,9]

Cardiovascular Emergencies

Pulmonary Edema/Volume Overload. Fluid overload with resulting pulmonary edema is a frequent cause of emergency presentation in dialysis patients. Contributing factors include excessive salt and fluid intake; missed dialysis sessions; failure to achieve dry weight during dialysis; uncontrolled hypertension; pre-existing congestive heart failure; and new-onset acute coronary syndrome (ACS) or cardiac arrhythmias. Patients with fluid overload present with the complaints of shortness of breath, dyspnea on exertion, fatigue, and increasing edema. Physical examination findings include hypertension, decreased breath sounds, rales, elevated jugular venous pressure, and peripheral edema or anasarca. Estimated dry weight versus actual weight, if known and feasible, may also be used to evaluate the degree of volume overload present.[7,10]

The chest X-ray will show pulmonary vascular congestion, interstitial edema, and/or pulmonary effusion. An ECG can demonstrate precipitants of volume overload such as arrhythmias or ischemia. Brain natriuretic peptide (BNP) is not a reliable marker of acute volume overload in ESRD.[11] The BNP is elevated in most HD patients and its interpretation in this acute setting has not been well established.[7] However, chronic elevations of BNP are associated with increased mortality in this patient population.[12]

Emergent HD is the definitive treatment for volume overload and acute pulmonary edema. However, several initial steps in the ED can temporize these patients while arranging for HD. High-dose intravenous loop diuretics, such as furosemide (maximum dose of 120-240 mg IV), are useful in those with residual urine output. Control of elevated blood pressure and vasodilatation with SL, IV, or cutaneous nitroglycerin is critical. The target should be a 20-25% decrease in MAP.

Noninvasive positive pressure ventilation (NIPPV) using CPAP or BiPAP has been used in patients with respiratory distress. It requires a cooperative patient who can follow instructions and can protect his/her airway. There are no evidence-based guidelines on the use of NIPPV for ESRD patients in pulmonary edema. Intubation may be necessary prior to emergent HD if the patient is unable to tolerate NIPPV and the above interventions are ineffective in reducing the respiratory distress.

Angina/Chest Pain. Cardiovascular disease accounts for the deaths of about 50% of patients with ESRD.[13] The relative risk of coronary artery disease in these patients is 5-30 times greater than that of the general population. The combination of traditional risk factors for coronary artery disease (CAD) (especially DM and HTN) and conditions related to ESRD, including uremia, hyperphosphatemia, and hyperparathyroidism, result in chronic systemic inflammation with endovascular injury and dysfunction.[7,10]

ESRD can lead to significant baseline ECG abnormalities that can

make the ECG interpretation more challenging, such as left ventricular hypertrophy and other changes related to electrolyte disorders.

Serum troponin T levels may be chronically elevated due to decreased renal clearance; however, an elevated value should not be attributed solely to ESRD, and an appropriate evaluation and observation period to rule out ACS is indicated.[10,14]

Dialysis-dependent patients with ACS should be treated with traditional therapies including aspirin, clopidogrel (Plavix®), and morphine. Thrombolytics and glycoprotein IIb/IIIa inhibitors do not require dose modification. However, if enoxaparin (Lovenox®) is used instead of unfractionated heparin, it is given only once daily, as it is renally cleared.

Hypertensive Emergencies

Approximately 85% of patients undergoing dialysis have hypertension. Contributing factors include volume overload, decreased vascular compliance, increased renin secretion, and increased sympathetic tone. Noncompliance with antihypertensives in this patient population may precipitate a hypertensive emergency.

In the ED, the initial goal of therapy for hypertensive emergency is lowering of the mean arterial pressure (MAP) by 20-25% using parenteral agents. Overly aggressive reduction of blood pressure can result is worsening of cardiac ischemia, worsening renal dysfunction, and cerebral ischemia. Intravenous agents of choice for hypertensive encephalopathy include labetalol and nicardipine. Sodium nitroprusside is best avoided in ESRD patients, as the metabolic by-product of its administration, thiocyanate, may accumulate with toxic effects. Parenteral nitroglycerin can be used to control hypertension if cardiac ischemia exists. Emergent HD may be necessary in cases of volume overload.

Pericarditis/Tamponade. The presentation of a pericardial effusion depends on how rapidly the fluid accumulated. The slower the rate of accumulation, the less dramatic the presentation, as the pericardium has time to stretch. These patients may be ill when first seen but will tolerate delays to management. Between 80-120 cc of pericardial fluid can accumulate without elevating the intrapericardial pressure. Beyond that, 20-40 additional milliliters almost doubles the intrapericardial pressure. This pressure is transmitted directly to the heart, causing increased atrial and central venous pressures. The end result is decreased cardiac output and decreased coronary perfusion.[15]

Beck described the classic physical findings of tamponade in 1935. However, up to two-thirds of patients will not have the triad of elevated central venous pressure (CVP), low arterial blood pressure, and muffled heart sounds. Jugular venous distention (JVD), in particular, may not be present, especially if the precipitant of tamponade is dehydration.

Pulsus paradoxus is described as another common finding in tamponade. It is an exaggeration of the normal drop in systolic blood pressure that occurs with inspiration. This finding is not reliably present in many patients with tamponade.[16]

The chest X-ray with a chronic effusion will reveal a "water bottle" shaped heart. However, a normal heart size does not exclude tamponade.

The ECG in a pericardial effusion shows low voltages and other nonspecific changes. Electrical alternans is also nonspecific, with the exception of total electrical alternans, which involves both the P and QRS waves.[16]

The management of a symptomatic pericardial effusion involves several steps. First, intravascular pressure should be restored, as low CVP due to low intravascular volume results in further decreases of cardiac output. Emergent HD should be arranged. Pericardial effusions in stable HD patients can often be treated with intensive HD alone, while a minority require pericardiocentesis.[15] However, for the unstable or crashing patient, the definitive management is with a pericardiocentesis. This procedure should be done with ultrasound guidance to minimize complications and improve success rates.[17]

Bedside transthoracic echocardiography is the fastest and most reliable diagnostic study. For the diagnosis of a pericardial effusion, two views are best: the subxiphoid and the parasternal long axis.[18,19]

Tamponade physiology is identified by the presence of right atrial early diastolic collapse. This is the most sensitive sonographic finding, especially when this collapse persists throughout diastole.[18] Less sensitive findings include increased right ventricular size with diastolic collapse and late expansion; minimal to no change to the IVC diameter with respiration; and paradoxical septal wall motion.[18]

Hematologic and Gastrointestinal Emergencies

Anemia and Bleeding. Most patients with CKD will develop a normocytic and normochromic anemia secondary to the diminished renal production of erythropoietin and decreased red blood cell survival. Erythropoietin or darbepoetin alfa is often given to these patients to maintain a hemoglobin level between 10-11 g/dL. Without intervention, most patients with CKD would eventually have their hematocrit stabilize between 15% and 25%.[20,21]

Platelet dysfunction frequently manifests as easy bruising or mucosal bleeding, but may present as abnormal bleeding after procedures, intracranial hemorrhage, or spontaneous retroperitoneal bleeding. The underlying pathology is not fully understood but seems to be a combination of both intrinsically abnormal platelet function and the effects of uremic toxins and anemia.

Patients on dialysis appear to have a moderately increased risk of esophagitis, gastritis, and angiodysplasia, creating an additional risk for GI bleeding.[22,23] The use of

anticoagulation during HD is an added risk for more severe bleeding.

As the pathogenesis of uremic bleeding is multifactorial, emergent bleeding in the patient with CKD may respond best to a combination of treatments. Administration of dDAVP is effective in patients with uremic bleeding through stimulation of factor VIII and von Willebrand factor release. The preferred route is IV, with a single dose of 0.3 mcg/kg given over 15-30 minutes. It may also be given subcutaneously with the same dosing. The intranasal dose is 3 mcg/kg. The onset of action is within one hour and the half-life between 3 and 9 hours.[24-26]

If patients are unresponsive to dDAVP, cryoprecipitate in a dose of 10 bags over 30 minutes may be administered. Conjugated estrogen infusions can be very helpful. Although the duration of action is long (approximately 1 week), the onset of effects is about 6 hours, limiting its use in the critically ill. Finally, dialysis may be employed to improve bleeding time.[25] If bleeding patients are found to be anemic, they should be transfused with packed red blood cells to a hematocrit of 25-30%.

Metabolic Bone Disease. CKD-MBD is a syndrome that encompasses abnormalities in calcium, phosphorus, parathyroid hormone (PTH), or vitamin D metabolism; changes in bone mineralization, volume, and turnover; and extra-skeletal calcification, including vascular or other soft-tissue calcification.[27] The disorder leads to notable morbidity and mortality. These patients have significantly increased relative risk of hip and lumbar fractures.[28,29]

In CKD, the measured serum concentrations of phosphate and calcium generally remain normal until estimated GFR drops to approximately 20 mL/min/1.73 m^2. Gradually, the proximal tubules' ability to produce 1,25-dihydroxyvitamin D3 (calcitriol) is significantly reduced, and calcium levels fall. This stimulates PTH release. PTH acts to increase renal phosphate excretion, but the damaged kidneys have impaired phosphate excretion. This, in combination with the promotion of bone resorption caused by PTH, results in elevated serum phosphate concentrations.[30]

The increased bone turnover in response to PTH results in weak bones that are prone to fracture. Osteitis fibrosa cystica occurs in this setting as bone is replaced with fibrous tissue. Rarely, there may be a localized occurrence called a Brown tumor or an osteoclastoma. This generally presents as a painful cystic area in the bone that appears as a lytic lesion on X-ray. As treatment of secondary hyperparathyroidism has improved, the prevalence of this disorder has decreased.[31]

Osteomalacia, a disorder wherein bones have defective mineralization, has also become less common. It was seen in patients in whom aluminum-containing antacids were used to help bind phosphate in the intestines. Aluminum interferes with the bone mineralization process, causing increased relative volumes of the soft, organic portion of the bone matrix, called the osteoid. Other trace metals may also result in osteomalacia.[32]

Adynamic bone disease is currently the most common presentation of renal osteodystrophy. This results from the exogenous suppression of the parathyroid gland through treatments such as calcium-containing phosphate binders and vitamin D analogues. The low activity of the osteoclasts and osteoblasts results in structurally weak bone, increasing fracture risk. Additionally, this form of renal bone disease is associated with increased extra-skeletal calcification, as patients often are hypercalcemic.[33,34]

Metastatic calcification is the final entity in CKD-MBD. While extra-skeletal calcification can occur anywhere, an important manifestation of this is calciphylaxis or calcific uremic arteriolopathy.[35] As the latter name implies, there is calcium deposition and hardening of small vessels, which can lead to ischemia. Calciphylaxis presents as very painful, violaceous lesions that may progress to necrosis. The risk factors include: female gender, obesity, chronic dialysis, and the use of certain drugs, such as corticosteroids and warfarin.

Treatment standards are not well established but may include discontinuation of vitamin D analogues and using non-calcium-containing phosphate binders such as sevelamer (RenaGel®, Renvela®). In cases of severe, secondary hyperparathyroidism, cinacalcet (Sensipar®) may be helpful. The most promising recent development in calciphylaxis treatment is the use of sodium thiosulfate, which acts as an antioxidant and increases calcium solubility.[36] Its use, however, is off-label, dosing varies, and administration is during or after dialysis.

For the emergency physician, it is important to recognize that CKD patients are at risk for fractures and vascular disease, both calciphylaxis and possibly also coronary artery disease.

Emergencies Related to Access

The hemodialysis patient must have vascular access. For urgent treatment, this is often accomplished using a double-lumen central venous catheter. A tunneled catheter with internal jugular access on the right, with the tip of the catheter ending at the caval-right atrial junction, has the lowest complication rate. Several companies manufacture double-lumen catheters to allow dialysis.

A common practice is to initiate HD with a central venous dialysis catheter with the ultimate goal to establish an arteriovenous (AV) fistula in the nondominant forearm.[37] An AV fistula uses no prosthetic material, but is a surgically created direct anastomosis of an artery and a vein. Dialysis via a forearm AV fistula using a radial artery anastomosis to the cephalic vein is preferable, although brachial artery anastomosis with the basilic or cephalic vein is also common. Other, less common sites used to create AV fistulas include the proximal thigh. AV fistulas are advantageous due to their lower infection rates when compared to either central venous catheter

access or AV grafts.[37]

AV fistulas typically will fail after prolonged use due to aneurysm or thrombosis. In other cases, adequate veins may not exist. These conditions will require the creation of an AV graft using a synthetic material such as polyurethane or polytetrafluoroethylene. The graft constructed is commonly in a U-shaped tunnel in the subcutaneous tissue of the forearm, although many configurations are possible when sites become exhausted or are no longer usable. Unfortunately, AV grafts have higher complication rates than AV fistulas, including thrombosis, infection, pseudoaneurysm, and limb loss.[37]

Infection is a common complication of central venous access in the dialysis patient and may require removal of the device. The patient may have signs of infection with local erythema, swelling, and purulent discharge at the catheter site. They may also have fever and chills and an elevated white blood cell count. The findings may also be more subtle, with only malaise and a history of fever being described.

These infections often require removal of the tunneled catheter if the patient appears ill or septic. When the catheter is removed, the tip should be sent for culture.[38] The traditional approach requires removing a catheter if infection is considered likely, while some authors do not think removal is necessary in all cases.[37,39]

Some infected AV fistulas can be salvaged with antibiotic therapy alone, while an infected AV graft will require surgical excision similar to any other prosthetic device that becomes infected. The most common bacterial pathogens in the renal patient are *Staphylococcus aureus*, including MRSA, as well as coagulase-negative *Staphylococcus* and *Streptococcus*. Initial antibiotic choices must include coverage for MRSA, and gram-negative and gram-positive organisms.[37]

Bleeding from a central venous catheter is initially treated with direct pressure at the site where the catheter enters the vessel. Unfortunately, pressure applied to where a tunneled catheter penetrates the dermis will not be adequate and should be directed to where the catheter penetrates the central vessel. Unfortunately, one of the disadvantages of a subclavian line is the inability to apply direct pressure at the catheter's entrance into the subclavian vein.

An AV fistula or graft with persistent bleeding should initially be treated with elevation and direct pressure. However, over-aggressive or prolonged pressure can cause thrombosis of the access site and should be avoided. Several methods of direct pressure can be attempted, including digital pressure, gauze and elastic wraps, or plastic vascular clamps supplied by a dialysis unit. Topical agents may be used as well, such as Surgicel®, or chemical cautery using a silver nitrate applicator.[40] A small figure-of-eight suture may be employed if persistent bleeding is noted at the hemodialysis puncture site. Nylon suture is less inflammatory, although absorbable suture material is becoming more common in the emergency setting due to patient compliance issues. Care must be taken to prevent injury to the actual AV fistula or graft, and the suture technique should be as superficial as possible.

A clotting disorder can be the reason for persistent bleeding. In those cases, PT/INR/PTT should be obtained. If the heparin used during dialysis causes a prolonged PTT, it can be reversed with IV protamine.

Bleeding must be controlled in the ED and the pressure device removed prior to discharge. AV fistula or graft patency, adequate flow and bruit, and hemorrhage control should be verified. Strict warnings must be given to the patient.

Clotted catheters, fistulas, and grafts are also common problems. Thrombosed catheters can be treated with tPA or urokinase. Dosing protocols vary widely between institutions, and consultation is recommended before treatment.[37,41]

If an AV fistula or graft becomes thrombosed, the cause should be investigated. Venous stenosis is a likely etiology. If stenosis is the cause, it will have to be treated or reoccurrence is likely. The thrombosis can be treated with pharmacologic intervention to dissolve the clot, or surgical removal may be undertaken. Immediate consultation with a vascular surgeon is needed to determine the treatment plan. Interventional radiology, when available, can also help with methods such as thrombolysis and angioplasty.[42]

Aneurysms, which are full-thickness dilatations of a vessel wall, develop over time with AV fistulas. Current recommendations are to repair an aneurysm in a fistula only if it occurs at the arterial anastomosis site, but other areas do not necessitate surgical intervention, as they are considered benign.[37]

Pseudoaneurysms occur in an AV graft after venipuncture when a leak develops and a hematoma forms outside of the graft. Increased swelling at a graft site, especially large enough to threaten the viability of overlying skin, should raise suspicion of a pseudoaneurysm. This will need surgical correction. If a pseudoaneurysm is inadvertently punctured during dialysis or otherwise, hemorrhage control can be very difficult.[37]

A steal syndrome may develop in which the required arterial blood for the distal limb is diverted through the fistula. That extremity develops ischemia. An arterial stenosis or thrombosis can also occur, limiting the supply of oxygenated blood to the distal tissue. The patient complains of a cold extremity, pallor, numbness, weakness, or severe pain. The resultant ischemia can cause permanent nerve and tissue damage in hours, and emergency surgical intervention is needed.[43]

High-output congestive heart failure (CHF) can also occur after placement of an AV fistula for hemodialysis.[44] AV fistulas have high flow rates. In such patients, the AV fistula flow rate exceeds 2000 mL/min.[45,46] In these cases, a flow reduction surgery or closure of the fistula is indicated.[47,48] The effect of an AV fistula on preload and high-volume flow on

Table 2: Opioid Use in ESRD Patients[57-59]

Opioid	Metabolism and Excretion	Dosing Recommendations
Morphine	The 6-glucuronide metabolite, which is very potent, can accumulate and cause respiratory depression. The 3-glucuronide also accumulates, and is thought to mediate CNS excitation.	Reduce the dose and the frequency of dosing. Beware of many different available formulations.
Hydromorphone and hydrocodone	Hydrocodone is metabolized by the liver into hydromorphone, which in turn is metabolized to the 3-glucuronide. This molecule has no analgesic activity, but is neuro-excitatory. Hydromorphone and its metabolites are renally excreted and easily dialyzable.	Use lower doses and less frequent dosing as a starting point. Beware of the acetaminophen component of the hydrocodone preparations.
Oxycodone	It is metabolized by the liver to several inactive and one active compound (oxymorphone), all of which are renally excreted.	The data on ESRD patients is scant, and it should be used with caution. It has higher abuse potential. Beware of the acetaminophen component in some preparations.
Codeine	It is mainly metabolized to the 6-glucuronide, and both compounds are renally excreted. Cases of excessive sedation due to drug accumulation have been reported. Codeine is not dialyzable.	Codeine should be avoided in ESRD patients.
Methadone	It is eliminated by both the kidneys and the feces. There are no reported cases of adverse effects in ESRD patients.	There is limited information on safety and dosing in ESRD.
Fentanyl	Fentanyl is metabolized by the liver to norfentanyl. Cases of respiratory depression due to accumulation of drug have been reported.	Use with caution.
Meperidine	Meperidine and its metabolites are renally excreted. The most toxic metabolite is normeperidine. Its accumulation results in CNS excitation and seizures.	Do not use in the ESRD patient.

the heart can be demonstrated at the bedside. Branham's sign, also known as Nicoladoni-Israel-Branham sign, is the development of bradycardia when an AV fistula is manually compressed. When the compression of the AV fistula is released, the bradycardia resolves.[49]

Problems During and After Hemodialysis

Hypotension. Hypotension after dialysis may result in the patient being sent to the ED for evaluation. Several factors may be causative, including excessive ultrafiltration during dialysis, which is treated with cautious isotonic crystalloid infusion. Other factors related to HD can contribute to hypotension, including using dialysate fluid warmed to 37° C, acetate-containing dialysate, and recent food ingestion.[37]

Hypotension before or after dialysis may also imply a more serious disease. Sepsis is an obvious concern due to both an impaired immune system and venous access devices such as central catheters. Vasopressors, such as dopamine or norepinephrine, may be required if the hypotension persists after rational fluid administration.[50]

Bedside ultrasonography is helpful to exclude pericardial tamponade, although many renal patients will have pericardial effusions that are not the cause of hypotension (see above).[51] Ultrasonography can also be used to identify volume status, and serial measurements of the IVC can guide fluid administration.

The exposure of the hemodialysis patient to heparin, as well as other clotting disorders, predisposes them to hemorrhage from several sites. Common sites of hemorrhage resulting in hypotension are the gastrointestinal tract, retroperitoneal space, and soft tissues. Complaints of abdominal pain or musculoskeletal pain in the setting of hypotension will require an investigation for occult hemorrhage.

Finally, cardiogenic shock, myocardial infarction, dysrhythmias, and hyper- or hypokalemia may also be the etiology for hypotension. Anaphylactic reactions due to dialysis have also been reported.[52]

Disequilibrium. Disequilibrium syndrome usually occurs after the first episode of hemodialysis in patients newly diagnosed with renal failure, when uremia is pronounced. The rapid removal of urea and other ions produces an osmolar gradient between the brain and plasma, with resultant cerebral edema. Patients present with nausea, vomiting, muscle cramps, altered consciousness, seizures, and focal neurologic signs that mimic ischemic stroke.[53,54] The required evaluation, therefore, can be quite extensive. Cerebral edema may be identified on CT scan or MRI. These patients may require admission to the intensive care unit.

Peritoneal Dialysis (PD) Emergencies

Insertion (exit) site infection is identified by the presence of purulent drainage at the site of the PD catheter. Redness may be a sign of infection, but also could be a localized skin reaction to recent instrumentation or trauma.[55] These infections often spread to the tunnel and into the peritoneum, resulting in peritonitis. *Staph. aureus* and *P. aeruginosa* are often the culprits. The infections can spread quickly and, therefore, must be aggressively treated.[55] Oral antibiotics for a minimum of two weeks, after site cultures, are indicated.[55]

Peritonitis remains a leading cause for mortality in PD patients. It is also a major reason for patients switching to HD. A cloudy effluent is presumed peritonitis, with or without the presence of abdominal pain.[55] Upon examination of the effluent, a WBC count greater than 100/µL, and at least 50% PMNs is the cutoff used to diagnose peritonitis. Make sure that the dialysate has at least an hour, but ideally two hours, of dwell time. In cases of shorter dwell times, the percentage of PMNs, regardless of the total WBC count, is a better indicator of peritonitis.[55] The gram stain is often negative, and cultures of the effluent, along with blood cultures, should also be sent.

Start empiric antibiotics in the ED.[55] Current guidelines recommend intraperitoneal (IP) antibiotics over IV antibiotics. Coverage should include both gram-positive and gram-negative organisms.

Pain Management in the ESRD/Dialysis Patient

About one-third of the dialysis patients receive pain medications, some of them chronically, and many of them in the ED. Pain is a common and multi-factorial complaint in ESRD patients.

Initial analgesia in those with mild to moderate pain should start with acetaminophen.[56] Nonsteroidal anti-inflammatory drugs (NSAIDs) can be used concomitantly. Due to the potential gastrointestinal and renal effects of the NSAIDs, make sure the dosing is restricted in time and amount.[57]

Opioid analgesics are generally recommended for moderate to severe pain.[57] Care must be taken when choosing and dosing opioid analgesics. Impaired clearance can lead to accumulation of the parent compound and active metabolites. (*See Table 2.*)

Drugs to Avoid in ESRD

Although it is widely taught that succinylcholine should be avoided as it increases serum potassium, it appears that this elevation only occurs in patients who have up-regulated receptors, such as those with neuromuscular diseases. In a retrospective search of hyperkalemic patients undergoing anesthesia, there were no adverse events. This study was small, locating only 38 patients with elevated serum potassium.[60] Given the lack of a large study, it is best to avoid succinylcholine when a known hyperkalemic patient needs to have rapid sequence induction.

Nitroprusside is a very potent antihypertensive agent. Its metabolites are renally excreted and will accumulate in renal failure. If using nitroprusside, monitor the patient closely for signs of toxicity and try to wean the medication as soon as feasible.

Conclusions and Take Home Points

HD and PD patients have many complications that require ED visits and management.

Among the electrolyte derangements, hyperkalemia is a major cause for morbidity and mortality.

Infectious complications are many. Specifically, peritonitis must be a consideration in PD patients with either a change in effluent color or abdominal pain. Line infection is also a common source of infection in those with tunneled catheters.

Metabolic bone disease has been decreasing in incidence and prevalence, but is still a common cause for fractures and a source for pain in ESRD patients. In managing moderate to severe pain, remember that most opioid analgesics require dosing modifications to prevent adverse drug effects.

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Physician CME Questions

1. Which statement best describes hyperkalemia and the ECG in the ESRD patient?
   A. Hyperkalemia should only be treated when there are ECG changes.
   B. Some of the ECG changes include peaked T waves, a narrowed QRS, and a sine wave.
   C. Hyperkalemia causes a stepwise progression of changes in the ECG that are very predictable.
   D. Hyperkalemia causes ECG changes not only depending on the magnitude but also the rapidity of the elevation.

2. Which pair of intervention for hyperkalemia and mechanism is *incorrect*?
   A. sodium bicarbonate: enhances the elimination
   B. calcium salts: antagonizes cell excitation
   C. albuterol and beta agonists: intracellular redistribution
   D. insulin and glucose: intracellular redistribution

3. Which of the following parenteral agents should be avoided in dialysis patients when treating hypertensive emergencies?
   A. labetalol
   B. nitroprusside
   C. nitroglycerin
   D. nicardipine

4. Which of the following electrolyte abnormalities does not require emergent HD when the patient is acutely symptomatic?
   A. hypermagnesemia
   B. hyperphosphatemia
   C. hypocalcemia
   D. hyperkalemia

5. Which of the following treatments should be avoided in the treatment of hypocalcemia in the HD population?
   A. calcium phosphate
   B. calcitriol
   C. calcium gluconate
   D. calcium carbonate

6. All of the following are sonographic signs of pericardial tamponade *except*:
   A. right atrial early diastolic collapse
   B. increased right ventricular size
   C. paradoxical septal wall motion
   D. IVC collapse with inspiration

7. The most common reason for ED visits for ESRD patients with metabolic bone disease is:
   A. pain and fractures
   B. pain and secondary hyperparathyroidism
   C. pain and hyperphosphatemia
   D. pain and a Brown tumor

8. Complications of HD access ports include all of the following *except*:
   A. infection with sepsis
   B. thrombosis of the AV fistula or the graft
   C. low-output heart failure
   D. bleeding

9. How is peritonitis diagnosed in a PD patient?
   A. a clear fluid
   B. an effluent WBC count greater than 250/µL
   C. a PMN percentage greater than 75%
   D. a PMN percentage greater than 50%

10. Common causes of hypotension during or after HD include which of the following?
    A. excessive ultrafiltration, sepsis, bleeding, tamponade
    B. sepsis, infection, overdose, anemia
    C. excessive ultrafiltration, bleeding, disequilibrium syndrome, tamponade
    D. sepsis, bleeding, tamponade, withdrawal

Emergency Medicine Reports

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AHC Media

Pediatric Spinal Fractures

Clinical Vignettes

You are working on a busy summer Friday night when you get a medic call on the radio that you will be receiving three children from the same motor vehicle crash (MVC). There was moderate damage to the vehicle, airbags did deploy, and there were no fatalities at the scene. Five minutes later, you have the following patients, in full spinal precautions, in your ED:

1. The driver, a 16-year-old boy who was appropriately restrained in this head-on collision at approximately 40 mph. He has two facial lacerations, distal forearm swelling to his left arm, and is covered with shattered glass. He complains of left arm pain only. You ask yourself if you can clear his cervical spine clinically with his arm injury.

2. A 5-year-old girl who was an unrestrained rear seat passenger at the time of the accident. She was found in the front seat on the passenger side. It is unclear if she had loss of consciousness. She has a hematoma on her forehead and a few small facial lacerations/abrasions from broken glass. Otherwise, her physical exam is normal, she has a GCS of 15, and is tearful. You ask yourself how you're going to clear this patient.

3. A 10-month-old boy presents who was appropriately restrained in a rear-facing car seat. Since he has been with the medic, he has been awake, alert, and age appropriate. They were unable to appreciate any injuries. He arrives in his car seat with towel rolls placed on either side of his head. You wonder how you will be able to keep him immobilized and what imaging you will need to do on this young child.

Introduction

Assessing pediatric patients for possible spinal injuries requires a thorough understanding of common injury patterns, the basics of imaging techniques, and appropriate and timely management. Children who have been immobilized after trauma present a special challenge for the emergency medicine physician. While the incidence of clinically significant spinal injuries is known to be quite low, the morbidity from a missed injury can be devastating. And, although imaging is easy to order, the feasibility of such studies, especially in an inconsolable, non-verbal child, is more daunting. Additionally, as we enter an age of increasing awareness of potential risks from radiation exposure, the management of these patients becomes even more difficult. This article examines the essential differences between the pediatric and adult spine that make imaging decisions differ in this population, reviews common injury patterns that occur in the pediatric spine, and discusses decisions regarding evaluation that neither over- nor under-utilize imaging modalities. SCIWORA will not be reviewed in this article.

Critical Appraisal of the Literature

A literature search of pediatric spinal fractures was undertaken using Ovid MEDLINE (1948-present). An initial query returned more than 200 articles,

Executive Summary

- Pediatric cervical spine injuries are rare. They have a high association with high-risk mechanisms, concomitant injuries, and mortality.
- Upper cervical spine injuries are more common in young children, an area that can be difficult to view on plain films. Limited CT scans of the upper cervical vertebrae can be helpful in evaluating this area for injuries.
- MRI can be useful in continuing the spinal evaluation in obtunded patients, but there is no evidence to suggest that it replace plain films or CT scans as a screening test. If a small child is splinting his or her neck, has neurologic symptoms, or you are concerned that there is an injury, further imaging in the form of MRI should be pursued.
- Because adolescents have similar cervical spine anatomy to adults, it is acceptable to use adult criteria to clear a cervical collar. For younger children, more judicious use of imaging is needed.
- Despite their inability to communicate verbally, children younger than 3 years of age can have their cervical collar removed without imaging. It requires very careful clinical judgment and a thorough clinical exam and understanding of the mechanism of the trauma.
- Children need to be awake and alert before removing a cervical collar, even in the setting of a negative CT, to avoid missing a ligamentous injury.

of which 89 are included for the reader's reference. A specific search for Cochrane systematic reviews in the last 10 years revealed one review regarding pediatric spinal fractures. Both the American College of Emergency Physicians' and the American Academy of Pediatrics' guidelines were searched for relevant material; however, no guidelines were found. International materials were searched as well, with the discovery of a United Kingdom guideline.

The vast majority of literature consists of case reports and retrospective reviews with case-cohort design. There are numerous studies regarding the use of clinical prediction rules to differentiate those who need imaging after trauma; some use modified adult protocols and some are based on logistic regression models at a single center. The single-center studies regarding sensitivities and specificities of plain films vs. clinical assessments vs. CT scans vs. MRIs reveal highly variable results. These studies were done at pediatric trauma centers where patient populations, resources, and personnel may differ from those of a community practice setting.

Epidemiology

Cervical Spine. Pediatric cervical spine injuries are rare, most often associated with high-risk mechanisms, more commonly occur in the upper spine, have high degrees of association with other injuries, and a high mortality rate. The incidence of cervical spine injury in children is quite low, ranging from less than 1-2% of total pediatric trauma victims in most studies; higher numbers found in the literature usually include less serious injuries such as paravertebral muscle strains.[1-5] Fractures that are isolated or in combination with dislocations make up the vast majority of injury patterns regardless of age, with isolated ligamentous injury occurring in 10-20%.[2,5] Although there are a few outliers in the literature, it is commonly held that cervical spine injuries make up a greater proportion of spinal injuries in children (60-80%) than they do in adults (30-40%).[3,6-8] Younger children (younger than 8 years old) have a higher proportion of cervical spine injuries than older children.[6]

MVCs, including pedestrians and cyclists struck by vehicles, are the leading mechanisms for cervical spine injury (CSI) and are responsible for 29-66% of these injuries, followed by sports injuries, falls, and non-accidental trauma (NAT).[1-4,6,7,9,10]

As would be expected by the forces of the different mechanisms, the incidence of cervical spine injury from a motor vehicle accident is 3% vs. 0.8% for falls.[2] It is apparent, however, that the inappropriate use of restraints is a huge risk factor for CSI. In studies in which restraint usage was documented, up to 80% of pediatric CSI patients were inappropriately restrained.[2,7,9,11] There is also some evidence in the literature that three- and four-point restraints may immobilize the torso so well as to cause transmission of the deceleration forces to the neck, causing hyperflexion and proximal cervical spine injury.[12] Unlike MVCs which are a consistent cause of cervical spine injury throughout all age groups, other mechanisms are more age-dependent and based on the activities of a child; sports injuries are more common in adolescents, while falls and NAT are more common in younger children.[2]

The type of cervical spine injury and mortality are age-dependent. Although many studies use different age cut-offs, making direct comparisons difficult, there is a preponderance of evidence showing that younger children are more prone to higher cervical spine injuries and higher mortality from these injuries.[1,2,5,7,9,13] Approximately two-thirds of CSI in children younger than 9 years of age involved the upper cervical spine (C1-C4), while in the older pediatric age groups

upper CSI occurs in half of the patients. Approximately one-half of all children who present with CSI have concomitant injuries, and this increases to up to 80% if one looks only at MVCs. The majority of these injuries, up to 78%, are head injuries.[5,7,9,13] Interestingly, when looking at all head-injured pediatric patients, the incidence of CSI is quite low, at 2.5%.[14] The preponderance of other spinal injuries is similar throughout the different age groups, in which the incidence of contiguous injuries ranges from 16-34% and non-contiguous involvement is approximately 7-9%.[9,15] In one study, neurologic deficits were found in 52% of patients with CSI, and 24% had complete deficits.[13] The mortality rate for CSI in children ranges from 16-27%, depending on the population studied, most of which are secondary to MVC.[2,5,7] This rate appears to be higher in younger children and has been reported to be as high as 48%.[2,7,10]

Pathophysiology

The age-specific differences in incidence, injury patterns, and mortality are easily understood with a thorough explanation of the pediatric cervical spine anatomy. The pediatric spine changes greatly in the first 15 years of life, but for the purposes of most studies, the age of 8 years is chosen as a cutoff between a "younger" child's spine vs. an "older" child's spine. This is reflected in a more adult pattern of injury seen in children older than 8 years of age. One of the more notable differences is the greater degree of ligamentous laxity in the younger child's spine.[16] The spinal column of an infant can be stretched up to 5 cm without ligamentous rupture (the spinal cord can only tolerate 6 mm of stretch).[17] Due to a relatively larger mass and volume of the head in comparison to that of the torso, and poorly developed neck musculature, the fulcrum of flexion is much higher in younger children: C2-C3 in infants, C3-C4 at 5 years of age, C4-C5 at 10 years of age, and C5-C6 by adulthood.[16] The facet joints in young children are more horizontally oriented, allowing for increased movement/subluxation between vertebrae; the degree of angulation changes from 30° at birth to 60-70° by adolescence.[18] Uncinate processes, which decrease lateral and rotational movement, are virtually nonexistent before the age of 10 years, and the anterior vertebral body is wedged. Occipital condyles are small, allowing for greater movement at the O-C1 junction.[18,19] The sum of all of these differences allows for increased mobility of the spinal column in young children and explains the increased incidence of upper cervical injuries, as well as subluxations and SCIWORA, when compared to adults.

Upper Cervical Spine Injuries

The pattern of injury in children is different than in adults, with upper cervical fractures and subluxations predominating. Injuries such as atlanto-occipital subluxation can cause a great deal of neurologic injury with few clues on bony imaging (usually an MRI is required). Upper cervical fractures seen include odontoid fractures, Hangman's fractures, and Jefferson fractures. In older children greater than 8 years of age, adult-pattern fractures occur with greater frequency.

Atlanto-occipital Dislocation. With shallow occipital condyles, there is a great degree of movement in the pediatric spinal-cranial junction that can allow for dislocation of the atlanto-occipital joint. These injuries can be neurologically devastating, as the brainstem is commonly injured. Although once thought to be universally fatal, with rapid cervical spine immobilization and vastly improved trauma care, children are surviving with greater frequency. In one study of 1098 patients with cervical spine injury, the mortality rate for atlanto-occipital dislocation was 48%.[4] In another case series of 2066 cases of spinal injury (already a small percentage of the pediatric trauma population), 16 cases of atlanto-occipital dislocation were studied. Thirteen of these patients were injured seriously enough to require intubation in the field, and 11 died.[20] These injuries are rare and occur with severe mechanisms.

Atlanto-Axial Subluxation. The next joint down from the atlanto-occipital joint is the atlanto-axial junction. This is a unique joint because of the presence of the odontoid. When there is subluxation at this joint, it is usually rotatory, and injury to the spinal cord occurs because of the space limitations in the spinal canal. In the spinal canal at this juncture, the odontoid process, the spinal cord, and the subarachnoid space each take up one-third of the space. The transverse ligament holds the odontoid in place but can be ruptured if rotation exceeds its normal limits, leaving the spinal cord at risk. These injuries are exceedingly rare and occur in very young children after trauma, or with nontraumatic mechanisms in children with Down syndrome or os odontoideum.[21]

C1 Fracture. The first cervical vertebra is at risk for a burst fracture with axial loading, also known as a Jefferson fracture. Common mechanisms include diving injuries or rollover MVCs in which the patient's head strikes the car top, which are more common in older children.[22] The spinal cord rarely is damaged in these instances, as the burst fracture widens the spinal canal. However, this injury can be incredibly unstable depending on whether the transverse ligament has remained intact or has been ruptured.[19]

C2 Fractures. There are two commonly described fractures of the second cervical vertebra: the hangman's fracture and the odontoid fracture. The hangman's fracture describes a break at the pars interarticularis of C2, between the superior and inferior facets. This injury occurs with either hyperflexion coupled with distraction or extension coupled with axial compression.[23,24] This injury is unusual in children younger than 10 years of age, as the odontoid usually breaks first in this younger age group. If it does occur in younger children, child abuse should be

Table 1. Clinical Prediction Rules

Characteristic	Jaffee 1987	Lee 2003	Canadian C-Spine Rule 2001	NEXUS 2001	PECARN (Leonard)
Study Type	Retrospective review	Retrospective	Prospective	Prospective	Case-control study, retrospective
Patients with CSI/ Total Patients	59/206	?/127	8924	30/3065	540/2774
Age Distribution	0-3: 58 (28%) 4-12: 87 (42%) 13-16: 61 (30%)	< 8: 127 (100%)	All > 16	0-2: 88 2 to < 8: 817 8-17: 2160	0-2: 27 (5%) 2 to < 8: 140 (26%) 8 to < 16: 373 (69%)
Factors	Neck pain or tenderness	Neck pain	Midline cervical tenderness	Midline cervical tenderness	Neck pain
	Direct trauma to the neck	Focal tenderness or physical signs of neck trauma	Immediate onset of neck pain (vs delayed)	Intoxication	Conditions predisposing to CSI•
	Limitation of neck mobility	Distracting injury	Not ambulatory	Distracting painful injury	Torticollis
	Abnormalities of strength/sensation	Abnormal neurologic exam	Parasthesias	Focal neurologic deficit	Focal neurologic findings
	Altered mental status	Transient symptoms suggestive of SCIWORA	Not simple rear-end MVC†	Altered mental status	Altered mental status
		High risk mechanism*	Dangerous mechanism^		High risk mechanism‡
		Substance use or unconsolable child	Unable to actively rotate neck 45°		Substantial torso injury
		Significant head/ neck trauma			
Sensitivity	98	ND		100 (88-100)	98 (96-99)
Specificity	54	ND		20 (18.5-21)	26 (23-29)

* High-speed MVC, fall greater than body height, bicycling/diving accident, forced hyperxtension injuries, acceleration-deceleration injuries of head

^ Fall from > 3 feet/> 5 stairs, axial load to spine, MVC high speed (> 100 km/h), rollover, ejection, motorized recreational vehicle, bicycle crash

† Simple rear-end MVC excludes: pushed into oncoming traffic, hit by bus/truck, rollover, hit by high-speed vehicle

• Down syndrome

‡Diving, MVC head-on collision, rollover, ejected, death in crash, speed > 55 mph

considered as a possible cause.[23] This injury will widen the canal, so neurologic sequelae are uncommon.[23]

Odontoid fractures in young children are different injuries than in adults. The fracture line usually occurs through the weaker subdental synchondrosis (which can be seen up to 11 years of age) that lies below the vascular supply to the distal dens. Thus, while fractures in adults can lead to nonunion and pseudoarthrosis after the blood supply has been compromised, pediatric fractures heal with fewer complications.[19] The distal odontoid will usually displace anteriorly but angle to the posterior due to the transverse ligament of C1. Potential mechanisms for this injury include severe falls and motor vehicle accidents, rapid acceleration/deceleration, or blows to the face.[23,25] In small studies of young children with spinal fractures, this type is thought to be one of the more common types; it must be emphasized that these injuries are rare in the general population.[22]

Lower Cervical Spine Injuries. Lower cervical spine injuries and

their mechanisms in children are similar to those in adults. Compression fractures of the vertebral bodies can occur with flexion and/or axial loading. Facet fractures and dislocations are more often caused by hyperflexion.[17] These types of injuries predominate in the older pediatric group and are often related to sports injuries or MVCs.[17]

Prehospital Care

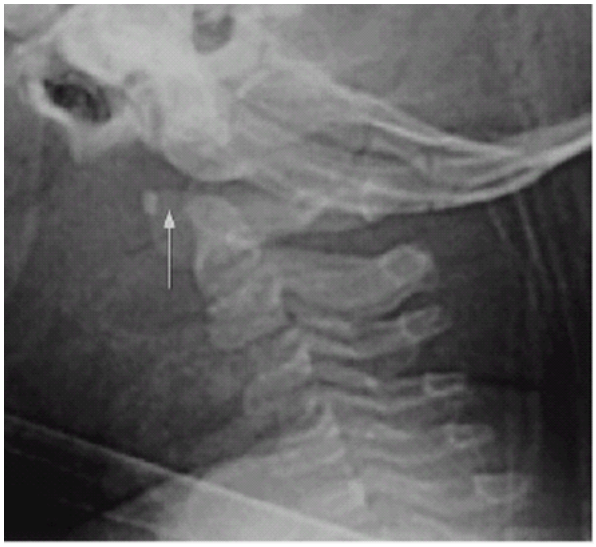
The prehospital care of a child in whom there is concern of a spinal injury consists of achieving neutral positioning and immobilizing. Current EMS protocols call for immobilizing patients if there is concern that there may be spinal injury either by mechanism or assessment. Although immobilization during transport is the standard of care, there are no clinical trials that compare transport with and without spinal immobilization.[26] Immobilizing the cervical spine is not without risks either; it can restrict normal breathing and can lead to aspiration, increased pain, increased intracranial pressure, increased combativeness, and pressure sores.[26,27]

Adolescent patients can be treated similarly to adults, with a rigid cervical collar and backboard. Cervical collars should be placed first, after which the child can be log-rolled onto the backboard. Cervical collar placement alone does not prevent exacerbation of spinal injury during transport; a backboard must be used as well.

An appropriate-sized collar is critical. It has been shown in multiple studies that using proxy devices such as towel rolls, foam rolls, or sandbags in place of cervical collar is not effective in preventing movement.[27] Children who are transported in their infant carriers with towel rolls to prevent lateral movement are not in "spinal precautions." To best limit movement, children need to be in a rigid collar, on at least a short spinal board, with tape securing their head.[28]

The pediatric anatomy makes neutral positioning different than that of adults. An adult patient may need towels under the head for the neck to remain in neutral position while in a cervical collar on the backboard. The small child, with a relatively larger occiput, requires either a special transport backboard with a depression for the head or, if that is not available, padding placed under the shoulders down to the lumbar spine to maintain the neutral position.[26] Criteria for which patients require cervical spine immobilization are a matter of some debate. Some studies demonstrate clearance algorithms that can be used in the field, while others believe that clearance should only be done once the patient is at the hospital. This is an area of ongoing research.

Figure 1. **Atlantodental Interval in a Child**



Lateral cervical spine film of a 2-year-old child. Although the atlantodental distance is wider than what would be considered normal in an adult, it is, in fact, less than 5 mm, or within the normal range for a child.
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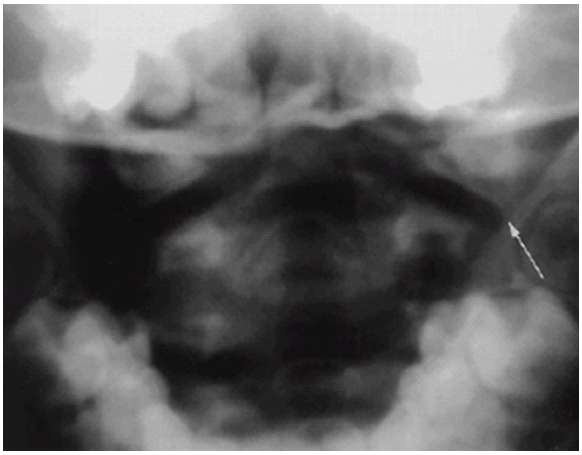
ED Evaluation

Physical Exam. There are two main areas of focus for the practitioner assessing a patient for potential spinal injury: an examination of the spine itself and careful neurologic assessment. The physical exam of a child with a potential cervical spine injury can be accurate, provided that the child has a normal mental status and that he or she is calm and cooperative. This may require a delay in decision-making as one waits for parents to arrive or the child to acclimate to the environment. If the child is awake, alert, and calm, the cervical collar can be gently opened while the practitioner assesses for midline tenderness, step-offs, or other deformities. The neck should be examined thoroughly anteriorly, laterally, and posteriorly for any ecchymosis, lacerations, or abrasions. If the child has no tenderness, the practitioner can then assess for range of motion. Without assistance, the child should be able to flex and rotate in both directions without pain. Younger children may need to have a visual stimulus, such as a light on either side, to encourage them to move their head. The child should not be assisted with head movement. If the child will not move in one direction, this should be taken as an indication of spinal injury.

Figure 2. Pseudospread of the Atlas on the Axis



In this odontoid view, the pseudospread of the atlas on the axis is visualized. Up to 6 mm of "spread" is considered normal in the developing spine and note that despite the spread, the lateral masses are symmetric and equidistant to the dens.

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If there is concern for thoracic spine injury, inspection and palpation are the only ways to assess the back, as range of motion of this area of the spine is severely limited (and protected) by the rib cage. The patient should be exposed so that the entire spine can be visualized and then a methodical exam of each vertebra can begin. Tenderness, deformities, and step-offs should be assessed at each level of the thoracic spine. Any anomaly should lead the practitioner to perform imaging. For lumbar spine injury from a single mechanism such as an MVC or fall, the assessment should be a continuation of that for the thoracic spine. Children who present with low back pain and have potential overuse injuries to their spine should be evaluated with inspection, palpation, and range of motion (flexion/extension) of the lower spine.

A full neurologic exam is mandatory for all children with concern for spinal injury, whether it is acute or chronic in nature. This includes a motor exam for strength and a sensory exam of all dermatomes, including pinprick if the child is old enough to cooperate. Reflexes should be measured in upper and lower extremities as well. The use of routine rectal exams is called into question in pediatric traumas as they are neither sensitive nor specific for spinal injury.[29,30]

Imaging Options

Plain Radiography. In recent years, the sensitivity of plain films in evaluating cervical spine fractures has been found to be inferior to CT; CT is becoming a more common first-line test in adults.[31] The increased ionizing radiation exposure from CT makes this test less than ideal for the growing child. The plain radiograph series (A/P, lateral, and odontoid views) is criticized for its variable sensitivity; however, it continues to be used as a screening device in many institutions.[31,32] Depending on the study, the sensitivity of plain films can range from 50-100%; most range between 79-94%.[22,33-36] Because the prevalence of cervical spine injury is so low, even when the sensitivity is 50%, the negative predictive value of radiographs is high at 97% (95% CI: 97-99).[33] The odontoid view is controversial in small children, as they are unable to cooperate. As it does not add much information in this age group, it can be forgone in the assessment of children younger than 5 years.[37]

Other images have been proposed, such as oblique views and flexion/extension radiographs, but these have not been found to add to the sensitivity of plain radiography.[38-40] There are numerous advantages to plain films, including less radiation exposure, no need for sedation (as might be required for CT), and more rapid evaluation of the cervical spine. The downsides to plain films include the difficulty of obtaining adequate films in the young children. In multiple studies, an average of 1.2 films needed to be performed on every child to get an adequate lateral view.[33,41]

The general approach to the pediatric cervical spine film is not dramatically different than that for an adult film and will not be discussed in depth here. It is vital, however, to understand that there are a few normal variants that can be mistaken as pathology if unrecognized.

- Atlantodental interval (ADI): This interval from the anterior aspect of dens to the posterior aspect of the anterior ring of the atlas should be 5 mm or less. In adults, only 3 mm or less is considered normal.[18] (*See Figure 1.*)
- Pseudospread of atlas on the axis: Until the age of 4 years, up to 6 mm displacement of the lateral masses can be a normal finding. (*See Figure 2.*)
- Apical odontoid epiphysis: This normal variant, which has potential for being mistaken for a fracture, can be visualized in 26% of children between 6 and 8 years of age.
- Prevertebral tissue prominence: At C3 in children, it is normal to see up to 5-6 mm of prevertebral tissue.
- Anterior wedging: This is most noticeable at C3 and can be confused for a compression fracture. Up to 3 mm of wedging is normal in a young child's spine.[18] (*See Figure 3.*)

• Pseudo-subluxation: This is most prominent at C3-C4 and can be present in 46% of patients younger than 8 years of age.[42] The Swischuk line is useful in these cases to differentiate normal variation from pathology. (*See Figure 3.*)

• Absence of lordosis: While potentially indicative of injury in adults, the tight ligamentous attachment between the skull and C1 can lead to an absence of lordosis that is completely normal in children.

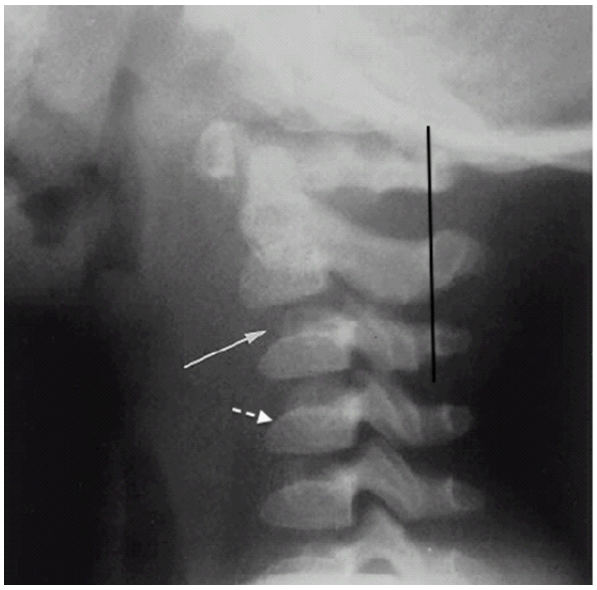
• Ossification centers: The development of the vertebrae is such that there are numerous cartilaginous ossification centers within each vertebra. These cortical breaks can be easily distinguished from pathologic fractures in that they are smooth and regular with sclerotic lines and they are found in predictable locations.[18,19]

CT Scans. CT scan of adult patients at high risk for injury has been proven to outperform plain film radiology as the initial screening test after trauma.[31,43,44] Due to the anatomic and physiologic differences in children, great caution should be used before applying similar rules to children. Although potentially more sensitive for identifying osseous injuries in children as well, the risks of ionizing radiation exposure, potential adverse reaction to sedation if it is needed, and increased cost should prompt the emergency medicine practitioner to be more judicious in its use.[40] As previously described, the risk of ligamentous injury, which can be missed on a CT scan, is much higher in younger children, decreasing this test's sensitivity for injury and, thus, making the increased risk of exposure less justified.[16,45] In fact, in a National Pediatric Trauma Registry review over 10 years, 50% of children with symptomatic spinal cord lesions had no radiographic abnormality. It cannot be overstated that no imaging takes the place of a careful neurologic examination.[4]

Assessing the absolute risk of this ionizing radiation is difficult, but there are numerous studies that suggest an increase in cancers should be expected from exposure to ionizing radiation from CT scans.[46-48] According to the BEIR VII study, 42 out of every 100 people will be diagnosed with a cancer in their lifetime; one of these 100 people will get cancer from a single dose of ionizing radiation at the level of 100 mSv (BEIR VII).[49] In a study using phantom models, a cervical spine CT exposed the thyroid gland to 59.28 mSv in a 1-year-old model (more than 200 times that received from conventional radiography), with an increased relative risk for development of thyroid cancer of 33.9 (95% CI: 18.6 – 57.2).[50] Thus, although CT scan may have increased sensitivity for bony injuries, there are definite risks involved in its utilization.

MRI. The improved availability of MRI, safety from a radiation perspective, and advantages in identifying soft-tissue injuries raises the question: Should MRI be used to help clear the cervical spines of young children?[51] The downsides, of course, are that these tests are expensive and would require sedation to be performed in the very young trauma patient. MRI's use in the clearance of children with potential cervical spine injuries is only beginning to be studied and, thus far, its routine use has only been studied in children who are intubated. In this group, it has been shown to be useful in allowing for earlier clearance of intubated patients, which leads to decreased ICU stays and medical costs.[52] In adult studies, MRI has been shown to miss osseous injuries and, thus far, no study supports the universal use of MRI as a screening test, nor for it to supplant CT scan and plain radiographs.[45] Clearly, if a child has an abnormal neurologic exam, regardless of whether the CT or plain films are normal, MRI should be obtained.

Figure 3. Pseudosubluxation of C2 on C3 and Anterior Wedging



The white arrow depicts the "pseudosubluxation" of C2 on C3 (white arrow). The line of Swischuk (black line) was developed to help differentiate between pseudosubluxation and real pathology. A line from the cortex of the posterior arch of C1 to the cortex of the posterior arch of C3 should pass, intersect or be < 1 mm away from the posterior arch of C2. Also demonstrated in this image is normal anterior wedging (dashed white arrow)

Clearing the Cervical Spine after Trauma

Many practitioners are hesitant to remove a cervical collar from a potentially injured child without definitive radiologic studies. Unfortunately, this has led to a substantial increase in CT utilization in the population most vulnerable to effects of ionizing radiation exposure.[53] This has occurred despite a lack of evidence that injury incidence has changed or that screening with CT increases detection of injury or decreases length of stay in the department.[54] In this section, possible algorithms for the clearance of cervical spines in children will be delineated, along with a description of the evidence used to create it. There is one important caveat to remember: There is no perfect algorithm for the clearance of a c-spine.[32] Clinical judgment, careful physical examination, and understanding of the different age-based mechanisms of injury of the pediatric spine are essential for appropriate cervical spine clearance. Clinical protocols in any institution should be flexible to minimize over-exposure to ionizing radiation and to allow for physician judgment.

In this section, a pragmatic approach to the literature was used to create some potential guidelines that would hopefully minimize exposure to radiation without missing cervical spine injury. Children will be divided into three groups: adolescents (older than 8 years of age) who have "adult" cervical spines and are able to communicate clearly; school-aged children (ages 4-7 years) who should be able to communicate clearly but have a developing spine; and toddlers/infants (0-3 years) who have undeveloped spines and are unable to communicate clearly. Within each of these groups there are two questions that need to be asked: Can this child be cleared clinically? If imaging is required, what imaging should be obtained?

Adolescents. Clinical criteria are commonly used to clear spines in adolescents, either by NEXUS or Canadian C-Spine Rule criteria.[32,55-57] This is based on work by Viccellio et al, who looked specifically at the 3065 patients (9.0%) in the NEXUS cohort who were younger than 18 years old, and two-thirds of whom were older than 8 years. The authors assert that the NEXUS decision rule found all 30 patients (0.98%) who had CSI, with a sensitivity of 100% (95% CI: 87.8-100%) and an NPV of 100% (95% CI: 99.4-100).[58] Although there are data to suggest that the Canadian C-Spine Rule outperforms the NEXUS rule in adult patients (both with sensitivity and lower radiography rates), a similar high-quality study in pediatric patients has not been accomplished.[59] Many institutions publish their own algorithms for cervical spine clearance that utilize physical as well as historical features, emphasizing the lack of consensus on this topic.[60,61] See Table 1 for a description of commonly referenced cervical spine clearance clinical decision rules.

While the adult literature supports the use of clinical decision rules such as NEXUS and CCR to determine who does not need imaging, there is no consensus regarding the type of imaging required once a patient falls out of the "low-risk group." In an elegant case report and literature review done by Greenbaum et al, once patients fell out of clinical clearance, they were placed into high-risk or low-risk groups.[62] Based on an article by Hanson et al and the Harborview Criteria, they delineated the following factors as being associated with high risk for cervical spine injury: multiple extremity fractures, pelvic fractures, GCS less than 8 or evidence of severe head injury, presence of neurologic deficit, MVC greater than 35 mph, or fall greater than 10 feet.[44] Initial CT is recommended for all patients with any high-risk criteria, age older than 65 years, known degenerative disease, intubated patients, or those with altered mental status. If they are not found to be high risk, a plain film series can be used to rule out cervical spine injury, with secondary use of CT for patients with persistent symptoms, those with injuries visualized on plain films, and those with inadequate plain films. Using these clinical criteria, 0.2% of patients in the low-risk group would have had missed cervical spine injuries. This approach seems exceptionally rational, as CT scans for all potential spinal injury victims (regardless of pre-test probability) would add dramatically to the cost of care for trauma patients, and underutilization of CT would lead to missed injuries. As adolescents have adult spinal anatomy, it seems reasonable to apply these recommendations in this population. Unfortunately, application of adult rules to adolescent trauma means that adolescents would receive the same radiation exposure that adult trauma patients do (three times that of pediatric trauma patients).[63]

For the obtunded or altered adolescent patient, cervical spine clearance falls outside the purview of the emergency medicine physician. While these older pediatric patients would clearly warrant CT of the cervical spine by the above criteria, imaging alone is not enough to allow for removal of a collar on an altered patient. These patients will require admission or transfer to a trauma service where further decisions regarding clearance can be made.[52,64]

School-Aged Children. In contrast to older children and adolescents, younger children provide a significant challenge to the emergency medicine provider, as both their anatomy and their ability to communicate are vastly different. Imaging of all children without any prediction rule has been demonstrated to be ineffective.[41] There are two schools of thought regarding school-aged children: Clinical decision rules are effective; or they are not sensitive enough to make a good decision. Before the large NEXUS work, there were numerous studies that developed sensitive clinical prediction rules to exclude CSI. Jaffe's rule in 1987 performed a logistic regression of 206 pediatric patients and found that any one of the following characteristics warranted imaging: neck pain or tenderness, direct trauma to the neck, limitation

of neck mobility, or abnormalities of strength, sensation, reflexes, or mental status. Sensitivity for this rule was 98%.[65] Lee et al developed another clinical prediction rule similar to Jaffe's, but included an evaluation of mechanism. Children warranted imaging if they were involved in a high-speed MVC, fell from greater than body height, had forced hyperextension, or had acceleration-deceleration of the head. Additionally, this rule included significant head/face injury and inconsolability as factors. This rule also had 100% sensitivity.[61] Both of these rules would lead to a decreased utilization of radiologic studies by approximately one-third.

The NEXUS study emerged in 2001 as the largest prospective validation study for a clinical prediction rule excluding low-risk trauma patients who do not require cervical spine imaging. This was a 21 center study that applied the rule in more than 34,000 patients. In adult patients, this rule proves highly sensitive at 99% (95% CI: 98-99.6%) and would lead to a 12.6% reduction in imaging.[56] Application of this rule for children requires a close look at the pediatric data from this study. In Viccellio's paper applying NEXUS criteria to children, a high sensitivity of this rule was found: 811 patients were younger than 8 years of age (very few were younger than 2 years of age) and NEXUS had a sensitivity of 100% (95%CI: 87.8-100%).[58] The lower end of the confidence interval demonstrates a worse performance of the rule in children, but has been attributed to the lower prevalence of injury. It will be a long time, if ever, before better data are obtained, as the number of patients that would need to be tested is prohibitive. It is important to note, however, that this is the only prospective study looking at NEXUS in this age group.[58] Retrospectively, one trial shows the NEXUS decision rule performing equally well (sensitivity 100%), and one study demonstrated a dramatically lower sensitivity (43%).[57,58,66] Interestingly, the Canadian C-Spine Rule in the latter study seems to have performed better and missed no clinically significant injuries (one spinous process fracture).[57] One can find case reports in the literature that demonstrate a "miss" by the NEXUS rule that would have been caught by the Canadian C-Spine Rule, but it is unclear how to interpret this information.[67] Overall, the Viccellio study, while not perfect, demonstrates the strongest data supporting the use of NEXUS to clear patients in the school-age group.

More recently, the PECARN group performed a large multicenter, case-control study of 540 patients with cervical spine injury and performed multiple logistic regression analyses to develop a clinical predication rule for children. They found the following to be risk factors for CSI in children: altered mental status, focal neurologic findings, neck pain, torticollis, substantial torso injury, conditions predisposing to cervical spine injury (Down syndrome), diving or high-risk motor vehicle crash (head on collision, rollover, ejection from vehicle, fatality in the crash, or speed greater than 55 mph). The sensitivity if a patient has one or more factors was 98% (95% CI: 96-99%), with a specificity of 26% (95%CI: 23-29%). This rule has yet to be validated prospectively.[68]

Unfortunately, these studies only help answer the question of who does not require imaging. As to what imaging is required for this group of verbal children, there remains great controversy. While the sensitivity of plain films in adults has been found in the literature to be so inferior to CT as to preclude its use in moderate to high-risk patients, the data are not as clear in pediatrics. As described above, the exposure to ionizing radiation with CT is such that this modality should not be used indiscriminately in this group. It does seem reasonable, though, to use mechanism of injury as part of the decision-making process. A 7-year-old who falls from standing and arrives to the department in a cervical collar complaining of neck pain with a non-focal exam could probably be cleared with plain films, while a 6-year-old in a high-speed MVC complaining of the same neck pain would likely require more sensitive imaging for clearance. Many groups continue to use plain radiography as a screening tool in young patients, although the evidence behind this is not as good. If the films are abnormal or inadequate, further imaging is required.[60,61,69] In the United Kingdom, clinical guidelines suggest that CT should be reserved only for children with a serious head injury (GCS less than 8), those who have inadequate or concerning plain films, and those who have signs of spinal injury (i.e., parasthesias, weakness).[70] Another option might be limited CT of the neck.[37,69] As the majority of injuries in younger children tend to be in the upper cervical spine, a notoriously difficult area to see on plain films, CT scan of the upper spine may have increased sensitivity while minimizing ionizing radiation exposure.[71,72] In a retrospective study by Garton et al, adding an occiput – C3 view of the neck to plain films increased the sensitivity from 75% to 94%.[73]

Infant/Toddler. For the infant or toddler, decision-making becomes more difficult. The NEXUS study included too few patients in this age group to make any meaningful conclusions.[58] For this group, it is vital to understand the rarity of actual injury, the mechanisms required for serious injury, and the anatomy of the child. Recall that upper cervical spine injuries are much more common in this age group and that there is greater ligamentous laxity, allowing for greater movement of bony elements without causing fractures. While this could cause more trepidation for practitioners, it is again important to emphasize that these injuries are rare. Published data may lead to more imaging as practitioners see the different algorithms created at large pediatric trauma centers for clearing pediatric spines. In one article, neurosurgeons were initially used to clear all potential spinal injuries in patients younger than 3 years. After implementation of an imaging protocol, neurosurgery consultation

went down, but CT scan usage increased as much as 21%.[74] This speaks to CT utilization as an indicator of practitioner discomfort with the pediatric cervical spine. Just as in older pediatric groups, the first decision to be made is whether a child warrants any imaging at all. While there were few patients in the NEXUS study and no small children in the Canadian C-Spine Rule, the lack of consistent verbal communication would likely preclude all but the exam and mechanism portions of these rules anyway.[57,58] This does not mean that a small child cannot be cleared clinically. In one large retrospective study of more than 12,000 blunt trauma patients younger than 3 years of age, up to one-third were cleared clinically.[75] From the remaining two-thirds, they created a logistic regression model that found the following variables to be associated with CSI: GCS less than 14 (3 points), GCS EYE equal to 1 (2 points), MVC (2 points), age greater than or equal to 2 years (1 point). A score of 0 or 1 had a negative predictive value of 99.93% in ruling out CSI. For the five children who would have been missed by this rule, all had clinical or historical findings that led to imaging. Again, these findings help reduce the use of imaging while reminding us that there is no algorithm that will supplant the use of a careful history and physical exam and good physician judgment. Unfortunately, it would also seem to suggest that any child in an MVC warrants imaging. Secondary to the low prevalence, specific details from the MVC were unable to be quantified. Clinical judgment must be used. One might apply the same high-risk criteria from the adult protocols such as the Harborview study. For example, a 2-year-old who is appropriately restrained in a car seat involved in a low-speed MVC with no evidence of injuries is going to have such a low pre-test probability, clearance can be done clinically or with plain films. This use of good clinical judgment is reflected in this study in which one-third of patients were cleared without the use of any imaging. In another study of 606 patients, 459 were cleared clinically or with the use of plain films.[34]

The unconscious patient does not present a decision-making challenge for the ED practitioner; the literature supports the use of CT scanning of the entire neck for these patients.[74,75]

It is clear from the literature that clinicians who see pediatric trauma patients more frequently are much more comfortable clearing a spine clinically.[53,76] Clinicians who see a large number of injured children are likely to have the support systems in their institutions that make for easier pediatric assessments, such as child-friendly environments, Child Life staff, and a dedicated trauma service that may be willing to observe patients. This does not mean that children outside of these centers cannot be cleared clinically. It may mean, however, that decisions to image may need to be delayed slightly to allow a child to calm down, be soothed by parental presence, and become accustomed to his or her environment. A small investment of time can allow for improved decision-making and, therefore, less radiation exposure.

Thoracolumbar Fractures

Epidemiology/Etiology/Pathophysiology. Thoracolumbar (TL) fractures are even less common than cervical spine fractures in children and account for only 0.6-0.9% of all spinal trauma cases. They tend to occur after either high-energy mechanisms such as MVCs and falls or from overuse as in sports-related injuries.[77] Because of the mechanism required, these injuries are far more common in the adolescent age group (12-18 years).[78,79] Injuries to these areas of the spine are often considered together, although it is important to remember that in the thoracic spine, the rib cage provides added support against translation and dislocation.[80] Just as in the cervical spine, the increased laxity of the pediatric TL spine allows for a great degree of movement prior to bony injury that explains the decreased incidence of injury in the pediatric population. Once again, the facet joints are more horizontally oriented and incompletely ossified, allowing for greater movement.[81]

To understand the injury patterns and associated risks, it is important to look at the structure of the developing vertebra. Similar to the long bones of the pediatric spine, the vertebrae have physes and inferior and superior end plates. The physeal cartilage is a weak area of the vertebrae, allowing for Salter-Harris type I injuries. Although more prone to injury, these areas of the vertebrae heal quite well. Unlike in adults in whom the disc is a weak point in the spine that can herniate into the spinal canal after trauma, the hydrophilic disc of the pediatric spine is firm and more resistant to injury than is the vertebral body. Thus, the apophyseal end plate is more likely to herniate into the spinal canal and cause neurologic damage than is the disc.[81] Because of the increased elasticity of the spine, compression forces are more likely to be spread across different levels of the spine; multiple compression fractures are more common in pediatric patients than they are in adults.[77] Just as in the cervical spine, it is important to realize that the amount of force necessary to cause fractures in the TL spine of a young child are high and, as such, the younger the child, the more likely they are to have associated neurologic injury.[80]

Wedge fractures are the most common type of thoracic spine injury seen, although with anterior compression, the nucleus pulposus may also get driven into the vertebral body below.[80] In these cases, one would expect to see loss of the disc space as opposed to wedging.[17] One specific type of flexion injury, known as the Chance fracture, occurs when hyperflexion of the lumbar spine occurs around a seatbelt during rapid deceleration. This leads to a distraction injury of the posterior elements of the spine with or without compression of the anterior elements. These injuries are highly associated with intra-abdominal injuries, and definitive imaging of the abdomen should be pursued.[80,82]

In the adolescent age group, pure axial load and compression may cause posterior movement of the apophysis into the spinal canal. Most compression fractures do not require surgical intervention and do not cause permanent height loss of the vertebra unless the amount of wedging is greater than 30 degrees or there is endplate damage.[80]

Hyperextension injuries are more often seen in the lumbar spine and come from overuse in the adolescent athlete. In adult athletes, back pain comes from disc problems or muscular strains, but in the adolescent athlete, the injuries occur in the posterior elements of the spine.[83] Football players, weight lifters, divers, and gymnasts can suffer fractures of the pars interarticularis (also known as spondylolysis) of L5 (L5-S1) from frequent hyperflexion against a large force. In fact, 40% of athletes with persistent back pain (longer than 3 months) have pars interarticularis defects.[84] Children aged 9-15 years are especially at risk for progressive injury from spondylolysis.[83]

Clinical Evaluation/Imaging. There are very few studies examining the efficacy of clinical exam and imaging in the evaluation of TL spine injury. There are no studies that compare the plain film and CT scan for evaluation of these injuries in children. Just as in the cervical spine, one would expect to see anterior wedging and horizontal facets on the plain films. Plain films of the adolescent spine may show the apophysis starting to fuse with the vertebral body. This can be misconstrued as a fracture, and careful physical exam correlation is required.

Although there is no great study regarding ED evaluation for the TL spine, plain films remain the mainstay of diagnostic procedure in a child with low suspicion for serious injury. Clearly, if multi-system trauma is suspected, CT scans of the chest and abdomen will demonstrate spinal fractures, especially if spinal reconstructions are done. As for who requires imaging, few studies have been done. In one retrospective study in adults, imaging was recommended if patients had any of the following characteristics: back pain or tenderness, local signs of TL injury, abnormal neurologic exam, cervical spine fracture, GCS less than 15, major distracting injury, or evidence of intoxication. The sensitivity of this amalgam of findings is fantastic (100%); however, the specificity is only 11.3%.[85] Again, common sense must prevail in the assessment of children. TL injuries are rare in children, and one must always be aware of the risk of radiation exposure.

Two larger studies in children have been performed. One retrospective study of 192 patients (96 with TL fractures and 96 without TL fractures chosen from a convenience sample from a trauma registry) demonstrated that the sensitivity of the physical exam is 87%.[79] A follow-up prospective study of 228 patients showed that the clinical exam is 81% sensitive for TL spine fracture diagnosis. The injuries among the three patients who would have been missed were one spinous process fracture and two with minimal anterior wedge compression.[86] While there are no definitive guidelines in the literature, a rational approach would be: If there is clinical suspicion due to mechanism or an abnormal physical exam, imaging is warranted. If the patient is undergoing CT scan for other reasons (suspicion for multi-system trauma), evaluation of the spine can be done with that imaging. Otherwise, if there is no suspicion for other injuries, plain films can be used to evaluate for thoracic or lumbar fractures. Young athletes who present with low back pain and have midline tenderness or findings should also have plain films obtained.

Special Circumstances

There are numerous conditions that can lead to increased movement of the cervical spine. Although each congenital anomaly need not be fully understood for the management of spinal trauma in these patients, the emergency medicine practitioner should be familiar with the most common diagnoses. Children with trisomy 21 (Down syndrome) can have increased instability in the upper cervical spine, most commonly between C1 and C2, but also in the atlanto-axial joint. The increased ligamentous laxity should be taken into consideration prior to clearing these patients from a cervical collar. Children with neurofibromatosis can have congenital vertebral body insufficiencies and dysplasias throughout their spine. Clearing from a cervical collar would definitely require CT in these patients. Juvenile rheumatoid arthritis can cause stiffening of the joints and subluxation of C1 on C2 in the first years of diagnosis. There are other numerous and rare congenital anomalies that can cause instability or abnormal development of the spine, including mucopolysaccharidoses, skeletal dysplasias, and Larsen syndrome. Again, the individual diagnoses need not be memorized. It is essential, however, that the practitioner obtain a medical history prior to making final decisions regarding cervical spine clearance. If there is a concern for underlying spinal instability, CT scan should be the first modality of choice. MRI may even be necessary if there is concern for ligamentous injury.[87]

Controversies/ Cutting Edge

As discussed at length in this article, the greatest controversies in managing children with potential spinal injury are who can be excluded with a clinical exam and history and what type of imaging is best. For now, there are decent clinical prediction rules that can be used in children, although not all are prospectively validated. Great care should go into the decision to conduct a CT scan of the entire cervical spine, as this test has some associated risk from ionizing radiation exposure. If spine injury is suspected and radiographs/CTs are normal, MRI is the next test of choice. Because of its low sensitivity for detecting osseus injuries, its high cost, and the need for sedation in young children, MRI is not effective as a first-line

screening test for cervical spine injury in children.

Disposition

After careful evaluation, most children will fall into one of two categories: those with cervical spine injury and those whose cervical spine can be cleared either clinically or with imaging. Those who have been cleared can be safely discharged home with analgesics and strict instructions to return to the emergency department immediately for worsening neck pain, decreased range of motion, numbness, weakness, or any other neurologic symptom. For those children with known cervical spine injuries, evaluation by a spine specialist, either orthopedic or neurosurgical, is required. If the practitioner is at an institution where those specialists are not available, or if he or she feels uncomfortable managing the injury, the child should be transferred to a pediatric trauma center. There are, unfortunately, children who fall out of these two categories: those who have persistent symptoms despite negative imaging. There is no great evidence to help guide practitioners in these situations. Older children with a low-risk mechanism will often be discharged in a firm cervical collar to follow-up in 1-2 weeks for reevaluation. Children who cannot be cleared because of abnormal GCS are observed in the hospital in their cervical collar until mental status normalizes.

Thoracolumbar injuries are slightly different in that there are a few injuries that can be discharged home if pain is adequately controlled. Minor spinous process or transverse process fractures can be managed with bed rest and analgesics. An adolescent child with such an injury may be considered for outpatient management after discussion with a spine specialist and with close follow-up arranged. Clearly, the younger the patient, the more likely that admission will be required. Smaller children should be observed in the hospital for analgesia and to continue evaluation for late-developing abdominal injuries or ileus.

Summary

The evaluation of the pediatric patient for spinal injury can be quite difficult as the practitioner weighs the risks and benefits of all of the imaging options available. It is important to remember that these injuries are rare and that judicious use of imaging modalities is imperative to reduce exposure to ionizing radiation in this most susceptible population. Clinical prediction rules can help reduce the number of patients who require imaging and, although not prospectively validated, can support the use of clinical judgment in assessing children with these potential injuries.

Vignette Conclusion

1. The 16-year-old undergoes an ATLS assessment and is found to have no injuries except for a distal radius fracture and some facial lacerations with some embedded glass pieces. He is left in his cervical collar while his facial wounds are irrigated and closed and his wrist is splinted. After his injuries are addressed, a cervical spine exam reveals no tenderness, his neurologic exam is completely normal, and he is awake and alert with a GCS of 15. His cervical collar is removed and the patient is discharged home.

2. The 5-year-old is brought in awake and alert, although she is tearful and obviously frightened by the events and by her environment. On ATLS evaluation, the only injury found is the hematoma on her head and small facial abrasions. Chest radiography is normal. She does appear to have cervical spine tenderness. Because of her hematoma and questionable loss of consciousness, you decide that she needs a head CT. You order a CT of the upper cervical spine (occiput – C3) along with the head and plain films of the lower cervical spine. These are all normal. After two hours of observation in the ED, the child is calm and cooperative. You remove her collar, and she has full range of motion without any splinting or pain.

3. There are two possible scenarios for this child. First, he is in an MVC with no high-risk mechanism concerns and he was appropriately restrained. As you know that the incidence of cervical spine injury in this age group is exceptionally low, you can assess his neurologic exam and, if normal, carefully remove him from the car seat and observe his movements. If he appears to have neck pain or tenderness on your exam, an appropriately sized infant cervical collar can be placed on him and imaging can be obtained. If you feel uncomfortable with any movement in this child before imaging, the cervical collar can be placed on the infant while he is still in his carrier and then the infant can be carefully extracted from the car seat to perform your ATLS survey. At that point imaging can be considered.

This infant does indeed appear to splint his neck and refuses to move it to the right. A CT scan of the upper cervical vertebrae reveals a dens fracture, and the patient is transferred to a trauma center to be evaluated by a neurosurgeon.

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CME/CNE Questions

1. Which of the following best represents the incidence of cervical spine injuries among pediatric trauma victims?
   A. 1%
   B. 5%
   C. 8%
   D. 10%
   E. 12%

2. Which of the following is the most common concomitant injury in children with cervical spine injuries?
   A. other vertebral injuries
   B. abdominal injuries
   C. extremity fractures
   D. head injuries
   E. pulmonary injuries

3. Which of the following regarding Jefferson fractures is true?
   A. It is a burst fracture of the second cervical vertebra.
   B. The spinal cord is frequently damaged.
   C. It is an axial load injury.
   D. It is more common in younger children.
   E. It usually is a stable fracture.

4. Which of the following statements is true regarding pre-hospital transport of young children?
   A. They often need a towel under their head to keep their neck in neutral position.
   B. Towel rolls and tape can be used to immobilize the cervical spine in a child restrained in a car seat.
   C. There is no risk to the patient by placing him or her in a cervical collar.
   D. If there is no appropriately sized cervical collar, sandbags and towels can be just as effective.
   E. To best limit movement, children should be in cervical collars and strapped to a spinal board to protect their spine.

5. Which of the following is true regarding the physical exam in children with potential cervical spine injury?
   A. To assess range of motion, gentle pressure can be applied to the head to help a child rotate the neck.
   B. Rectal exams provide important information and should be done in all children in c-spine immobilization.
   C. Even small children who are scared of their surroundings can be evaluated clinically for cervical spine injury.
   D. A neurologic exam is not necessary if a patient is getting CT scans of the neck.
   E. Neurologic assessment with soft touch sensation is adequate for assessing for spinal cord injury.

6. Which of the following best describes the use of plain films in evaluation of cervical spine injury?
   A. Because of higher cervical spine injury potential, children younger that 5 years should undergo odontoid views.
   B. Because of the low prevalence of cervical spine injury in pediatric patients, the NPV of X-rays is low.
   C. One of the downsides of plain films is that often, multiple pictures will need to be obtained to get adequate images.
   D. The use of flexion/extension films can enhance the sensitivity of plain films.
   E. The sensitivity of plain films has been universally found to be too low to effectively exclude cervical spine injury.

7. Which of the following best describes the use of CT in the evaluation of pediatric CSI?
   A. CT is more sensitive than plain films and should be used as a screening test for all pediatric patients who can't be cleared clinically.
   B. The risk of cancer from CT scan is too high to use this test in the evaluation of pediatric CSI.
   C. CT scan is sensitive for ligamentous injury.
   D. There are not enough data to suggest that the use of CT contributes to the risk of cancer.
   E. CT is a sensitive test for CSI in children as well as adults and can be used, with caution, in the evaluation of children at high risk for CSI.

8. Which of the following best describes the use of MRI in cervical spine clearance in pediatric patients?
   A. MRI is a far more sensitive test and, due to its lack of ionizing radiation exposure, it should be used as a screening test for CSI in children.
   B. MRI is sensitive for bony injury.
   C. For intubated patients, MRI has been shown to decrease time to cervical spine clearance and decrease ICU stays and medical costs.

D. Young patients should be able to undergo an MRI of the cervical spine without sedation.

9. Which of the following statements is true regarding the application of clinical decision rules to pediatric patients?
   A. Just as in adults, the Canadian C-Spine Rule is more sensitive and specific in excluding cervical spine injury in children.
   B. There were enough patients younger than 3 years of age in the NEXUS study to prove its sensitivity in this population.
   C. NEXUS is the only clinical decision rule to be prospectively validated in pediatric patients.
   D. Because there are no clinical prediction rules in younger children, all children younger than 3 years require imaging to clear their c-spines.
   E. There are numerous criteria physicians can use to exclude cervical spine injury, but nothing can replace good clinical judgment.

10. Which of the following is most accurate regarding cervical spine clearance in children?
   A. Once a negative CT scan is obtained, the cervical collar can be removed on an altered patient.
   B. Clearance and imaging decisions need to be made during the initial assessment of stable children.
   C. There have been no studies that looked at risk factors for cervical spine injury in children younger than 3 years.
   D. CT scans should never be used in young children due to the risk of ionizing radiation exposure.
   E. Allowing the stable child to relax in their environment prior to making imaging decisions is acceptable.

11. Which of the following statements regarding thoracolumbar (TL) spinal injury is true?
   A. TL spinal injury is more common in children than adults.
   B. TL spinal injury can occur with a minor mechanism.
   C. TL spinal injury is more common in younger children than in adolescents.
   D. The increased laxity of the pediatric spinal column allows for more TL spinal trauma.
   E. The thoracic and lumbar spine have different injury patterns secondary to the stabilizing nature of the rib cage.

12. Which of the following is true regarding trauma to the TL spine in pediatric patients?
   A. Chance fractures are the most common type of lumbar wedge fracture.
   B. Chance fractures are highly associated with abdominal injuries
   C. Most compression fractures will not lead to loss of vertebral height unless the endplate is involved.
   D. Wedge fractures never cause loss of vertebral height, as only the anterior aspect is affected.
   E. The most common type of disc injury in children is herniation.

13. Which of the following is true regarding imaging for pediatric TL spinal injury?
   A. CT is more sensitive test for TL spine injury.
   B. Normal apophyseal fusion can be mistaken for a fracture in plain films.
   C. The physical exam is highly sensitive for TL spinal injury.
   D. Decision rules for TL spine injury are highly sensitive and specific.
   E. Young athletes who present with back pain do not require imaging as their injuries are likely muscular or ligamentous.

CNE/CME Objectives

Upon completing this program, the participants will be able to:
- discuss conditions that should increase suspicion for traumatic injuries;
- describe the various modalities used to identify different traumatic conditions;
- cite methods of quickly stabilizing and managing patients; and
- identify possible complications that may occur with traumatic injuries.

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Trauma Reports

2012 Reader Survey

In an effort to learn more about the professionals who read *Trauma Reports,* we are conducting this reader survey. The results will be used to enhance the content and format of *Trauma Reports*.

Instructions: Fill in the appropriate answers. Please write in answers to the open-ended questions in the space provided. Please insert this survey in the provided envelope along with your continuing education evaluation. Return the questionnaire by **July 1, 2012.**

1. Are the articles in *Trauma Reports* written about issues of importance and concern to you?

O A. Always
O B. Most of the time
O C. Some of the time
O D. Rarely
O E. Never

2. How would you rate your overall satisfaction with your job?

O A. Very satisfied
O B. Somewhat satisfied
O C. Somewhat dissatisfied
O D. Very dissatisfied

3. What are you most dissatisfied with in your job?

O A. staffing
O B. heavy workload
O C. low morale in your department or facility
O D. impact of cost-cutting on quality of care
O E. other _____

Questions 4-9 ask about coverage of various topics in *Trauma Reports*. Please mark your answers in the following manner:

A. very useful B. fairly useful C. not very useful D. not at all useful

4. Knee and patellar dislocations (July/Aug. 2011)	O A	O B	O C	O D
5. Pain control in trauma patients (Sept./ Oct. 2011)	O A	O B	O C	O D
6. Pulmonary contusions (Nov./Dec. 2011)	O A	O B	O C	O D
7. Gunshot wounds (Jan./Feb. 2012)	O A	O B	O C	O D
8. Penetrating and blunt cardiac trauma (March/April 2012)	O A	O B	O C	O D
9. Pediatric spine fractures (May/June 2012)	O A	O B	O C	O D

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O B. I receive it as a supplement to another publication (skip to question 12)

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If not, why? _____

12. How would you describe your satisfaction with your subscription to *TR*?

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O B. Somewhat satisfied
O C. Somewhat dissatisfied
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O C. 1-2 hours
O D. more than 2 hours

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O B. 4-6
O C. 7-9
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16. On average, how many articles do you find useful in *TR* each year?

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Please rate your level of satisfaction with the following items.
A. excellent B. good C. fair D. poor

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19. Article selections	○ A	○ B	○ C	○ D
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21. Length of newsletter	○ A	○ B	○ C	○ D
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Upon completion of this educational activity, participants should be able to:
- discuss conditions that should increase suspicion for traumatic injuries;
- describe the various modalities used to identify different traumatic conditions;
- cite methods of quickly stabilizing and managing patients;
- identify possible complications that may occur with traumatic injuries.

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This activity is valid 24 months from the date of publication. The target audience for this activity emergency medicine physicians and nurses, trauma surgeons, and nurses.

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Emergency Medicine Reports
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Emergencies in the Dialysis Patient

Management of Hyperkalemia

Drug	Dose	Onset of Action	Duration of Action	Mechanism of Action (MOA)
Calcium chloride	10 mL of 10% solution IV (use only central line or in code situation) over 10 minutes Can repeat in 5 min	Immediate	30-60 min	Antagonizes cell excitation
Calcium gluconate	10 mL of 10% solution IV over 10 min; can repeat in 5 min	Immediate	30-60 min	Antagonizes cell excitation
Insulin	10 units regular insulin IV, with 25 g of D50W IV; should use D5W or D10W infusions thereafter	20 min	4-6 hours	Intracellular K redistribution
Albuterol	10-20 mg by nebulizer	30 min	2 hours	Intracellular K redistribution
Terbutaline	7 mcg/kg body weight, SQ	< 60 min		Intracellular K redistribution
Sodium bicarbonate	150 mEq/L at 1-2 cc/kg/hr	Hours	While infused	Intracellular K redistribution
Furosemide	40-80 mg IV	15 min	2-3 hours	Urinary K excretion
Bumetanide	2-4 mg IV	15 min	2-3 hours	Urinary K excretion
Sodium polystyrene sulfonate	30-60 g PO or enema	At least 12 hours (probably longer)	At least 24 hours	Fecal K excretion

Adapted from: Weisberg LS. *Crit Care Med* 2008;36:3246-3251.

Opioid Use in ESRD Patients

Opioid	Metabolism and Excretion	Dosing Recommendations
Morphine	The 6-glucuronide metabolite, which is very potent, can accumulate and cause respiratory depression. The 3-glucuronide also accumulates, and is thought to mediate CNS excitation.	Reduce the dose and the frequency of dosing. Beware of many different available formulations.
Hydromorphone and hydrocodone	Hydrocodone is metabolized by the liver into hydromorphone, which in turn is metabolized to the 3-glucuronide. This molecule has no analgesic activity, but is neuro-excitatory. Hydromorphone and its metabolites are renally excreted and easily dialyzable.	Use lower doses and less frequent dosing as a starting point. Beware of the acetaminophen component of the hydrocodone preparations.
Oxycodone	It is metabolized by the liver to several inactive and one active compound (oxymorphone), all of which are renally excreted.	The data on ESRD patients is scant, and it should be used with caution. It has higher abuse potential. Beware of the acetaminophen component in some preparations.
Codeine	It is mainly metabolized to the 6-glucuronide, and both compounds are renally excreted. Cases of excessive sedation due to drug accumulation have been reported. Codeine is not dialyzable.	Codeine should be avoided in ESRD patients.
Methadone	It is eliminated by both the kidneys and the feces. There are no reported cases of adverse effects in ESRD patients.	There is limited information on safety and dosing in ESRD.
Fentanyl	Fentanyl is metabolized by the liver to norfentanyl. Cases of respiratory depression due to accumulation of drug have been reported.	Use with caution.
Meperidine	Meperidine and its metabolites are renally excreted. The most toxic metabolite is normeperidine. Its accumulation results in CNS excitation and seizures.	Do not use in the ESRD patient.

Supplement to *Emergency Medicine Reports*, May 7, 2012: "Emergencies in the Dialysis Patient."
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