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Although pediatric cardiac diseases infrequently are seen in the emergency department (ED), early diagnosis and aggressive management is critical. Most importantly, the clinician must include these diseases in their differential and have a thorough understanding of typical and atypical presentations for congenital heart disease (CHD), dysrhythmias, myocarditis and pericarditis. Any child who has a clinical presentation suggestive of cardiac disease, must receive appropriate diagnostic testing and timely referral to optimize the child's outcome. The authors provide a thorough, focused review of the most commonly encountered cardiac diseases in the ED and key aspects to stabilization.

—The Editor

Cardiac Disorders in the Pediatric Patient

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Introduction

Pediatric cardiac disorders, though not common in the ED, may be associated with significant morbidity and mortality. Emergency physicians cannot afford to miss these problems; a missed diagnosis may result in further decompensation or death. Further challenging the clinician, the presenting complaints for many pediatric cardiac disorders may be nonspecific. Vague chief complaints such as fussiness or difficulty feeding may be the initial presentation for an infant with supraventricular tachycardia or congestive heart failure (CHF). Complaints commonly associated with cardiovascular disorders in adults, such as chest pain or palpitations, may be absent in children. This article provides an overview of the diagnosis and management of pediatric cardiac disorders.

Dysrhythmias

Normally, the heart rate is fastest in the newborn, and decreases with age until adolescence, when the upper and lower parameters of normal are similar to those of an adult. Normal variations do occur, but the explanation should follow expected physiologic patterns. Sinus tachycardia may result from fever, pain, anxiety, dehydration, anemia, or acute blood loss. Sinus bradycardia may be caused by vagal stimulation, hypoxemia, increased intracranial pressure, hypothyroidism, or acidosis. A sinus dysrhythmia is not uncommon in children, and does not necessarily denote a pathological condition.

Supraventricular Tachycardia. Paroxysmal supraventricular tachycardia (SVT) is the most common symptomatic dysrhyth-

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mia in infants and children. (*See Figure 1.*) The history for infants with SVT may include nonspecific complaints, such as fussiness, lethargy, poor feeding, pallor, sweating with feeds, or simply "not acting right." Chest pain, pounding in the chest, dizziness, or shortness of breath may be the initial complaint of older children with SVT. In contrast, patients with sinus tachycardia generally have a more specific history, such as fever, dehydration due to fluid or blood loss, anxiety, or pain that accounts for the tachycardia.

In newborns and infants with SVT, the heart rate is greater than 220 beats per minute (BPM).[1] SVT in older children is defined as a heart rate of more than 180 BPM.[1] The electrocardiogram (ECG) for supraventricular tachycardia will show a narrow complex tachycardia without discernible p-waves or beat-to-beat variability. (*See Figure 1*).

An accessory atrioventricular pathway is the most common cause of SVT in children younger than 12 years, whereas atrioventricular node re-entry tachycardia becomes more common in adolescents.[2] The term Wolff-Parkinson-White (WPW) syndrome is applied when there is an extra atrioventricular accessory pathway.

Treatment for supraventricular tachycardia depends on patient stability. Immediate cardioversion with 0.5 to 1 J/kg is indicated for unstable patients. In the stable patient, vagal maneuvers, such as blowing through an occluded straw or placing an ice bag on an infant's face, may prove successful. If vagal maneuvers are unsuccessful, adenosine 0.1 mg/kg (up to 6 mg) may be given through the most central vein possible. A normal saline flush of 5 mL should then be administered. If the first dose is unsuccessful, an increased dose of 0.2 mg/kg (up to 12 mg) may be administered.[1] If these measures are unsuccessful, a pediatric cardiologist should be consulted; there are several options, including procainamide or amiodarone. Verapamil should be avoided in children younger than 1 year; cardiovascular collapse and death can occur due to electromechanical dissociation.[3] Long-term management of children with supraventricular tachycardia may include beta-blockers, procainamide, sotalol, amiodarone, or flecainide. Another option may be radio-frequency catheter ablation, which has reported success rates of 85-95%.[4]

Digoxin is effective in converting SVT; however, the onset of action is slow, and it may be associated with dangerous complications if used to treat a patient with SVT and WPW.[5]

Wolff-Parkinson-White Syndrome (WPW). WPW syndrome denotes an accessory atrioventricular pathway (Kent bundle) that predisposes individuals to supraventricular tachycardias. Episodes of SVT in children with WPW syndrome usually occur early in the first year of life, frequently resolve, and then recur later in life, usually between 6-8 years of age.[6]

Clinical manifestations of WPW syndrome are secondary to the occurrence of dysrhythmias and dependent on the patient's age, and the duration and rate of the dysrhythmia. Infants typically present with symptoms of fussiness, irritability, and poor feeding. If CHF is present, caretakers may describe pallor, cough, and respiratory distress. The heart rate in infants will be greater than 220 BPM, and may be as fast as 280 BPM.[7] The presentation in older children differs due to their ability to describe symptoms and the fact that CHF rarely develops secondary to SVT in older children.[7] Episodic palpitations are a common complaint in older children, and often occur during rest. Older patients also describe symptoms of dizziness, shortness of breath, and chest discomfort.

SVT in WPW syndrome generally is initiated by a premature atrial depolarization that travels to the ventricles via the normal atrioventricular pathway, travels retrograde through the accessory pathway and reenters the AV node to start a reentrant type of tachycardia.[8] Antegrade conduction through the AV node followed by retrograde conduction through the accessory pathway

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Figure 1. Rhythm Strip Demonstrating Supraventricular Tachycardia

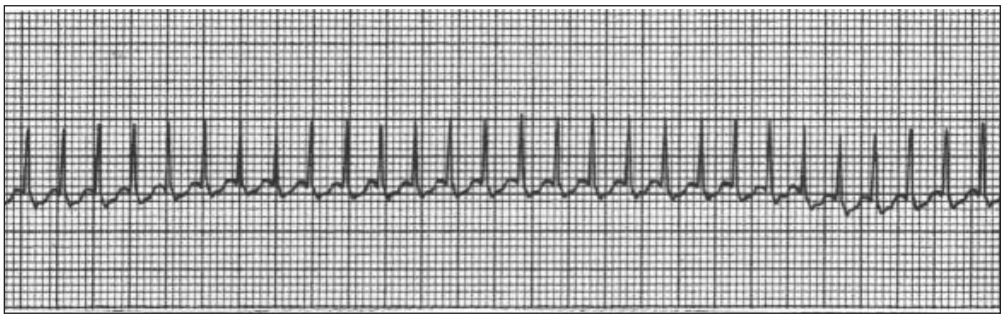

Figure 2. ECG Showing Wolff-Parkinson-White (WPW) Syndrome

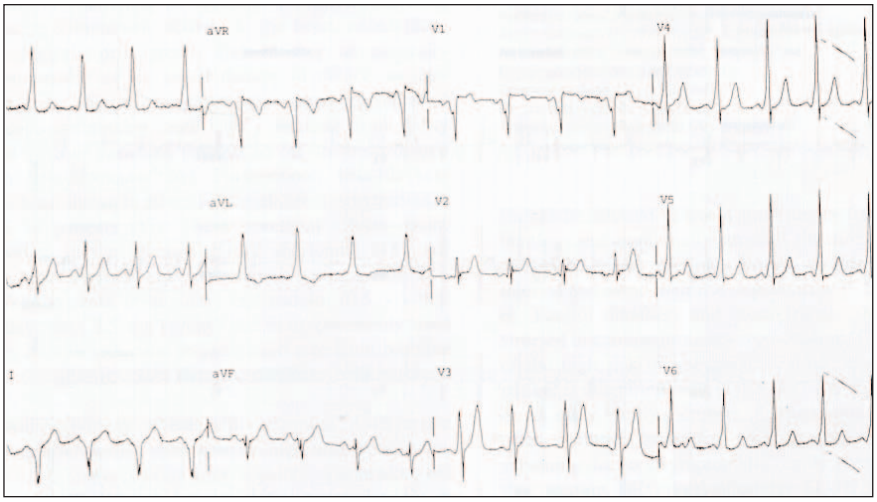

Figure 2. Note the delta waves.

produces a narrow complex tachycardia (orthodromic tachycardia), and is the most common form of SVT found with WPW syndrome.[5] Less commonly, reentry occurs with antegrade conduction through the accessory pathway and retrograde conduction through the AV node (antidromic tachycardia), to produce a wide complex tachycardia.[5]

ECG findings consistent with WPW syndrome include a short PR interval, a widened QRS, and a slurred upstroke of the QRS complex known as a delta wave. (*See Figure 2.*) The characteristic ECG findings of WPW syndrome are not evident during a tachydysrhythmia. As described earlier, the ECG will demonstrate a narrow QRS supraventricular tachycardia if conduction is antegrade through the AV node (orthodromic). A wide QRS tachycardia may be present with antegrade conduction through the accessory pathway (antidromic), and this can be mistaken for a ventricular tachycardia. Atrial fibrillation in patients with WPW syndrome, although rare in infancy, does occur in late childhood, particularly after 12 years of age.[7]

Acute management of SVT in patients with WPW syndrome is similar to that of other forms of SVT with some specific precautions. In unstable patients, synchronized cardioversion at 0.5 to 1 J/kg is the recommended initial therapy.[1] Repeat attempts at cardioversion with 2 J/kg are acceptable if the initial attempt is unsuccessful.[1] If intravenous access is available immediately, adenosine at a dose of 0.1 mg/kg as a rapid IV bolus (maximum of 6 mg for initial dose) is also acceptable as initial therapy.[1] Subsequent doses of adenosine can be doubled to 0.2 mg/kg and again to 0.4 mg/kg (maximum of 12 mg) as needed.[9] Cardioversion should not be delayed for attempts at intravenous access or sedation in unstable patients. In stable patients, adenosine (same dose as above) is the first line of therapy. Failure to terminate the dysrhythmia with adenosine in a stable patient should prompt consultation with a pediatric cardiologist. Digoxin should not be used in patients with WPW syndrome secondary to the risk of enhancing conductivity through the accessory path and subsequent ventricular fibrillation and sudden death. It also should be noted that intravenous verapamil and propranolol are contraindicated in children younger than 1 year of age.[7] Following stabilization and conversion of the SVT to a normal sinus rhythm, long-term prophylactic therapy should be instituted by a pediatric cardiologist. Generally, prophylactic therapy is instituted in all infants until at least 1 year of age.[5] Beta-blockers are recommended as the drug of choice for prophylactic therapy in infants with WPW syndrome.[5] Procainamide may be used if beta-blockers fail.[5]

Bradydysrhythmias. Acute bradycardia, in pediatric patients, may be attributable to vagal stimulation, hypoxemia, acidosis, or increased intracranial pressure. Complete heart block is a common cause of significant bradycardia in pediatric patients and may be acquired or congenital. Causes of congenital heart block include structural lesions like L-transposition of the great arteries, or maternal connective tissue disorders. Acquired heart block may result from disorders such as Lyme disease, lupus, muscular dystrophies, Kawasaki disease, or rheumatic fever.[8,10]

Management of bradycardia includes identification of the etiology and appropriate cardiopulmonary resuscitation, with assisted ventilation, oxygenation, and chest compressions as indicated. Epinephrine or atropine is given if symptomatic bradycardia persists despite initial resuscitative measures.[1] Pharmacologic management of complete heart block includes atropine or isoproterenol. A transcutaneous pacemaker may serve as a bridge to a transvenous pacemaker in the acute setting.

Ventricular Tachycardia/Fibrillation. Ventricular tachycardia (VT) may result from electrolyte abnormalities, congenital heart disorders, myocarditis, or drug toxicity. Both ventricular

Figure 3. ECG Demonstrating Long QT Syndrome

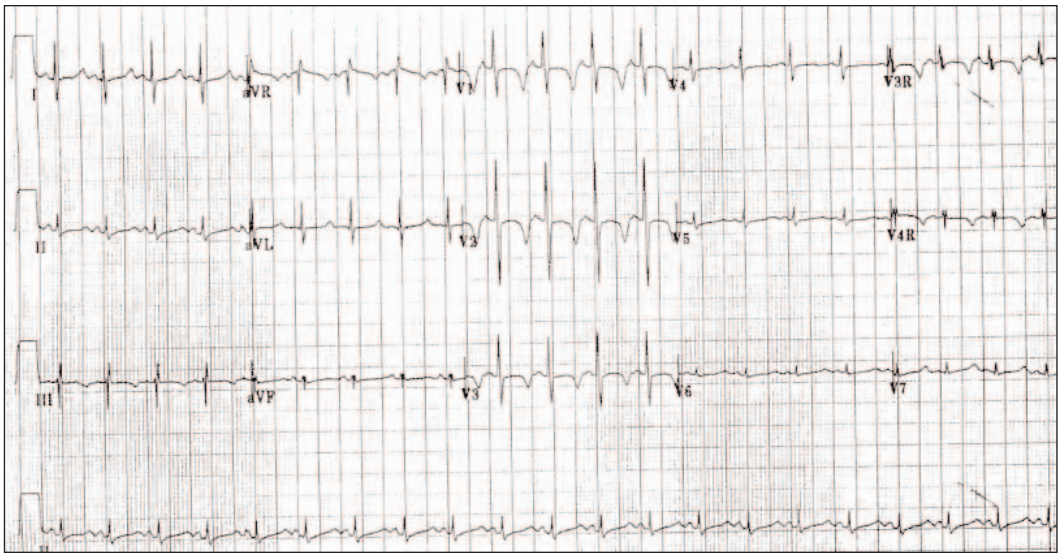

Figure 3. Note the prolonged QT interval as calculated with the Bazett formula: QTc = QT/square root of the preceding RR interval. QTc = 0.36/(square root of 0.44). QTc = 545 msec**.**

Courtesy of Nicholas Tsarouhas, MD, The Children's Hospital of Philadelphia

tachycardia and ventricular fibrillation (VF) are uncommon rhythms in pediatric patients. VT with a pulse in an unstable patient warrants immediate synchronized cardioversion at 0.5 to 1 J/kg.[1] Pharmacologic alternatives also may include amiodarone, procainamide, or lidocaine.[1] Amiodarone and procainamide should not be given together. Pulseless VT and VF should be treated with defibrillation at 2 J/kg, then 2-4 J/kg, and then 4 J/kg if it does not respond to the initial attempts at defibrillation.[1] If defibrillation is unsuccessful, epinephrine should be given, and repeated every 3-5 minutes as necessary.[1] Amiodarone, lidocaine, and magnesium also may be considered if VF or pulseless VT is refractory to the above measures.[1]

Prolonged QT Syndrome. Congenital prolonged QT syndrome, also known as long QT syndrome (LQTS), is a disorder of delayed ventricular repolarization characterized by prolongation of the QT interval. (*See Figure 3*.) It may be either hereditary or acquired. Jervell-Lange-Nielsen syndrome is an autosomal recessive form of prolonged QT syndrome associated with congenital deafness, whereas Romano-Ward syndrome is an autosomal dominant form that is not associated with deafness. The hallmark dysrhythmia is torsades de pointes (twisting of the points), although other dysrhythmias may occur. Patients commonly present between the ages of 9 and 15 years with recurrent episodes of near-syncope or syncope.[11,12] Syncopal episodes may be precipitated by intense emotion, vigorous physical activity, or loud noises. Spontaneous return of consciousness usually follows a syncopal episode, but the dysrhythmia has the potential to degenerate into ventricular fibrillation and sudden death.[11,13]

Approximately 10% of children with LQTS present with sudden death, with younger children being more likely to die suddenly.[11-13] Several reports of death due to abrupt awakening by an alarm clock or ringing telephone have been documented in patients with LQTS.[11,13] LQTS also may present in infancy as sudden infant death syndrome.[14-16]

The Bazett formula is the most commonly used manual method to measure the QT interval. (*See Figure 3*.)[17] This equation accounts for the normal physiologic shortening of the QT interval that occurs with increasing heart rate. The QT interval on ECG represents the time from onset of depolarization to completion of repolarization. Lead II generally is the lead accepted for QTc calculations. The QT interval is measured from the onset of the QRS complex to the end of the T wave where it returns to the baseline. The QT and preceding RR intervals should be measured for three consecutive beats and averaged for the greatest accuracy.[18] Other ECG abnormalities that suggest LQTS include prominent U waves, a T-U complex with an indistinct termination of the T wave, and broad T waves, which may be notched, biphasic, or inverted. T-wave alternans, an alternating amplitude and polarity of the T waves, also may be present.[19]

Computer-generated interpretations of ECGs generally are useful and correct, but may be less accurate as a screening tool for LQTS.[20] Errors in the automated measurement of the QTc increase when the precise end of the T wave cannot be determined easily. One recent study shows that, despite a prolonged QTc by automated measurement, the computer generated a normal ECG reading in 50% of family members who proved to be genetic carriers of LQTS.[20]

Any patient with a suspicious history, borderline prolongation of the QT interval with symptoms, or identified prolonged QT syndrome should receive a cardiology consult for further management. Therapy is aimed at reducing sympathetic activity to the heart, either pharmacologically or surgically. Beta-blockers generally are recommended as the initial therapy of choice and have been shown to significantly reduce episodes of syncope and sudden death, with a decrease in mortality from 71% in untreated patients to 6% in those treated.[21] Beta-blockers also have been shown to effectively eradicate dysrhythmias in 60% of patients.[12] These beneficial effects occur, however, in the absence of QT

shortening.[13] All beta-blockers appear to be effective, but propranolol and nadolol are used most commonly.[22]

Patients presenting with LQTS may require emergency intervention. Patients with polymorphic ventricular tachycardia or torsades de pointes of unknown etiology, should receive IV magnesium (25-50 mg/kg; max 2 g). Serum electrolytes and a toxicology screen should be obtained. Beta-blockers may be useful in suppressing catecholamine surges and further dysrhythmic activity. Patients with recurrent ventricular tachycardia may require temporary transcutaneous ventricular pacing.[23]

Physicians should be aware of drugs known to prolong the QT interval and avoid their use in patients with LQTS. Family members and close friends should be instructed in cardiopulmnary resuscitation because of the high risk of unexpected cardiac events. Once a patient is diagnosed with LQTS, ECGs should be done on all other family members. Asymptomatic carriers often are identified by routine ECG or after familial ECG screening due to a symptomatic family member.

Brugada Syndrome. First described in 1992 by Brugada and Brugada, the Brugada syndrome is associated with sudden death in patients with a structurally normal heart, and characterized by specific ECG findings, which include a right bundle-branch block (RBBB) and ST-segment elevation in leads V_1-V_3.[24] Although it has been found throughout the world, occurring in both women and children,[24-28] the syndrome most commonly occurs in southeast Asian males, and the initial cardiac event usually occurs in the 30s and 40s.[25,29,30] It also has been suggested that Brugada syndrome may cause sudden death in children during the first few months of life and may be misdiagnosed as sudden infant death syndrome.[27]

Brugada syndrome is a genetically inherited disease with an autosomal dominant inheritance pattern and variable expression.[25,27,30] The mutation occurs in the SCN5A gene that encodes for the human cardiac sodium channel. The improperly functioning sodium channels predispose to developing ventricular tachydysrhythmias, specifically a rapid polymorphic ventricular tachycardia, which can degenerate into ventricular fibrillation.[26,30] Dysrhythmic episodes are unpredictable and may or may not terminate spontaneously. Episodes that do not terminate spontaneously result in sudden cardiac arrest, whereas those that do terminate spontaneously may present as syncope or near-syncope. Unfortunately, sudden death may be the initial presentation. Sudden death seems to occur commonly during sleep, particularly in the early hours of the morning.[30] Medications, such as Type I antidysrhythmics, which affect the cardiac sodium channels, can provoke dysrhythmias.[26]

A presumptive diagnosis of Brugada syndrome should be made in patients with sudden cardiac arrest that are resuscitated successfully, syncope, or near-syncope, and have an ECG pattern suggestive for Brugada syndrome. The ECG pattern, as originally described, consists of a RBBB with ST-segment elevation in leads V_1-V_3.[24] It has been noted since that the RBBB pattern may be incomplete.[26] Three types of ST-segment patterns have been described. Type 1 is characterized by a coved ST-segment elevation with amplitude of more than 2 mm at its peak followed by a negative T-wave.[30] The ST-segment pattern in Type 2 has a high take-off ST-segment elevation that descends, followed by a positive T-wave resulting in a saddle-back configuration.[30] Type 3 has a ST-segment elevation of less than 1 mm with a saddle-back or coved morphology.[30] The morphology of the ST-segment elevation does not appear to be predictive of sudden cardiac events.[29] It is important to note that the ECG pattern is intermittent and may transiently normalize.[29,30]

Management in the ED of symptomatic patients with a suggestive ECG includes initial stabilization as necessary and cardiology consultation. All patients suspected of having Brugada syndrome require confirmatory testing and definitive management by a cardiologist. The only effective long-term treatment for patients at risk for sudden cardiac events is placement of an implantable cardioverter-defibrillator (ICD). Antidysrhythmic medications have not been shown to have any beneficial effects in patients with Brugada syndrome.

Further inpatient studies, such as administration of pharmacologic agents to accentuate the ECG changes or electrophysiologic studies, can be performed to confirm the diagnosis of Brugada syndrome. Although there has been some debate, these studies appear to be useful as predictors of dysrhythmic events.[31,32]

Congenital Heart Disease (CHD)

CHD occurs in eight of 1000 live births; many of the structural CHDs present in the neonatal period.[33] The signs and symptoms of CHDs may be nonspecific and include: tachypnea, sudden onset of cyanosis or pallor that worsens with crying, sweating with feedings, lethargy, or failure to thrive. Time of presentation for cyanotic and acyanotic CHD and the common associated ECG and pulmonary blood flow pattern findings are listed in Tables 1 and 2.[34]

Congenital heart lesions that present in the first two to three weeks of life are typically ductal-dependent. The ductus arteriosus sustains blood flow for these infants and when the ductus closes anatomically at 2-3 weeks of life, these infants decompensate. The ductus arteriosus supplies blood to either the lungs, as with tetralogy of Fallot (TOF) or tricuspid atresia (cyanotic CHD), or to the systemic circulation as in the case of coarctation of the aorta, hypoplastic left heart syndrome, or aortic stenosis. When the ductus closes, the infant develops respiratory distress, shock, or an altered mental status.

Any neonate presenting with cyanosis or respiratory distress should be evaluated immediately with close attention to the ABCs (airway, breathing, and circulation). A pulse oximetry measurement or arterial blood gases (ABG) measurement from the right hand and foot may help in the diagnosis. Significantly lower oxygen saturation will be found in the lower extremities in a patient with a coarctation of the aorta or interrupted aortic arch. However, transposition of the great arteries may be associated with higher oxygen saturations in the lower extremities than in

the upper extremities.[35]

The main etiologies of cyanotic CHD are TOF, tricuspid atresia, transposition of the great arteries (TGA), truncus arteriosus, total anomalous pulmonary venous return (TAPVR), and pulmonary atresia or stenosis. If cyanosis is present, one must first determine if the cyanosis is central (cyanosis of the lips, tongue, and oral mucous membranes) or peripheral. If the cyanosis is central, then performing the hyperoxia test can help distinguish a cardiac from a pulmonary etiology. To perform the hyperoxia test, the cyanotic infant should be placed on 100% oxygen. If the oxygen saturation increases significantly, then the infant probably has pulmonary pathology. If the oxygen saturation does not increase by 10%, then consider a congenital heart disorder. The most accurate way to perform this test is to obtain a baseline ABG measurement, and then repeat it approximately 10 minutes after administration of 100% oxygen.

Table 1. Typical ECG and Radiographic Findings in Cyanotic Congenital Heart Disease Based on Age of Presentation

AGE		ECG	X-RAY
Birth-first week of life	Transposition of the great vessels	RVH	PBF (inc)
First week of life	Total anomalous pulmonary venous return	RVH	PBF (inc)
1-4 weeks of life	Tricuspid atresia	LVH	PBF (dec)
	Severe pulmonic stenosis	RVH	PBF (dec)
1-12 weeks of life	Tetralogy of Fallot	RVH	PBF (dec)
Anytime in infancy	Truncus arteriosus	BVH	PBF (inc)

Key: RVH = right ventricular hypertrophy. PBF = pulmonary blood flow. inc = increased. LVH = left ventricular hypertrophy. dec = decreased. BVH = biventricular hypertrophy.

Other congenital cardiac lesions that present in the first month of life are the left-to-right intracardiac shunts, such as ventricular septal or atrioventricular canal defects. As the normal pulmonary vascular resistance falls during the first month of life, any preexisting left-to-right shunt will see a gradual increase in flow across the shunt resulting in CHF. The differential diagnosis of congenital heart diseases that cause CHF include the left-to-right intracardiac shunts, hypoplastic left ventricle, coarctation of the aorta, truncus arteriosus, endocardial cushion defect, patent ductus arteriosus (PDA), aortic stenosis, interrupted aortic arch, aortic atresia, and mitral valve atresia.[36,37]

An ECG and chest X-ray should be obtained in all infants suspected of having CHD. Frequently it is difficult in the ED to distinguish between sepsis and CHD. Therefore, all children who present with cyanosis or shock should receive a full septic evaluation and early initiation of antibiotic therapy. The lumbar puncture may be postponed if there is any concern about the infant's respiratory status.

If a ductal-dependent CHD is suspected, a prostaglandin E1 (PGE1) infusion should be initiated immediately at a rate of 0.05-0.1 mcg/kg/minute. Ideally, a pediatric cardiologist should be consulted prior to initiation of the infusion and echocardiography used if immediately available to confirm the diagnosis of CHD.[38] However, therapy with PGE1 should not be withheld; there are no other temporizing medications to maintain ductal patency. Prostaglandin is a very potent vasodilator and will have immediate effects on the ductus, with improvement usually seen within 15 minutes. The practitioner should be prepared to intubate because apnea is a common occurrence with PGE1 initiation. Although routine intubation is not necessary prior to initiation of PGE1, it may be prudent to prophylactically intubate patients requiring transportation to another facility. Other complications of prostaglandin use include fever, hypotension, and seizures.

If the patient has CHF, furosemide (1 mg/kg) should be administered with the possible addition of morphine, dobutamine, dopamine, or angiotensin inhibitors.[39] These patients can deteriorate rapidly and should be admitted to either a neonatal or pediatric intensive care unit (PICU).

Hypertrophic Cardiomyopathy

Most cases of hypertrophic cardiomyopathy (HCM) are diagnosed in patients between 30 and 40 years of age, but 2% of cases occur in children younger than 5 years, and 7% occur in children younger than 10 years of age.[40] The hallmark anatomic finding in patients with HCM is an asymmetric, hypertrophied, nondilated left ventricle with greater involvement of the septum than the ventricle. Although the left and right ventricles are small to normal in size, there is atrial enlargement and thickening of the mitral valve in 95% of cases.[41]

Presenting symptoms range from chest pain, palpitations, or shortness of breath, to near syncope, syncopal episodes, and sudden death. Children with HCM who experience syncope are at significant risk for sudden death, typically occurring during strenuous exercise.

The physical examination typically reveals a loud S_4 gallop with a harsh mid-systolic crescendo-decrescendo murmur. This murmur is accentuated with Valsalva maneuvers or the standing position. Squatting, isometric hand grip, or lying down will decrease the murmur due to an increase in left ventricular end-diastolic volume.

Classic ECG findings include left atrial and ventricular hypertrophy, ST-segment abnormalities, T-wave inversions, Q waves, and diminished or absent R waves in the lateral leads. Premature atrial and ventricular contractions, supraventricular tachycardia, multifocal ventricular dysrhythmias, or new onset atrial fibrilla-

Table 2. Age of Presentation, ECG Findings, and Pulmonary Blood Flow Patterns with Acyanotic Congenital Heart Disease

AGE		ECG	X-RAY
First week of life	Hypoplastic left heart syndrome	RVH	PBF (inc)
	Coarctation of the aorta	LVH	PBF (nl)
First 2-3 weeks of life	Complete AV canal	BVH or LVH	PBF (inc)
1-4 weeks of life	Patent ductus arteriosus	LVH	PBF (inc)
2-12 weeks of life	Ventricular septal defect	LVH	PBF (inc)

Key: RVH = right ventricular hypertrophy. PBF = pulmonary blood flow. inc = increased. LVH = left ventricular hypertrophy. dec = decreased. BVH = biventricular hypertrophy. AV = atrioventricular. nl = normal.

tion also may be present. The echocardiogram is the diagnostic procedure of choice.

A cardiologist should be consulted as soon as the diagnosis is suspected to assist with management. Unless the patient is symptomatic, it is not necessary to start medications in the ED. The goal of therapy is to decrease the heart rate to increase the diastolic filling time with beta-blockers the cornerstone of therapy. Calcium-channel blockers also are used, particularly if there is no response to the beta-blocker. Nitrates should be avoided if HCM is suspected, as they decrease ventricular volume and resultant outflow tract volume. Digoxin also is contraindicated.

Ventricular dysrhythmias may be treated with amiodarone. Surgical intervention, typically a septal myomectomy, may be warranted in patients with systolic gradients more than 50 mmHg, minimal response to medical management, or severe symptoms. Antibiotic prophylaxis against bacterial endocarditis also is indicated. Patients undergoing an evaluation for HCM, or in whom the diagnosis is suspected, should refrain from strenuous activity or physical exertion as sudden death can occur.[42]

Myocarditis

Myocarditis is an inflammatory condition of the myocardium that can occur in conjunction with pericarditis or in isolation. Although there are numerous causes, including infections, drugs, endocrine disorders, radiation, and collagen vascular diseases, the most common etiology in North America is viral (coxsackievirus A and B, ECHO viruses, and influenza viruses).[40,43] The clinical presentation varies depending on multiple factors, including the etiology and age of the patient. Neonates and infants may present with symptoms such as lethargy, poor feeding, irritability, pallor, fever, or failure to thrive. Symptoms suggestive of heart failure, such as diaphoresis with feeding, rapid breathing, or respiratory distress, may be present. Older children and adolescents can present similarly, and also have complaints of weakness, fatigue, chest pain, or shortness of breath. A recent history of a nonspecific viral-syndrome type illness also is common. Potential findings on physical examination include tachypnea, tachycardia, hyperthermia or hypothermia, and hypotension. Signs of poor perfusion and heart failure, such as tachycardia, weak pulses, decreased capillary refill, cool mottled extremities, jugular venous distention, hepatomegaly, and lower extremity edema, may be present. Heart tones may include an S_3, that may be muffled if pericarditis is present. Although several types of dysrhythmias occur, the most common dysrhythmia is sinus tachycardia. Tachycardia faster than expected for the degree of fever (10 BPM for each degree of temperature elevation) may be suggestive of myocarditis.

A complete blood count (CBC) with differential may show an elevated white blood cell count. A lymphocytic predominance on the differential suggests a viral etiology. The sedimentation rate and C-reactive protein level usually are elevated, but normal values do not exclude the diagnosis of myocarditis. Elevation of creatine kinase-MB isoenzyme (CK-MB), lactate dehydrogenase (LDH), and troponin levels can occur. Blood cultures as well as throat, nasopharyngeal, stool, and urine specimens should be obtained to identify bacterial or viral pathogens.

The most common finding on ECG is sinus tachycardia. Other abnormalities, such as premature ventricular beats, junctional tachycardias, ventricular tachycardias, and even second- and third-degree atrioventricular blocks also may be present. A low-voltage QRS, fewer than 5 mm in all limb leads, suggests myocarditis. As pericarditis may occur simultaneously, the ECG may show ST-segment elevation and PR depression (see section on pericarditis).

The chest x-ray frequently demonstrates cardiomegaly, and pulmonary edema may be present. An echocardiogram is valuable to determine myocardial function. If heart failure is present, an echocardiogram will show increased left ventricular end diastolic and systolic dimensions. Other potential echocardiogram findings include left ventricular wall dysfunction, decreased ejection fraction, segmental wall motion abnormalities, or global hypokinesis.

Initial management should focus on the patient's respiratory and circulatory status. All patients in respiratory distress should be started on supplemental oxygen, and cardiac monitoring and pulse oximetry should be initiated. If the patient continues to deteriorate, or is in cardiogenic shock, endotracheal intubation and ventilatory support may be necessary. Management for patients with signs of heart failure includes diuretics, digoxin, inotropic agents as needed, and afterload reduction. Intravenous furosemide (1 mg/kg IV) is used to decrease fluid overload and preload. Digoxin may be used to improve left ventricular function following consultation with a cardiologist. Digoxin has potential risk in patients with increased myocardial sensitivity secondary to the inflammatory changes of myocarditis. Inotropic agents, such as dopamine and dobutamine may be necessary if

hemodynamic instability is present. Afterload-reducing agents help reduce the workload for the poorly functioning myocardium. If hypotension is not present, intravenous nitroprusside or milrinone may be used. Oral agents such as ACE inhibitors may be used if the patient is stable.

Intravenous gamma globulin in the setting of acute myocarditis may improve ventricular function and survival.[44] The use of immunosuppressive agents (e.g., steroids or cyclosporine) in acute myocarditis is controversial, and therefore should not be initiated in the ED. All patients with myocarditis should be admitted to a PICU for further management and observation.

The diagnosis of myocarditis may not be made in the ED; endomyocardial biopsy is the gold standard for diagnosis of myocarditis. Patients may present with a seemingly benign viral illness with symptoms of weakness, fever, and emesis; it is important for the clinician to maintain a high degree of suspicion for this disease.

Pericarditis. The pericardium consists of two layers: the visceral layer and the parietal layer. In a healthy person, a small amount (10-15 mL in a child) of serous fluid normally exists. When inflammation of the pericardium occurs, fluid accumulates between the two layers of the pericardium and forms a pericardial effusion. Normally, the pericardium does not affect the filling of the heart. However, when a pericardial effusion is present, the filling capacities of the heart chambers are limited, resulting in increased end-diastolic filling pressures and decreased cardiac output. Acute development of an effusion is more likely to result in such complications because the pericardium does not have time to stretch and accommodate the increased volume. Significant effusions may lead to cardiac tamponade.

The frequency of pericarditis is unknown, and there does not appear to be any sex or age predilection. Symptoms vary depending on the etiology and how rapidly the pericardial effusion develops. Common presenting symptoms include respiratory difficulty, fever, and substernal chest pain that may radiate to the left shoulder. The chest pain may be accentuated by lying flat or respiratory motion and improved with sitting forward. Patients with a viral etiology may have a recent history of illness, including upper respiratory tract symptoms and fever. There are both infectious and noninfectious causes of pericarditis. Among the infectious causes, viral etiologies (e.g., coxsackievirus A and B, ECHO viruses, and influenza) are more common.

Physical findings vary depending on the severity of disease. With no significant effusion, the only sign on physical examination may be a friction rub. With more serious disease, there will be tachypnea and tachycardia. Signs of cardiac tamponade include distended neck veins, clear lungs, weak peripheral pulses, tachycardia, distant heart tones with auscultation, and a pulsus paradoxus.

A CBC count may reveal an elevated white blood cell count in the presence of bacterial pericarditis, but may be normal with other etiologies. Measurements of electrolyte, BUN, and creatinine levels are indicated. Appropriate cultures (e.g., blood, urine, stool) and viral serology should be obtained, and an erythrocyte sedimentation rate (ESR) may be helpful.

The ECG typically shows diffuse ST-segment elevation in both the limb and precordial leads with associated upright T-waves. ST depression in aVR and PR-segment depression also may be present. (*See Figure 4.*) With chronic pericarditis, the ST-segment elevation resolves and widespread T-wave inversion develops. A large effusion may be associated with generalized low voltage and electrical alternans on ECG, although the latter is an uncommon finding.

The initial chest X-ray study usually demonstrates clear lung fields and a cardiac silhouette that may be mistaken for cardiomegaly. A large effusion may give the appearance of a water-bottle shaped heart. An echocardiogram is the diagnostic study of choice and demonstrates the effusion, quantifies the amount and thickness of the pericardium, and allows evaluation of the cardiac hemodynamic function.

Advanced techniques of non-surgical percutaneous pericardial biopsy, pericardioscopy, cytologic analysis of pericardial fluid, and molecular techniques (e.g., polymerase chain reaction and in situ hybridization) currently are available.[45-47] Such techniques significantly improve diagnosis of specific etiologies, but are not available immediately in the ED.

Following a cardiology consultation, stable patients with idiopathic or viral pericarditis may be treated as outpatients with nonsteroidal anti-inflammatory agents. Salicylates can be used, as can ibuprofen.[48] The patient should be placed on bed rest for 1-3 weeks with close follow-up. In stable patients with no signs of decompensation, pericardiocentesis may be deferred safely, particularly if removal of fluid is for diagnostic purposes only. In an unstable patient with evidence of tamponade, pericardiocentesis should be performed immediately. In addition, an initial fluid bolus should be given if hypotension is present. Antibiotics should be initiated until a bacterial source can be excluded. Recommended antibiotics include nafcillin and cefotaxime.[48] Corticosteroids have been found effective only with certain etiologies of pericarditis (i.e., autoreactive).[49] Steroids should not be used until a bacterial etiology can be excluded, and are not recommended in acute viral pericarditis. A pediatric cardiology consult is required, and all unstable patients should be admitted to an ICU setting.

Congestive Heart Failure (CHF)

CHF can be defined as an inability of cardiac output to meet the metabolic needs of the body. The causes of CHF in children tend to differ from those in adults. In developed countries, the main etiologies of CHF in children include congenital heart defects, cardiomyopathy, and myocardial dysfunction following heart defect repair.[50] Of these three etiologies, congenital heart defects are the most common. The incidence of congenital heart defects is approximately eight of every 1,000 live births.[33] Among children with congenital heart defects, the prevalence of CHF can be as high as 20%.[51] Cardiomyopathy has multiple eti-

Figure 4. ECG of Patient with Acute Pericarditis

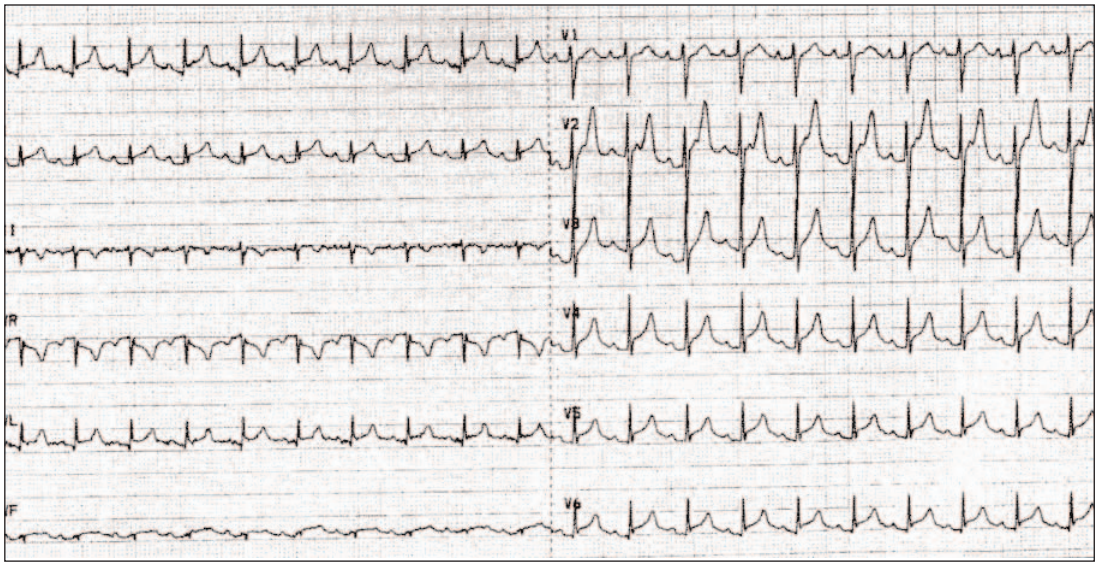

Figure courtesy of Amal Mattu, MD, University of Maryland School of Medicine.

ologies including genetic neuromuscular and metabolic disorders, collagen vascular diseases, hypothyroidism, hyperthyroidism, viral infections, drugs (e.g., anthracycline and cyclophosphamide), malnutrition, and hematologic disorders (e.g., sickle cell anemia). Studies have shown an annual incidence of cardiomyopathy ranging from 0.34-0.6 cases per 100,000 persons.[52,53] The prevalence of CHF following heart defect repair is unclear and appears to depend on the specific defect and repair performed.

Common symptoms of CHF in infancy include poor feeding, a prolonged feeding time, an increased respiratory rate and effort, and excessive sweating.[54,55] Physical exam findings for an infant with CHF classically include pallor, tachycardia, tachypnea, diaphoresis, and hepatomegaly. An S_3 gallop rhythm may be present. A sternal heave and laterally displaced point of maximal impulse indicates cardiomegaly. Rales are not always heard on auscultation of the lungs, and the absence of rales does not exclude the possibility of CHF. Peripheral edema is a very uncommon finding in infants. Failure to thrive in an infant is a hallmark of heart failure.[55]

Older children with heart failure often have poor exercise tolerance and fatigue in addition to poor appetite, growth failure, and increased respiratory rate and effort.[50] Atypical symptoms (e.g., abdominal pain, nausea, weight loss, and anorexia) also may occur in older children.[55] Signs of CHF in older children include tachycardia, tachypnea, rales on auscultation of the lungs, and hepatomegaly. Jugular venous distention and peripheral edema also may be present.

Laboratory tests in the ED should include a CBC count, and measurements of electrolytes, BUN, creatinine, and blood glucose levels. Hyponatremia may be present with fluid overload. An ABG measurement may help evaluate oxygenation and identify respiratory acidosis. Cardiac enzymes are indicated if ischemia or myocarditis is suspected.

A chest x-ray should be performed in all children evaluated for CHF. The chest x-ray helps identify pulmonary vascular congestion and cardiomegaly. A cardiothoracic ratio on the chest x-ray greater than 0.55 indicates cardiomegaly in infants, and a ratio greater than 0.5 is indicative in children older than 1 year.[56,57]

Other essential tests include an ECG and echocardiogram. The ECG may help identify causes of CHF, such as myocardial ischemia or dysrhythmias, or it may be nonspecific. It also can help identify heart chamber enlargement and electrolyte abnormalities. A low voltage in the QRS-complex may suggest myocarditis. Echocardiography is valuable in identifying structural lesions, valve abnormalities, potential effusions, ventricular wall motion abnormalities, and assessing the ejection fraction.

As in other situations involving unstable patients, or potentially unstable patients, the first priorities of management are assessment and stabilization of the patient's airway, breathing, and circulation. The patient should be provided supplemental oxygen, and ventilation assisted as necessary. Cardiac monitoring, pulse oximetry, and frequent blood pressure measurements should be initiated immediately and obtained regularly to assess for changes in hemodynamic status. Agents such as dopamine or dobutamine may be required for cardiovascular support. At low doses (2-5 mcg/kg/min), dopamine improves renal perfusion. Moderate doses of dopamine (5-10 mcg/kg/min) increase myocardial contractility via beta1-adrenergic receptor activation, and evoke vasoconstriction through alpha-adrenergic receptor effects. Doses greater than 10 mcg/kg/min have predominantly vasoconstrictive effects. The improvements in hemodynamic status with dopamine come at the expense of tachycardia, increased myocardial oxygen demand, and the risk of tachydysrhythmias. Dobutamine (2-15 mcg/kg/min) primarily functions as an inotrope and vasodilator. Secondary to the vasodilatory effects, dobutamine may not increase blood pressure significantly, and actually decreases peripheral resistance. Therefore, if significant hypotension is present, dobutamine should not be the primary inotrope initiated. Furosemide (1mg/kg IV) reduces preload and decreases fluid overload in patients with pulmonary edema. In some situations, vasodilators may help by decreasing afterload to improve cardiac output. Nitroprusside (0.5-10 mcg/kg/min IV)

has both venous and arterial effects and provides afterload reduction and venodilation. Intravenous nitroglycerin also may be used, but is primarily a venodilator.

In stable patients, following consultation with a cardiologist, digoxin is the indicated drug for inotropic support.[8,48] Digoxin acts to decrease the heart rate, increase myocardial contractility, and decreases sympathetic outflow.[58] The initial loading dose, or total digitalizing dose (TDD), is given during a 24-hour period, with half of the total dose given initially, then a quarter of the total dose given 8-12 hours after the first dose, and the remaining quarter dose given 8-12 hours after the second dose.[8,48] The oral TDD in full-term newborns is 30 mcg/kg, and in children older than 1 year the TDD is 30-50 mcg/kg. It is important to note that the intravenous TDD is only 75% of the oral TDD.

Other agents like angiotensin-converting enzyme (ACE) inhibitors have been shown to improve survival in adults with CHF and may have some benefit in children with CHF.[59] However, these agents generally are used for chronic maintenance and not in the ED setting.

Conclusions

Cardiac disorders are uncommon in children, and present infrequently to the ED. However, as illustrated in this discussion, significant morbidity and mortality are associated with these conditions if not recognized and treated promptly. Cardiac diseases must be considered in the differential of infants who present with acute illnesses in the first month of life and older children with suggestive signs and symptoms, and particularly children who appear ill and fail to respond to common management. Early recognition and aggressive management may significantly improve the child's chance for an optimal outcome.

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CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge.

To clarify confusion surrounding any questions answered incorrectly, please consult the source material. After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion. When your evaluation is received, a certificate will be mailed to you.

CME Objectives

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

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

b.) Understand the epidemiology, etiology, pathophysiology, historical and physical examination findings associated with the entity discussed;

c.) Be educated about how to correctly formulate a differential diagnosis and perform necessary diagnostic tests;

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

e.) Provide patients with any necessary discharge instructions.

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Physician CME Questions

1. Which of the following statements is true regarding paroxysmal supraventricular tachycardia (SVT) in infants and children?
   A. The history for infants with SVT may include non-specific complaints such as fussiness, lethargy, poor feeding, or pallor.
   B. Infants with SVT always have a fever and dehydration.
   C. Older children usually present with a cough and fever.
   D. SVT always is associated with WPW.

2. Which of the following statements is true regarding ECG changes associated with Wolff-Parkinson-White syndrome?
   A. A short PR interval is typical.
   B. A wide QRS typically occurs.
   C. A delta wave is typically present.
   D. All of the above are true.

3. Which of the following statements is true regarding the congenital prolonged QT syndrome, also known as long QT syndrome (LQTS)?
   A. It is a disorder of accelerated ventricular repolarization characterized by shortening of the QT interval.
   B. Jervell-Lange-Nielsen syndrome is an autosomal recessive form of prolonged QT syndrome associated with congenital deafness.
   C. Romano-Ward syndrome is an autosomal dominant form that is associated with deafness.
   D. The hallmark dysrhythmia is bradycardia.

4. A 4-year-old child presents with weakness, fatigue, and shortness of breath following a cold. On physical examination the child has tachypnea and tachycardia. The ECG shows a sinus tachycardia and a low-voltage QRS, fewer than 5 mm in all limb leads. What is this patient's most likely diagnosis?
   A. Myocarditis
   B. Pericarditis
   C. Long QT syndrome
   D. L-transposition of the great arteries

5. Causes of congenital heart blockage include which of the following conditions?
   A. Structural lesions such as L-transposition of the great arteries
   B. Maternal connective tissue disorders
   C. Acquired heart blockage from disorders such as Lyme disease, lupus, muscular dystrophies, Kawasaki disease, or rheumatic fever
   D. All of the above

6. Which of the following statements is true regarding the ECG of a patient with LQTS?
   A. The Bazett formula is not used to measure the QT interval.
   B. The QT interval on ECG represents the time from onset of depolarization to completion of repolarization.
   C. Lead III is generally the lead accepted for QTc calculations.
   D. T-wave alternans excludes the diagnosis of LQTS.

7. Which of the following statements is *not* true regarding the pericardium of a child?
   A. The pericardium consists of two layers: the visceral layer and the parietal layer.
   B. In a healthy person, a small amount (10-15 mL in a child) of serous fluid normally exists.
   C. When inflammation of the pericardium occurs, fluid accumulates between the two layers of the pericardium and forms a pericardial effusion.
   D. Chronic fluid accumulation in the pericardium leads to more serious complications than acute development of an effusion.

8. Which of the following children is at highest risk for sudden death?
   A. An 8-year-old with HCM and chest pain
   B. A 10-year-old with HCM and shortness of breath
   C. A 12-year-old with HCM and a syncopal episode
   D. A 4-year-old with HCM and a cough and fever

9. The physical examination of a child with HCM classically shows which of the following symptoms?
   A. A loud S_4 gallop with a harsh mid-systolic crescendo-decrescendo murmur
   B. A loud S_3 gallop without a murmur
   C. A murmur that decreases with Valsalva maneuvers or the standing position
   D. Squatting, isometric hand grip, or lying down will increase the murmur due to an increase in left ventricular end-diastolic volume.

10. Which of the following statements is *not* true regarding the Brugada syndrome?
    A. It is a genetically inherited disease with an autosomal dominant inheritance pattern and variable expression.
    B. Dysrhythmic episodes are unpredictable and may or may not terminate spontaneously.
    C. Medications, such as Type I antidysrhythmics, which affect the cardiac sodium channels, can provoke dysrhythmias.
    D. The ECG pattern, as originally described, consists of a LBBB with ST-segment depression in leads V_1-V_3.

Answer Key: 1. A; 2.D; 3.B; 4.A; 5.D; 6.B; 7.D; 8.C; 9.A; 10.D

In Future Issues: **The Febrile Child**

PEDIATRIC Emergency Medicine Reports
The Practical Journal of Pediatric Emergency Medicine

Cardiac Disorders

Drugs That Prolong the QT Interval

- Anti-arrhythmics (Class 1A and 3)
- Antiemetics (e.g., droperidol)
- Antifungals (e.g., ketoconazole)
- Antihistamines (e.g., astemizole, terfenadine)
- Antimicrobials (e.g., erythromycin, trimethoprim-sulfamethoxazole)
- Antipsychotics (e.g., haloperidol, risperidone)
- Organophosphate insecticides
- Phenothiazines (e.g., thioridazine)
- Promotility agents (e.g., cisapride)
- Tricyclic antidepressants (e.g., amitryptyline)

Factors Warranting ECG Evaluation for LQTS

- First-degree relatives of known LQTS carrier
- Emotional, exertional, or stress-induced syncope
- Syncope associated with chest pain or palpitations
- Recurrent episodes of light-headedness or presyncope
- Family history of syncope, seizures, or sudden death
- Sibling with sudden infant death syndrome
- Seizure of unknown etiology
- Unexplained near-drowning incident
- Bradycardia in infants
- Congenital deafness

Key: ECG = electrocardiogram. LQTS = long QT syndrome.

Normal Pediatric Vital Signs

AGE	RESPIRATORY RATE (BPM)	HEART RATE (BPM)	SYSTOLIC BP
Newborn (28 days)	30-60	120-160	50-70
Infant (1 month - 1 yr)	20-40	80-140	70-100
1-5 years	20-30	80-130	80-110
6-12 years	20-30	70-110	80-120
Adolescents	12-20	60-100	110-120

KEY: BP = blood pressure.

Typical ECG and Radiographic Findings in Cyanotic Congenital Heart Disease Based on Age of Presentation

AGE		ECG	X-RAY
Birth-first week of life	Transposition of the great vessels	RVH	PBF (inc)
First week of life	Total anomalous pulmonary venous return	RVH	PBF (inc)
1-4 weeks of life	Tricuspid atresia	LVH	PBF (dec)
	Severe pulmonic stenosis	RVH	PBF (dec)
1-12 weeks of life	Tetralogy of Fallot	RVH	PBF (dec)
Anytime in infancy	Truncus arteriosus	BVH	PBF (inc)

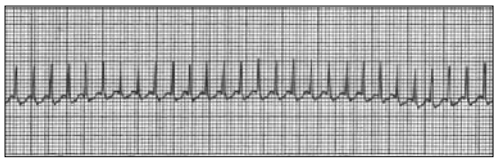
Key: RVH = right ventricular hypertrophy. PBF = pulmonary blood flow. inc = increased. LVH = left ventricular hypertrophy. dec = decreased. BVH = biventricular hypertrophy.

Age of Presentation, ECG Findings, and Pulmonary Blood Flow Patterns with Acyanotic Congenital Heart Disease

AGE		ECG	X-RAY
First week of life	Hypoplastic left heart syndrome	RVH	PBF (inc)
	Coarctation of the aorta	LVH	PBF (nl)
First 2-3 weeks of life	Complete AV canal	BVH or LVH	PBF (inc)
1-4 weeks of life	Patent ductus arteriosus	LVH	PBF (inc)
2-12 weeks of life	Ventricular septal defect	LVH	PBF (inc)

Key: RVH = right ventricular hypertrophy. PBF = pulmonary blood flow. inc = increased. LVH = left ventricular hypertrophy. dec = decreased. BVH = biventricular hypertrophy. AV = atrioventricular. nl = normal.

Rhythm Strip Demonstrating Supraventricular Tachycardia



ECG Demonstrating Long QT Syndrome

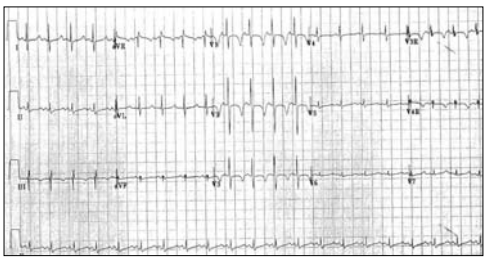


Figure 3. Note the prolonged QT interval as calculated with the Bazett formula: QTc = QT/square root of the preceding RR interval. QTc = 0.36/(square root of 0.44). QTc = 545 msec.

Courtesy of Nicholas Tsarouhas, MD, The Children's Hospital of Philadelphia

ECG Showing Wolff-Parkinson-White (WPW) Syndrome

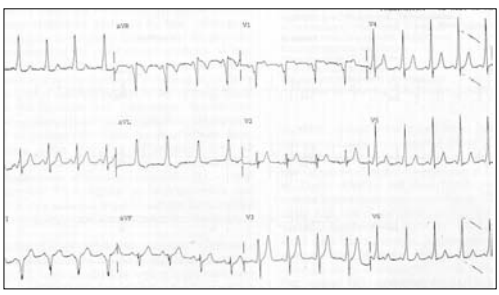


Figure. Note the delta waves.

ECG of Patient with Acute Pericarditis

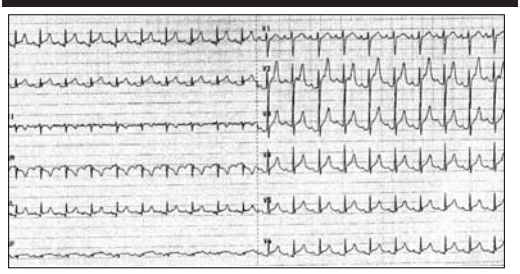


Figure courtesy of Amal Mattu, MD, University of Maryland School of Medicine.

Etiology of Heart Failure Categorized by Age of Common Occurrence

AGE	ETIOLOGY
Birth	• Heart muscle dysfunction - Asphyxia - Sepsis - Hypoglycemia - Hypocalcemia - Myocarditis • Structural defects - Tricuspid regurgitation - Pulmonary regurgitation - Systemic arteriovenous fistula • Paroxysmal supraventricular tachycardia • Congenital complete heart block
1 week	• Structural defects - Hypoplastic left heart - Aortic stenosis - Pulmonary stenosis - Total anomalous pulmonary venous return • Paroxysmal supraventricular tachycardia
1-6 weeks	• Structural defects - Coarctation of the aorta - Ventricular septal defects - Atrioventricular (AV) canal defects
Infancy	• Many conditions above may present later in infancy, but usually appear before 6 weeks. • Endocardial fibroelastosis • Hypertension • Endocrine disorders - Hypothyroidism - Adrenal insufficiency
Older children	• Valvular regurgitation • Postoperative complication of congenital heart disease • Myocarditis • Rheumatic fever • Bacterial endocarditis

Supplement to *Pediatric Emergency Medicine Reports*, December 2004: "Cardiac Disorders in the Pediatric Patient."
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Eye injuries present a significant challenge to emergency personnel. Patient stress and coexisting periorbital findings can complicate any evaluation, and many of the signs of serious injury may be quite subtle. Because the majority of eye injuries present between 10 p.m. and 4 a.m.,[1] when ophthalmology consultation is not available immediately in most hospitals, a tremendous burden is placed on the emergency health care provider to identify and manage potential vision-threatening disorders. The following is a review of ocular trauma with a focus on clinical findings, their implications, and management.

— The Editor

Initial Evaluation of the Traumatized Eye

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Epidemiology and Introduction

More than 2.5 million eye injuries occur in the United States annually.[2] From 1988- 2000, the United States Eye Injury Registry (USEIR) reported more than 10,000 major eye injuries,[3] an annual hospitalized incidence of 13.2 per 100,000.[4,5] Eighty percent of those injured were male, with a mean age of 29 years. Traditionally, the workplace presented the most common site of ocular injuries, but the USEIR reports 40% of these injuries occurring in the home, 13% in the workplace, and another 13% during recreation. Of these injuries, 31% were caused by a blunt object (e.g., rock, fist, baseball, or lumber), 18% by a sharp object, and 12 % were attributed to projectiles such as firearms and pellet (BB) guns.[6-8] Alcohol consumption was involved in 10% of cases, and 57% of all eye injuries occurred duirng warm weather months (spring and summer).[9] In the United States, 1.7% of all eye injuries will progress to permanent visual loss,[9] resulting in 60,000 new cases of monocular blindness related to trauma annually.[10]

Among the pediatric population (birth–16 years of age), eye injuries occur at an annual rate of 15.2 per 100,000 with males outnumbering females four to one. Males, ages 11-15, have the highest incidence of eye injuries at 23.7 per 100,000 annually, and 40% of these injuries are attributed to sports.[11] In victims of child abuse, an ocular injury will be the presenting injury in up to 40% of all cases[12] and in 95% of cases of shaken baby syndrome.[13]

Physical Exam

Dannenberg et al reported that 33% of all registered penetrating eye injuries were in the setting of multisystem trauma.[6] Thus, Advanced Trauma Life Support (ATLS) guidelines and a thorough general examination must precede any ocular evaluation. Secondly, any eye with a potential exposure to hazardous materials (e.g., acids, alkali, particulate matter, or heat) should be irri-

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gated prior to evaluation. Whenever possible, a thorough history should be obtained detailing the context, mechanism, time of injury, and use of eye protection. Past medical history should include previous ophthalmologic conditions, surgeries and trauma, current ocular medications, and pre-injury vision status (e.g., glasses/ contact lenses wearer, and visual acuity).

Figure 1 demonstrates a simple guide for the trauma examination of the eye. The first step is an assessment of visual acuity, which can be accomplished with a Snellen Eye Chart or near card. For pre-literate children, Allen cards or HOTV vision test letters may be used. For infants, fixation and smooth pursuit can be assessed using a hand light or colorful target. In many cases, the visual acuity may be too poor to assess by standard charting, and thus a gross assessment of visual acuity should be obtained using finger counting, motion perception, and light perception.

Most patients, regardless of mechanism, will complain of some degree of eye pain, but care must be taken to differentiate between globular and periorbital sources of pain. On inspection, examine the orbit for any step-off deformities or crepitus that may reveal an orbital wall fracture. Although there may be evidence of significant periorbital ecchymosis and edema, the lids still should be examined closely for lacerations and inverted when possible to remove retained foreign bodies. In the setting of prominent periorbital edema, a speculum or retractor may be employed to visualize the globe; however, if there is any evidence of a globe rupture (e.g., prolapse of intraocular contents, hemorrhagic chemosis, or enophthalmos), then this step should be foregone in favor of imaging studies and immediate ophthalmologic consultation. When possible, examine the anterior chamber for hyphema or pupil irregularities, findings that require further examination by slit lamp. Globe pain or foreign body sensation imply, at the minimum, corneal irritation and can be evaluated using fluorescein dye with a Wood's lamp to reveal corneal abrasions, ulcerations, or lacerations. All corneal exams that require fluorescein should be followed by a slit-lamp examination for occult foreign bodies.

Ocular motility should be assessed in both the vertical and horizontal planes. Classically, orbital wall fractures will present with deficits of ocular motility, but a proptotic eye with any motion deficit is the hallmark of a retrobulbar hemorrhage. For this reason, any defects in ocular motion should be evaluated further with a computerized tomography (CT) scan.

Finally, the posterior segment should be assessed by pupil reactivity to direct light and accommodation. Even in cases of prominent trauma to the anterior segment, the retina and optic nerve can be assessed by direct light to the affected eye and monitoring accommodation in the unaffected eye, known as the swinging flashlight test. A pupil that fails to constrict with direct light stimulation, but responds to stimulation of the other eye implies a defect in the posterior segment. Children may have a corresponding lack of a red reflex (Bruckner's test) in the affected eye. Both should be investigated further with an ophthalmoscope, when possible, or imaging studies such as ultrasound or CT scan.

In theory, any patient with a painless eye with intact visual acuity, a normally reactive pupil, and intact ocular motility in the absence of any anatomic deformities following a traumatic event may be discharged without further study and appropriate follow up. Nevertheless, an examining physician should have a low threshold for advanced study in the setting of high energy trauma or any projectiles where these findings may be masked by either trauma elsewhere or the delicate size of the particulate matter. Any new visual deficits or any impairment in mental status that complicates the examination should prompt a full evaluation.[10,14-17]

Classification of Eye Trauma

In 1993, a group of ophthalmologists from Birmingham, Ala., developed a standardized classification of eye trauma that would ease communication between peers and provide prognostic significance. In 1995, the International Society of Ocular Trauma, The USEIR, and the American Academy of Ophthalmology endorsed this new system. Ocular Trauma Classification (Tables 1 and 2) involves identifying the type of injury (open vs closed globe), the grade of the injury (visual acuity), pupil response, and

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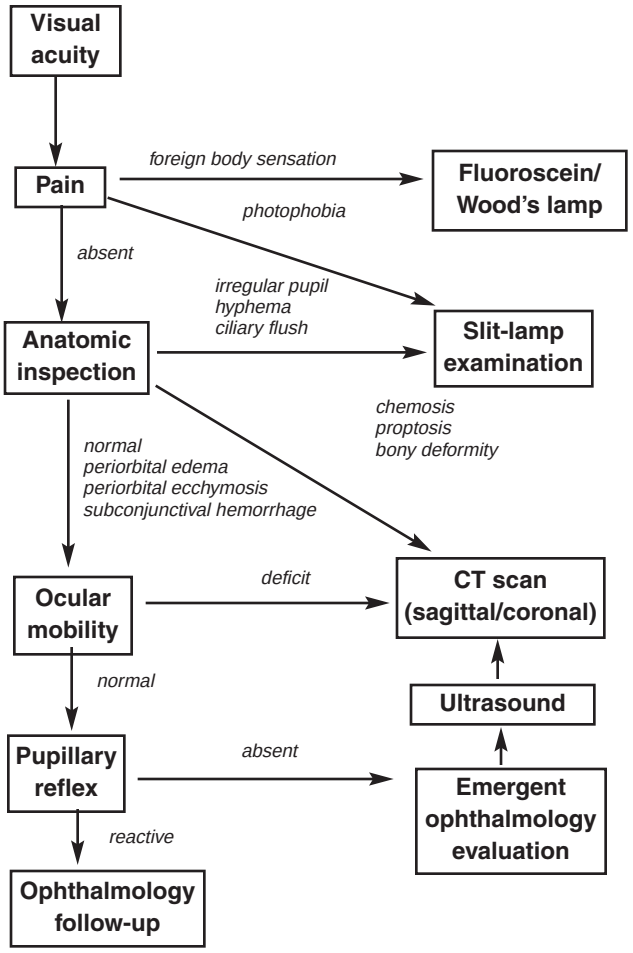
Figure 1. Trauma Examination of the Eye



the zone of the injury. Zone defines the anterior-posterior relationship of the injury on the globe.

An *open-globe injury* contains a full thickness wound of the eyewall (sclera or cornea). Open-globe injuries are then divided into lacerations (sharp mechanism) and ruptures (blunt mechanism). Lacerations are further divided into penetrating injuries (i.e., one entrance wound only), perforating injuries (i.e., two full thickness entrance and exit wounds), and intraocular foreign bodies. A *closed-globe injury* has no full thickness wound of the eyewall, and these injuries are divided into contusion (blunt mechanism), lamellar laceration (sharp mechanism), and superficial foreign body categories.

The Ocular Trauma Classification System provides a universal means of communicating the type and severity of an eye injury to an ophthalmology consultant or between emergency personal. In addition to quantifying an injury, this system, within the ophthalmology community, provides a foundation for management requirements as well as prognostic value for regaining vision.[18-21]

Superficial Injuries of the Eye

Superficial injuries of the eye, such as periorbital edema and ecchymosis, are very common sequelae of eye trauma, and often are quite dramatic on initial presentation. Despite the stress they may cause patients, these injuries involving the eyelids are quite benign in the absence of any globe injury and can be managed conservatively. For this reason, evaluation of lid injuries, regardless of mechanism, should be delayed for the evaluation and management of intraocular injuries.

Eyelid lacerations are divided into three types: vertical, horizontal, and canilicular (involving the medial lacrimal duct). Essentially, all eyelid laceration repairs may be delayed safely for 24-48 hours.[22-23] Thus, it is recommended that these injuries be irrigated to remove any foreign bodies, covered with a semi-moist pressure dressing, and ophthalmology evaluation arranged within one day. This is acceptable management for all pediatric and uncooperative patients as well.

Partial thickness injuries may be closed by emergency personnel, especially if ophthalmology evaluation will be delayed more than two days. However, all canalicular lacerations should be left to a specialist; the most superficial lacerations require proper exploration prior to closure.

Horizontal lacerations are more serious than vertical lacerations due to potentially significant impairment of the levator muscle. Evidence of ptosis or prolapsing fat indicates violation of the orbital septum, and, due to the close proximity of the levator aponeurosis, is highly suggestive of a levator injury. These repairs also should be delayed for a specialist.

Partial or full thickness vertical lacerations can be repaired by first approximating the tarsoconjunctival edge with simple, interrupted polyglycolic acid suture material with a D-1 needle.[22,24] The muscle layer can be sutured with 6-0 catgut, and the external eyelid can be closed with 6-0 silk. Once sutured, a pressure patch should be placed to reduce eye swelling and horizontal tension on the eyelid.[17] Although suture removal is recommended in 7-10 days, an ophthalmology follow-up evaluation should be scheduled in 2-3 days to ensure adequate wound healing.

Injury by Presentation: Pain

Pain is the hallmark of a corneal injury. Corneal abrasions are the single most common ocular condition evaluated in the ED,[25] as well as the most common eye injury related to airbag deployment.[26] For injuries confined to the superficial corneal epithelium, patients will present with a painful eye with conjunctival injection, ciliary flush, and tearing. Prior to examination, the eye should be anesthetized with tetracaine or proparacaine. Findings on fluorescein dye examination of the eye under a cobalt blue light or Wood's lamp will vary depending on the mechanism of the injury. Large abrasions with sharp borders are most often due to blunt trauma. Diffuse, punctuate lesions represent mild burn injuries from chemical exposure or ultraviolet light. Foreign bodies under the eyelid will produce multiple linear defects. Treatment includes removal of any foreign bodies, broad-spectrum antibiotics for the eye (e.g., ciprofloxacin/polymyxin B-trimethoprim), oral analgesia such as non-steriodal anti-inflammatory drugs (NSAIDS) or acetaminophen, tetanus prophylaxis as needed, and ophthalmology follow-up in 24-48 hours.[20]

Burns. Corneal burns account for up to 18% of all ocular trauma, with 68% of these injuries occurring in an occupational setting.[27] Burning agents can be divided into three broad cate-

gories (alkaline chemical agents, acidic chemical agents, and thermal agents), and the severity of the injury is related to the composition of the offending agent, volume, pH, and duration of exposure. Alkaline agents, accounting for 58% of all burn injuries, damage the cornea by saponifying the fatty acid components of the corneal cell membranes. This induces a liquefactive necrosis and allows deeper penetration into the globe. Ammonia is the most common alkaline agent causing eye injuries, but others include lye, potassium hydroxide, and lime.

Acids damage the cornea by inducing protein precipitation and denaturation into the epithelium and superficial stroma. The resultant coagulative necrosis tends to protect deeper intraocular structures. The most common acid-burning agents include sulfuric, sulfurous, hydrofluoric, and hydrochloric acids.[22]

Thermal injuries are usually the result of splash injuries from hot liquids or molten metal. A temperature greater than 100° C is required to injure the corneal epithelium. Cigarette burns to the cornea are a common injury in children ages 2-4 years. Usually the result of a child running into a cigarette at eye level; this burn pattern is rarely a manifestation of abuse.[10]

Eye burns are classified into four grades. A Grade I injury presents with hyperemia, conjuntival ecchymosis, and a cornea with a ground-glass appearance. A cigarette burn will produce this pattern of injury. In addition, a Grade II injury will have conjunctival chemosis and minor eschar formation. Grade III burns are deeper injuries characterized by conjunctival ischemia, thrombosed blood vessels, and reduced corneal clarity with preservation of the remaining anterior segment. Complete opacification of the cornea and lens, in addition to diffuse conjunctival ischemia and a grey iris in mydriasis, typify a Grade IV injury. Necrosis of the conjunctiva is accompanied by a dramatic inflammatory response that will induce further corneal ulceration within 4-6 weeks.

The mainstay of burn therapy is irrigation, preferably with a solution that is isotonic to the corneal stroma. Ideal solutions include lactated Ringers, balanced saline solution, or diphoterine; however, any irrigation (water, or normal saline) is far better than none at all. Upon presentation, patients should be anesthetized topically and then irrigated promptly for 15 minutes or 1L of fluid using a Morgan lens. Irrigation should not stop until normalization of the corneal pH (7.4). Lime and cement products will react with water to produce calcium hydroxide (pH 12.4), exacerbating their effect. These patients will require aggressive cleaning of the cul-de-sac with a cotton-tipped applicator dipped in 1% ethylenedinitrilotetraacetic acid (EDTA).[22]

Grade I and II burns may be discharged home on topical antibiotics and oral analgesics with ophthalmology follow-up within 24 hours. Grade III burns require admission for operative microscopy to determine the depth of tissue destruction. Grade IV lesions require steroids (oral or IV), as well as topical antibiotics prior to admission. Grade III lesions may be admitted to a local hospital with ophthalmology services; however, a Grade IV burn will most likely require a tertiary center with expertise in plastic reconstruction of the eye.[22]

Table 1. Open-Globe Injury Classification

TYPE

- Rupture
- Penetrating
- Intraocular foreign body
- Perforating
- Mixed

GRADE (VISUAL ACUITY)

- > 20/40
- 20/50 to 20/100
- 19/100 to 5/200
- 4/200 to light perception (LP)
- No light perception (NLP)

PUPIL

- *Negative*: Relative afferent pupillary defect absent in affected eye
- *Positive*: Relative afferent pupillary defect present in affected eye

ZONE

- I: Isolated to cornea (including corneoscleral limbus)
- II: Corneoscleral limbus to 5 mm posterior into the sclera
- III: Posterior to the anterior 5 mm of sclera

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Ultraviolet Keratitis. Ultraviolet keratitis is another form of exposure injury to the cornea classically known as snow blindness. Caused by the cumulative effect of ultraviolet light from electric arcs (welding) or tanning lamps, this disorder initially will present as a bilateral foreign body sensation and photophobia. Symptoms progress to severe bilateral eye pain with conjunctival erythema and tearing. Fluorescein staining reveals diffuse punctuate lesions on the cornea. Symptoms usually will resolve in 24-36 hours once removed from the inciting agent. Management includes topical antibiotics and narcotic analgesia.[20]

Lacerations. A corneal laceration is defined as a traumatic disruption of the cornea involving all three layers (epithelium, stroma, and endothelium) due to a penetrating mechanism; 75% of these injuries in children were caused by a sharp object.[28] Like other corneal injuries, pain is the most common presenting complaint. An examination of the eye may reveal a shallow anterior chamber or teardrop-shaped pupil directed toward the laceration. Prolapse of intraocular contents is a confirmatory finding; however, in many cases, the eye will appear normal due to the self-sealing properties of the elastic corneal stroma. Fluorescein evaluation of the cornea under blue light will reveal a bright green stream (Seidel test) caused by egress of fluid from the anterior chamber. A slit-lamp examination on oblique illumination may reveal the length and depth of a laceration. Nevertheless, a normal fluorescein study does not completely eliminate the possibil-

Table 2. Closed-Globe Injury Classification

TYPE

- Contusion
- Lamellar laceration
- Superficial foreign body
- Mixed

GRADE (VISUAL ACUITY)

- > 20/40
- 20/50 to 20/100
- 19/100 to 5/200
- 4/200 to light perception (LP)
- No light perception (NLP)

PUPIL

- *Negative:* Relative afferent pupillary defect absent in affected eye
- *Positive:* Relative afferent pupillary defect present in affected eye

ZONE

- I: External (limited to bulbar conjunctiva, sclera, and cornea)
- II: Anterior segment
- III: Posterior segment

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ity of a corneal laceration. Partial-thickness lacerations can be managed like corneal abrasions.[20,29]

Foreign Bodies. Forty-one percent of all open-globe injuries involve an intraocular foreign body.[30] The majority of these projectiles (80%) are generated in the setting of hammering either a chisel, nail, or stone.[30,31] Other causes of ocular foreign bodies include motor vehicle collisions, explosions, projectile weapons, and activities involving power tools such as drilling. Magnetic metals (iron, lead, and copper) account for the majority of intraocular foreign bodies (IOFB); however, nonmetallic items such as glass, wood, and especially organic materials in pastoral settings, may be a nidus for a disastrous Bacillus infection. More than 80% of IOFBs will penetrate the cornea and settle within the vitreous.[32] Other common sites for foreign bodies include the underside of the upper lid, the infracorneal recess within the lid fissure, and the inferior angle of the anterior chamber.[33]

Missed foreign bodies constitute 56% of all eye trauma-related legal claims, and therefore, IOFBs should be excluded in any patient with a suspicious mechanism. One-fifth of all patients with ocular foreign bodies will have no pain and intact visual acuity.[30] Many patients, especially children, will be unaware of the exposure, resulting in a significant delay to presentation. Like other corneal injuries, pain or foreign body sensation will be the most common complaint, but visual deficits will vary depending on the size of the projectile, extent of the injury, and delay in presentation. Fluorescein examination of the cornea should be followed by a slit lamp examination of the cornea, anterior chamber, and the area under the lids. Any superficial foreign body may be removed with either a cotton-tipped applicator (lid) or a 27-gauge needle.[34]

Assault-related Injuries. Dannenberg et al reported that one-third of all occupational eye injuries[11] and 56% of all assault-related injuries involve the sclera.[5] Scleral rupture is a traumatic disruption of the sclera commonly in the supranasal quadrant at the insertions of the rectus muscles on the globe. Most often this injury is the result of a blunt mechanism and has a prevalence of 3.5% in blunt trauma.[35] In the absence of obvious prolapse of intraocular contents, evidence of a scleral rupture may be quite subtle. Low intraocular pressures (< 6 mmHg), reduced visual acuity on initial presentation, an afferent pupillary defect, and a shallow anterior chamber are clinical findings highly specific to scleral ruptures, and thus, warrant further investigation. In addition, one-quarter of hyphemas and 22% of bloody chemosis cases are associated with a full-thickness scleral injury. [12,30,36-38]

Because full thickness injuries of the eyewall (e.g., corneal lacerations, scleral rupture, and intraocular foreign bodies) are open-globe injuries, any suspicion of such requires prompt ophthalmology evaluation. CT imaging (axial and coronal views) may be diagnostic (sensitivity, 93%; specificity, 75%; PPV 95%)[39,40] of scleral injuries, but is the modality of choice to detect intraocular foreign bodies (sensitivity 100% for objects more than 0.06 mm).[3,41] Magnetic resonance imaging (MRI) is contraindicated for IOFB due to migration of metallic fragments.

The examiner should avoid placing any pressure on a suspected globe rupture to limit further prolapse of intraocular content; therefore, tonometry should be deferred despite the value of confirming low intraocular pressures. An eye shield should be placed promptly pending further evaluation. Tetanus vaccinations should be updated, and prophylactic antibiotics should be started in the ED. Up to 16% of open-globe injuries may progress to bacterial endophthalmitis caused by Staphylococcus, Streptococcus, or Bacillus species, which are particularly aggressive. Current antibiotic recommendations include ceftazidime (1 g every 8 hours) and vancomycin (1 g every 12 hours). Ciprofloxacin (400 mg every 12 hours) and vancomycin may be used in cases of penicillin allergy.[42]

Metallic intraocular foreign bodies may induce similar localized inflammatory reactions known as metallosis (chalcosis for copper IOFB; siderosis for iron IOFB), which can progress to hypopyon, retinal detachment, and irreversible visual deficits within hours. Therefore, prompt surgical evaluation is the definitive management for open-globe injuries, ideally performed by an ophthalmology specialist familiar with anatomical reconstruction to optimize visual outcomes.

Injury by Presentation: Diplopia

The two major components of the external eye are the extraocular muscles and the bones of the orbit. Post-traumatic deficits in ocular motility are suggestive of extraocular injuries leading to the common complaint of binocular diplopia.

The conical orbit comprises six bones that integrate to create a floor (maxilla), roof (frontal bone), lateral wall (sphenoid and zygoma), and medial wall (maxilla, lacrimal bone, ethmoid, and sphenoid). Orbital wall fractures are largely the result of blunt trauma from an object greater in diameter than the orbital rim such as a fist, ball, or dashboard. The energy from the trauma is dispersed across the orbital rim and elastic globe, which will transmit this force into the orbit. The evolutionary shape of the orbit minimizes the effect of these sudden elevations in intraocular pressure by collapsing, like a safety valve, at its weakest points – the floor and ethmoid aspect of medial wall (lamina papyracea). Sports-related injuries (e.g., baseball, softball, and soccer) are the most common causes of orbital wall fractures in children.[43] In adults, these injuries often are attributed to motor vehicle collisions, assault with a blunt object, and falls.[44]

Blow-out Fractures. Impairment of upward gaze due to entrapment of the inferior rectus muscle is the classic presentation of a blow-out fracture, a fracture of the floor or medial wall of the orbit. Patients commonly will complain of binocular diplopia with periorbital edema, ecchymosis, and an acutely proptotic eye. This can be associated with periorbital crepitus due to subcutaneous emphysema,[45] as well as hypoesthesias of the upper lip and maxillary teeth secondary to infraorbital nerve injury. Enophthalmos, usually a delayed finding, may develop proportional to the size of the maxillary fracture.

Blow-in Fractures. A blow-in fracture is a fracture of the orbital roof (frontal bone) caused by high-velocity blunt trauma directed at the superior orbital rim. A rare fracture (5% of all facial fractures), orbital roof fractures have a strong association with other facial fractures (73% of cases) and intracranial injuries (44% of cases).[46] Because this fracture reduces orbital space, exophthalmos will persist after the periorbital edema has regressed. As in a blow-out fracture, patients will complain of diplopia, but this may be associated with supraorbital hypoesthesia and ptosis secondary to entrapment of the levator palpabrae. In rare cases, intracranial contents may herniate inferiorly, and cerebrospinal fluid (CSF) may leak from the eye.

A CT scan with fine-cut axial and coronal images of the face and orbit is the modality of choice for the diagnosis of orbital bone fractures. Due to the bony immaturity, children will have a higher rate of false negatives despite obvious clinical signs, and management should proceed as if a fracture is present.[47] Surgery is the definitive treatment, and indications include fractures greater than 50% of the orbital floor, extraocular muscle entrapment, and enophthalmos greater than 2 mm. In most cases, intervention will be delayed 7-10 days to allow reduction of post-traumatic edema; thus, emergent management is conservative. Ice packs with head elevation are primary, and the patient should be instructed to avoid nose blowing. Nasal decongestants and a 10-day course of antibiotic prophylaxis against sinusitis may be initiated. Because practice varies, ophthalmology should be notified, but does not require urgent consultation unless the patient demonstrates evidence of an orbital roof fracture, elevated orbital pressures, or stimulation of the oculocardiac reflex (bradycardia/hypotension).[17,22,33,40,43-45,48-52]

Extraocular Muscular Avulsion. Another cause of post-traumatic diplopia is extraocular muscle avulsion. The superior oblique most often is affected due to the close proximity of the trochlea to the superior orbital rim. Penetrating mechanisms are almost always the cause including projectiles (e.g., pellets [BBs], bullets), bone fragments from orbital wall injuries, and dog bites in children. Presentation will vary depending upon the degree of transection. Patients will complain of diplopia, and examination will reveal a motion deficit or, in cases of complete transection, deviation of the globe. CT imaging may confirm a muscle injury, and isolated injuries may be managed conservatively with ophthalmology follow-up within 24-48 hours.[33,44,52]

Retrobulbar Hemorrhage. Because the orbit is a rigid, enclosed space, it is susceptible to a compartment syndrome. Retrobulbar hemorrhage is a very rare injury with serious consequences. Unfortunately studies in the United Kingdom have suggested that an overwhelming majority (73%) of emergency personnel are not familiar with the presentation and management of this disorder.[54] Post-traumatic retrobulbar hemorrhage will occur in the setting of either penetrating or blunt mechanism. Like compartment syndrome elsewhere, pain is the hallmark of this injury with rigid, rock-hard proptosis, which may be accompanied by painful ocular motility progressing to diplopia. Increasing intraocular pressures will reduce perfusion pressures to the optic nerve causing a progressive visual deficit and an afferent pupillary defect that is irreversible within 1-2 hours of onset.[55]

Management begins with recognition of a painful proptotic eye. CT imaging will confirm the presence of an intraorbital hematoma, but if the patient has any visual or pupillary deficits on presentation, immediate intervention is necessary. Concerning presentations with negative CT scans can be assessed with tonometry to confirm normal ocular pressures prior to discharge.

An ophthalmologist should be notified. The patient's head should be elevated, and he should be instructed to avoid any valsalva maneuvers (e.g., coughing, sneezing, and straining). Mannitol, acetazolamide, and steroids should be given to reduce intraocular pressures and protect the optic nerve. If symptoms continue to progress, a lateral canthotomy should be performed with lateral cantholysis of the inferior crus. If intraocular pressures are normalized within a timely manner, symptoms typically resolve within hours, including a return to baseline vision.[44-46,56-58]

Injury by Presentation: Photophobia

The uvea is composed of the iris, ciliary body, and choroids. Because its primary role is pupillary regulation, injuries usually will present as pain with pupillary constriction, and patients will complain of photophobia.

Traumatic Iritis. Traumatic iritis is acute inflammation of the anterior uvea, secondary to blunt or penetrating trauma. The chief complaint will be photophobia, but patients also may complain of headache, blurred vision, or light sensitivity due to traumatic mydriasis. They also will have pain in the affected eye when a light is shone in the unaffected eye, termed consensual photophobia. Slit-lamp examination will reveal ciliary flush with characteristic cell and flare. Treatment consists of symptomatic

relief with a cycloplegic agent such as 5% homatropine and ophthalmology follow-up in 2-3 days.[16,33]

Sympathetic Ophthalmia. Sympathetic ophthalmia is a bilateral, autoimmune, granulomatous uveitis incited by penetrating injuries to the eye. The incidence ranges from 1% in open-globe injuries[59] to 10 cases per 100,000 penetrating eye wounds.[61] Onset can range from five days to 66 years, but the average is 4-8 weeks following trauma.[61] Patients will complain of photophobia with tearing and visual deficits, but the key element in the history is the bilateral presentation (consensual photophobia) despite sustaining an injury to only one eye. If left untreated, the inflammatory conditions related to sympathetic ophthalmia can progress rapidly to profound visual impairment from secondary cataract, glaucoma, or retinal detachment. Slit-lamp examination will reveal cell and flare, and the standard of care in the ED is steroid therapy with prompt ophthalmology follow-up.[62]

Blurred Vision with Intact Afferent Pupillary Reflex

A normal pupillary response is indicative of an intact posterior segment, mainly the retina and optic nerve. Visual deficits with normal accommodation reflect injury anterior to the retina. These injuries obscure the visual axis to the posterior eye; therefore, patients will complain of aberrant vision without a complete deficit.

Traumatic Hyphema. Traumatic hyphema is the presence of blood in the anterior chamber secondary to trauma. The annual incidence is 17 to 20 per 100,000 with a peak age range of 10-20.[63,64] Two-thirds of all hyphemas are due to blunt mechanisms with 44% percent due to assaults,[5,62] 44% sports-related,[59,65] and the remainder associated with motor vehicle collisions and the workplace.[1] Hyphemas are also the most common eye injury associated with paintball trauma.[66] In the pediatric population, 65% of these injuries are sports-related.

A hyphema results from shearing forces on the uvea, leading to disruption of the iris, ciliary body, or choroid with subsequent hemorrhage into the anterior chamber. It is the hallmark of a severe ocular injury, and 25% are associated with a scleral rupture.[35] Most patients will present with eye pain and visual deficits, but the degree of visual impairment will be proportional to the percentage of the anterior chamber occupied by blood. For that reason, hyphemas are graded:

- A Grade I lesion occupies less than one-third of the anterior chamber;
- a Grade II lesion occupies between one-third and one-half of the anterior chamber;
- a Grade III lesion occupies more than half of the anterior chamber; and
- a Grade IV lesion is a total hyphema (black ball or eight ball).[63]

The severity of hyphemas is related to the corresponding complications. One-third of hyphemas are associated with elevated intraocular pressures, which may progress to acute angle-closure glaucoma secondary to obstruction of the trebecular meshwork by blood products or direct compression of outflow channels. Sickle cell disease or trait may exacerbate this effect, progressing to dramatic increases in intraocular pressure even in low volume hyphemas. Persistently elevated intraocular pressures can lead to optic nerve atrophy, and irreversible visual deficits. Finally, large hyphemas may cause corneal blood staining, a yellow discoloration of the cornea contributing to permanent visual deficits.

The first step in examining a hyphema is to rule out an open-globe injury. In the absence of any signs or symptoms of a full-thickness eyewall injury, a thorough history should be obtained, focusing on a medical history of blood disorders such as sickle cell disease, leukemia, Von Willebrand disease, or hemophilia. The history also should note any use of anticoagulant or antiplatelet medications such as aspirin, NSAIDS, clopidogrel, warfarin, or enoxaparin. In children, any delay in presentation or historical inconsistency warrants concern of child abuse.

A CT scan may be necessary to rule out associated orbital fractures or open-globe injury. All hyphemas require slit-lamp examination to evaluate the anterior chamber, and tonometry once an open-globe injury is ruled out.

Twenty-five percent (Grade I) to 67% (Grade III) of hyphemas will rebleed, usually 2-5 days post-trauma.[63] For this reason, management is directed at limiting rebleeding and subsequent elevations in intraocular pressure. This management may vary among specialists; ophthalmology should be notified prior to initiating treatment. The patient should be positioned with the head of the bed elevated to promote settling of the hyphema, and a rigid shield should be placed over the eye to prevent further injury. Topical cycloplegics, such as 1% atropine and topical steroids, may be given to relieve photophobia and intraocular inflammation. To limit rebleeding, recommendations include a five-day regimen of either aminocaproic acid or prednisone. Aminocaproic acid is favored in the setting of sickle cell disease, and may require concomitant, antiemetic therapy. For elevated intraocular pressures (> 25 mmHg), topical beta-adrenergic antagonists, alpha-adrenergic agonists, and carbonic anhydrase inhibitors are recommended. Table 3 outlines indications for outpatient management of hyphemas. If a patient is discharged, he should be instructed to discontinue all anticoagulant/antiplatelet medication, avoid any rigorous activity, and follow up with ophthalmology within 24 hours.[12,62,63,67]

Cyclodialysis. Cyclodialysis is a disruption of the ciliary muscle attachment at the sclera that results in a cleft allowing extravasation of aqueous humor into a potential space within the choroids. It will present in an estimated 4% of blunt eye traumas.[67] Patients will complain of poor vision with possible pain, erythema, and tearing. Because of the loss of volume, the anterior chamber will appear shallow on slit-lamp examination, and intraocular pressures will be low. This hypotony can lead to corneal edema, choroidal detachment, and optic disc edema. All cases of cyclodialysis will require surgical correction, but most of the sequelae are chronic manifestations. Normal vision still can be restored within eight weeks of the injury. Thus, patients may be discharged home with topical cycloplegics, and follow-up with an ophthalmologist within 3-4 days.[58,68]

Lens Subluxation. Lens subluxation is a partial displacement of the lens off the visual axis due to disruption of the lens zonule fibers that anchor it to the ciliary body. Blunt trauma is the most common mechanism. A complete dissociation of the lens from the ciliary body is known as a lens dislocation.

Patients may present with reduced visual acuity, monocular diplopia, or a visual glare, but symptoms may be delayed until months after the initial trauma. Conditions that predispose to lens dislocation with minimal trauma include Marfan's syndrome, homocystinuria, syphilis, Weill-Marchesani syndrome, and retinitis pigmentosa. A slit-lamp examination with pupillary dilation will reveal a displaced lens, prolapse of vitreous into the anterior chamber, or iridodonesis (a trembling of the iris with rapid eye movements). In children, an asymmetric red reflex will characterize lens abnormalities. Subluxation and posterior dislocation are benign conditions that can be managed with a corrective lens. Anterior displacement of a dislocated lens into the anterior chamber is an ocular emergency leading to pupillary obstruction and elevated intraocular pressures (pupillary block glaucoma). Any concern for anterior dislocation requires immediate ophthalmology evaluation for surgical correction, assessment of intraocular pressures, and management with topical beta-adrenergic antagonists, alpha-adrenergic agonists, or carbonic anhydrase inhibitors.[16,17,69]

Traumatic Cataracts. A traumatic cataract results from swelling and opacification of the lens secondary to disruption of the external capsule. This is the most common lens injury in trauma occurring in 39% of open-globe injuries and 11% of closed-globe injuries.[69] In addition, traumatic cataracts accounts for 10% of eye injuries secondary to assault,[6] 32% of workplace-related injuries,[1] and 12% of sports-related injuries.[2] Symptoms tend to be delayed in onset (weeks to months) with a common complaint of a unilateral, progressive blurring of the vision. Because the majority are associated with open-globe injuries and associated vitreoretinal injuries, many traumatic cataracts are diagnosed and managed in the operating room. Chronic, isolated injuries will be apparent on slit-lamp examination and are managed conservatively with ophthalmology follow-up in 2-3 days. Because a swollen, deformed lens may obstruct the pupil lending to pupillary block glaucoma, intraocular pressures should be assessed prior to discharge.[16,70]

Vitreous Hemorrhage. Vitreous hemorrhage is the extravasation of blood into the vitreous space. It is one of the most common post-traumatic ocular injuries, accounting for 40% of assault-related eye injuries,[6] 42% of work-related eye injuries,[1] and more than 60% of paintball-related injuries.[65] Vitreous hemorrhage also has a strong association with shaken baby syndrome.[17] The sources of bleeding include the iris, ciliary body, choroids, and retina. Although the mechanism may be blunt or penetrating, vitreous hemorrhage most often is associated with a closed-globe injury. Visual deficits represent the most common presentation, with patients complaining of a haze, floaters, cobwebs, smoke signals, or simply a shadowy appearance to their vision. This may be associated with pain or photophobia, depending on the site of disruption. Work-up is directed at excluding retinal detachment. Direct ophthalmoscopy should be employed, but may be obscured by blood. Ultrasound is useful in diagnosing a corresponding retinal detachment, but CT imaging will provide additional information on the integrity of the choroids, sclera, or any evidence of intraocular foreign body. Treatment of an isolated vitreous hemorrhage is conservative with symptomatic relief of pain as needed. Patients can be discharged home with instructions for bed rest with head elevated and ophthalmology follow-up in 2-3 days.

Complications related to vitreous hemorrhage include retinal detachment secondary to traction from the hemorrhage and ghost cell glaucoma, elevated intraocular pressures secondary to obstruction of aqueous humor outflow tracts by the byproducts of hemoglobin degradation. Onset is usually 1-3 weeks post-trauma, and patients will present with a painful eye with a beige collection of cells within the anterior chamber that often is confused for a hypopion. The presence of ghost-cells, elevated intraocular pressures, and impending retinal detachment are all indications for immediate surgical evacuation of the vitreous.[33,71,72]

Table 3. Traumatic Hyphema: Indications for Outpatient Management

- No associated ocular injury mandating hospitalization
- Hyphema less than Grade II
- Intraocular pressure < 35 mmHg
- No history of sickle cell disease, blood dyscrasias, or coagulopathic disorders
- No concern regarding the safety of home environment (including child abuse), ability to comply with limited activity, ability to comply with medication regimen, or ability to follow up with ophthalmology within 24 hours

Vision Loss with an Afferent Pupillary Defect

Trauma to the posterior segment of the eye (retina/optic nerve) may impair the pupillary light reflex, causing a pupil that will fail to constrict with direct light stimulation, known as a relative afferent pupillary defect.[73] In addition, posterior segment injuries also may have profound visual deficits, including complete loss of vision.

Retinal Detachment. Retinal detachment is a separation of the superficial neurosensory layer of the retina from the underlying pigmented layer. Fifteen percent of all retinal detachments are caused by trauma with blunt mechanisms (70-85% of cases), the most common etiology.[74] The development of a detached retina is a three-step process. The first is a break in the retinal layer generated during the initial trauma. Retinal dialysis (detachment from ciliary body) is the most common of these precipitants, but others include operculated holes, horseshoe flaps, or areas of necrosis. Secondly, fluid will seep into this defect and weaken the bond between the two superficial layers. Finally, traction must be provided to separate the two layers. During the initial trauma, this is provided by a blunt mechanism and the subsequent elastic recoil of the globe that places traction at the vitreous base. In many instances, this traction is a delayed process caused by the contraction of the vitreous secondary to healing.

Due to the separation from its blood supply in the choroids, the separated layer becomes ischemic and irreversibly atrophies.

In many cases, a retinal detachment will be delayed months to years after the initial injury. Patients will complain of seeing light flashes progressing to a "falling curtain of darkness." This may lead to profound visual deficits with a relative afferent pupillary defect, an unreactive iris to direct stimulation that constricts with stimulation of the opposite eye (consensual response). Examination in children may reveal a diminished red reflex. On direct ophthalmoscopy, detachments appear as grayish billowing of the retina; however, many smaller or peripheral detachments may not be seen with simple fundoscopy (even after dilating the pupil). Ultrasound will reveal a smooth membrane within the vitreous cavity or a triangular shape extending from the optic disc in cases of total detachment. Immediate surgical intervention is indicated, thus, any pupillary deficit or suspicion of retinal detachment requires immediate ophthalmology evaluation.[16,71,72,75-77]

Because of the high incidence of ocular findings in child abuse, retinal detachment in children should raise suspicion, but retinal hemorrhage in children younger than 3 years is pathognomonic for shaken baby syndrome.

Optic Nerve Injuries. Optic nerve injuries have a 7% incidence in major trauma[9] and will result in permanent visual loss in 50% of cases.[78] The optic nerve can be injured by two major mechanisms. Direct injuries cause trauma to the nerve usually by penetrating, open-globe mechanisms such as bone fragments, foreign bodies, or projectiles (e.g., bullets, BBs). Walsh and Hoyt described indirect injuries as a "traumatic loss of vision which occurs without external or initial ophthalmoscopic evidence of injury to the eye or the nerve."[79] These injuries are caused by transmitted forces upon the optic nerve due to bony apposition or globe mobility. Indirect injuries are the most common cause of traumatic optic neuropathy usually attributed to blunt, deceleration injuries with impact at the supraorbital or frontal regions of the head. Such forces are encountered in motor vehicle collisions, bicycle accidents, assaults, and falls, but optic nerve injuries also have been described in lower energy mechanisms such as falling debris and skateboard accidents.[77,80,81]

The optic nerve can be divided into four anatomic sections: the intraocular portion that we recognize as the optic disc; a mobile intraorbital section; a fixed intracanalicular section that is accompanied by the ophthalmic artery as it runs through the optic canal; and an intracranial section that joins the complimentary optic nerve to form the optic chiasm. The intracanalicular portion is the most common site of injury and follows a dual insult paradigm. The primary insult, at the time of initial trauma, results from shearing forces due to a mobile globe and intraorbital nerve section placing stress on an immobile intracanalicular section and vessels. This results in permanent axonal injury, as well as, disruption of the vasculature with possible hemorrhage. This loss of blood supply produces a secondary ischemia and circulation of toxic metabolites such as free radicals, bradykinin, and calcium. Any axons that survive the initial insult may succumb to the secondary effects of ischemia, contributing to the delayed presentation of optic nerve injury.[75,77,79,82,83]

The most common presentation for traumatic optic neuropathy is a visual deficit with an afferent pupillary defect (Marcus-Gunn pupil) on exam. Typically, patients present with vision of 20/400 or less, with 10% having a delayed-onset of symptoms. Visual fields deficits are indicative of intracranial injury, but in most instances will be difficult to elicit; more than 50% of optic nerve injuries are associated with a loss of consciousness.[50,77]

Any deficits in vision should raise suspicion of an optic nerve injury. Again, an afferent pupillary defect is diagnostic of a posterior segment injury and requires immediate ophthalmologic evaluation. Delayed loss of vision can be described as a lucid interval before normal vision rapidly fades. Often it is associated with compression from an expanding hematoma; in most cases, full visual acuity is returned with prompt surgical decompression. This rapid deterioration of vision has a good prognosis, while delayed regressions during a 1-2 week period are associated with axonal atrophy and suggest more permanent visual deficits.

The physical exam should focus on reversible causes of optic nerve injury—specifically signs of retrobulbar hemorrhage such as a rigid globe and diplopia. Ophthalmoscopic examination will vary depending upon the location of the injury. Anterior injuries involving the intraocular portion of the nerve will demonstrate an edematous retina with a pale optic disc if the central retinal artery is involved. Otherwise, an avulsed intraocular optic nerve will produce a hemorrhagic ring on the fundus, with the optic disc appearing as a deep round pit. Posterior injuries involving the intracanalicular portion of the nerve will appear normal on initial presentation. Only after 3-6 weeks will the disc appear pale and atrophic. In more subtle cases, be aware of the close association of optic nerve injury with midface fractures (2.5% incidence). All patients with suspected ocular nerve injuries should receive a CT scan to evaluate for fractures, as well as, pathologic nerve sheath hematomas or retrobulbar hemorrhages.[16,77,84,85]

In 1998, The International Optic Nerve Trauma Study[86] attempted to evaluate the current management recommendations for optic nerve trauma, high-dose steroids vs. surgical decompression. Researchers concluded that neither improved visual outcomes, but this study was retrospective and grossly lacked power (n=133). There has never been a prospective, randomized trial to determine efficacy in the management of these injuries; one of the reasons is that the incidence of the disorder is too low to generate statistical significance within a given community. Thus, high-dose steroids commonly are used in some centers. Surgical decompression also is employed, but due to a lack of a universal protocol, the use of these therapies will vary by region, therefore, a consulting ophthalmologist should be notified prior to initiating any treatments.[77,79,81]

Prognosis and Conclusion

In general, post-traumatic eye injuries with poor visual acuity on initial presentation have a lower probability of regaining baseline vision. Pieramici et al demonstrated that 88% of patients with post-traumatic visual acuity of 20/40 or better retained that degree of acuity on follow-up visits. In contrast, 79% of patients with a post-traumatic inability to perceive light on initial evalua-

tion required enucleation of the affected eye.[87] Much of this prognostic data related to eye trauma focused on open-globe injuries in recent years; other initial indicators of poor visual outcome in the setting of these injuries include the length of the eye-wall defect, presence of an afferent pupillary defect, prolapse of intraocular contents, and finally, the presence of a hyphema.[88,89]

Pitfalls in assessing and managing eye trauma are related to the rarity of true vision-threatening injuries. The majority of the eye injuries evaluated by emergency health care providers will be benign, but complacency can be overcome by maintaining a systematic approach to all eye injuries, while being mindful of the hallmarks of severe injuries such as rigid proptosis and the absence of a pupillary response. As a final precaution, all eye injuries, regardless of their triviality, should be referred for follow-up ophthalmology evaluation.

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CE/CME Objectives

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

a.) recognize or increase suspicion for traumatic injuries that present to the emergency department;

b) describe the various modalities used to identify different traumatic conditions covered in the newsletter;

c) describe how to correctly and quickly stabilize, and then to manage patients with the particular condition covered in the newsletter; and

d) identify both likely and rare complications that may occur with traumatic injuries.

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

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CE/CME Questions

1. Which of the following conditions is *not* characterized as an open-globe injury?
   A. Intraocular foreign body
   B. Corneal abrasion
   C. Corneal laceration
   D. Scleral rupture

2. Which of the following conditions is *not* associated with diplopia?
   A. Orbital wall fractures
   B. Lens subluxation
   C. Retrobulbar hemorrhage
   D. Retinal detachment

3. A rigid rock-hard proptotic eye is characteristic of what post-traumatic eye injury?
   A. Retrobulbar hemorrhage
   B. Traumatic hyphema
   C. Scleral rupture
   D. Orbital wall fracture

4. Which of the following conditions is *not* associated with elevated intraocular pressures?
   A. Traumatic hyphema
   B. Vitreous hemorrhage
   C. Anterior lens dislocation
   D. Posterior lens dislocation

5. Which of the following eye injuries is the most specific for shaken baby syndrome?
   A. Retinal hemorrhage
   B. Retinal detachment
   C. Traumatic optic neuropathy
   D. Vitreous hemorrhage

6. Which of the following is *not* part of the anterior segment of the eye?
   A. Lens
   B. Iris
   C. Retina
   D. Cornea

7. Which of the following groupings has the highest incidence of eye trauma?
   A. Male, ages 10-15
   B. Female, ages 10-15
   C. Male, ages 25-30
   D. Female, ages 25 -30

8. Which of the following agents account for the majority of corneal burns?
   A. Acidic chemical agents
   B. Alkali chemical agents
   C. Thermal agents
   D. Ultraviolet light

9. What are the two most common bones injured in orbital wall fractures?
   A. Maxilla and sphenoid
   B. Maxilla and frontal
   C. Maxilla and ethmoid
   D. Maxilla and zygoma

10. Where is the most common anatomic site of optic nerve injury from blunt trauma?
    A. Intraocular (optic disc)
    B. Intraorbital
    C. Intracanalicular
    D. Intracranial (optic chiasm)

Answer Key: 1.B; 2.D; 3.A; 4.D; 5.A; 6.C; 7. A; 8.B; 9.C; 10.C

In Future Issues:

Management of the Burned Patient