Diagnosis and Management of Nursery Arrhythmia

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Introduction

Cardiac arrhythmias presenting at birth are rare and, in the majority of cases, are asymptomatic and self-limiting. Approximately 1% of newborns are recognized with rhythm abnormalities. The majority display asymptomatic atrial ectopy or benign sinus bradycardia. The challenge is to discern between these benign rhythms and the less frequent hemodynamically significant rhythm disturbances requiring intervention.

Many hemodynamically significant arrhythmias are first noted during prenatal evaluation and perinatal monitoring. Irregular contractions are often evident on fetal ultrasound. Discontinuity on ultrasound of the frequency of atrial and ventricular contractions along with slow fetal pulse rate is a typical presentation of congenital forms of heart block.

Sustained fetal tachyarrhythmias are associated with risk of fetal myocardial dysfunction, intrauterine growth retardation, and possible development of fetal hydrops requiring initiation of antiarrhythmic maternal drug therapy. Neonatal intensive care management is typically prearranged in cases of a symptomatic fetal arrhythmia. Initial recognition or stabilization of these newborns is typically not performed in the well-baby nursery.

Benign asymptomatic rhythm abnormalities and destabilizing symptomatic rhythm disturbances can however first present without prior suspicion in the nursery and must be promptly recognized. Every well-baby nursery needs to be equipped with personnel and equipment allowing the prompt awareness of rhythm abnormalities. Benign arrhythmias require continued monitoring without other intervention. Potentially hemodynamically destabilizing arrhythmias need to be recognized, documented, and monitored with stabilization and therapy initiated prior to transfer to a higher acuity neonatal care unit.

Every well-baby nursery should have trained clinical personnel to recognize the presentation of both benign and symptomatic arrhythmias. On-site and immediately available equipment for diagnosis and monitoring of newborn heart rate and rhythm is mandatory along with the ability to administer first-line drug therapy and other treatments. In the event of acutely destabilizing arrhythmias, personnel trained in emergency life support must be available along with resuscitative equipment, means of delivery of life support medications, and cardioversion capability.

After initial stabilization, hemodynamically significant arrhythmias will require subsequent monitoring and intervention in an NICU setting.

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Newborns with benign rhythms will require ongoing routine clinical monitoring prior to discharge home. Arrhythmias persisting at discharge need scheduled cardiology evaluation as outpatients with immediate reassessment if any post-discharge symptoms are suspected.

The Equipped Nursery

The prerequisite for prompt recognition and timely management of nursery arrhythmias is diligent and ongoing clinical observation by on-site medical and nursing personnel including routine serial measurement of heart rate and pulse regularity along with blood pressure, oximetry, respiratory, and perfusion status.

Required equipment includes 12-lead EKG recorder with rhythm strip printout for use in diagnosis and real-time management.

First-line antiarrhythmic drugs need to be on-site or promptly available from the hospital pharmacy. Required drugs (Table 14.1) include propranolol IV/PO, digoxin IV/PO, lidocaine IV, adenosine IV, and prostaglandin E1.

In nursery availability of additional neonatal resuscitation medications including atropine, epinephrine, sodium bicarbonate, and calcium gluconate.

On-site or on-call echocardiography to assess for structural and functional heart abnormality.

Neonatal cardioversion/defibrillator with low-wattage capability and infant-sized electrode pads (Table 14.2).

The cardioversion procedure should be well organized with a protocol which is consistent and includes a step-by-step approach (Table 14.3).

Table 14.2 Neonatal cardioversion required equipment

External pediatric defibrillator
(Typically minimum charge level 2 joules, alternatively may use pediatric-capable adult defibrillator with energy-reducing pads)
Neonatal-sized electrode pads maximum 4.5 cm diameter
Cardiac monitor with strip recorder
Airway management equipment
Antiarrhythmic and resuscitation medications

Table 14.3 Cardioversion procedure (step by step)

Neonatal cardioversion/defibrillation		
Pre-sedation		
Pad placement		
	Anterior/ posterior	Front at left of mid sternum
		Back at left mid-chest (alternatively at subxiphoid apex)
Defibrillator charging		
	Power	*ON*
	Mode	*MANUAL*
	Energy	0.5–1.0 joule/kg to max 2 joule/kg
	Sync mode	Cardioversion *ON*
		Defibrillation *OFF*
	Charge	*CHARGE* button *PRESS*
Clear personnel from bedside		
Administer shock		*SHOCK* button *PRESS*

Table 14.1 Antiarrhythmic medications

Drug dosages			
Propranolol	0.1 mg/kg/dose	Po	Every 6–8 h
	1 mg/kg/dose	IV	Every 6–8 h
Digoxin	Digitalization		
	Preterm		30 mcg/kg/ total dose divided in 3 doses every 12 h
	Full-term		40 mcg/kg/ total dose divided in 3 doses every 12 h
	Maintenance		
	Preterm		5–10 mcg/kg/day divided every 12 h
	Full-term		10 mcg/kg/day divided every 12 h
Lidocaine	1 mg/kg		IV bolus
	50 mcg/kg/min		IV continuous drip
Adenosine	50 mcg/kg		IV bolus increasing to effect every 5 min by
			50 mcg increments to 200 mcg/kg maximum dose
Prostaglandin E1	0.05–0.1 mcg/kg/min		IV infusion with maintenance 0.01–0.05 mcg/kg/min

Bradyarrhythmia

Case Presentation

Full-term female neonate arrives in the well-baby nursery after vaginal delivery appearing well but with an irregular pulse and good perfusion and in no respiratory distress. By palpation heart rate averages 70–90 beats/minute (Fig. 14.1)

1. What immediate management is indicated?
    (A) Transfer to NICU for cardiac monitoring
    (B) 12-lead EKG and 2D echocardiogram
    (C) 12-lead EKG/rhythm strip
    (D) Septic workup
2. What rhythm is displayed on the EKG initially obtained?
    (A) PACs with aberration and nonconduction
    (B) Junctional rhythm with aberrant conduction
    (C) Second-degree AV block
    (D) Ectopic ventricular contractions
3. What initial therapy is indicated?
    (A) Transfer to NICU for cardiac monitoring and possible chronotropic drug support
    (B) Ongoing nursery vital sign monitoring with repeat EKG prior to discharge
    (C) Start low dose oral beta-blocker
    (D) Cardiac consultation including 2D echo evaluation

Answers: 1B 2A 3B

An abnormally slow heart rate, whether regular or irregular, when detected by auscultation or EKG monitoring is indicative of a bradyarrhythmia. The majority of slow heart rate rhythms such as sinus bradycardia or sinus arrhythmia are asymptomatic and self-limiting. Alternatively, severe dysfunction or the sinus node, AV node, or the conduction pathways may present with profoundly low heart rates and impaired cardiac output.

Sinus Bradycardia
Sinus bradycardia presents with slow regular rate less than 90 beats/minute with normal P wave access and PR interval. Lower heart rates are often promoted by increased vagal tone induced by drugs, hypothyroidism, CNS abnormality, and hypoxemia or as a reflex to airway maneuvers or suctioning inducing a vagal reflex. Low rate may also be a presenting feature of prolongation of the QT interval. A 12-lead EKG should be obtained whenever bradycardia is suspected to confirm the absence of heart block or QT prolongation. Assessment should be made for the presence of contributing systemic factors provoking increased vagal tone. Isolated sinus bradycardia is asymptomatic, self-resolving, and does not require antiarrhythmic therapy.
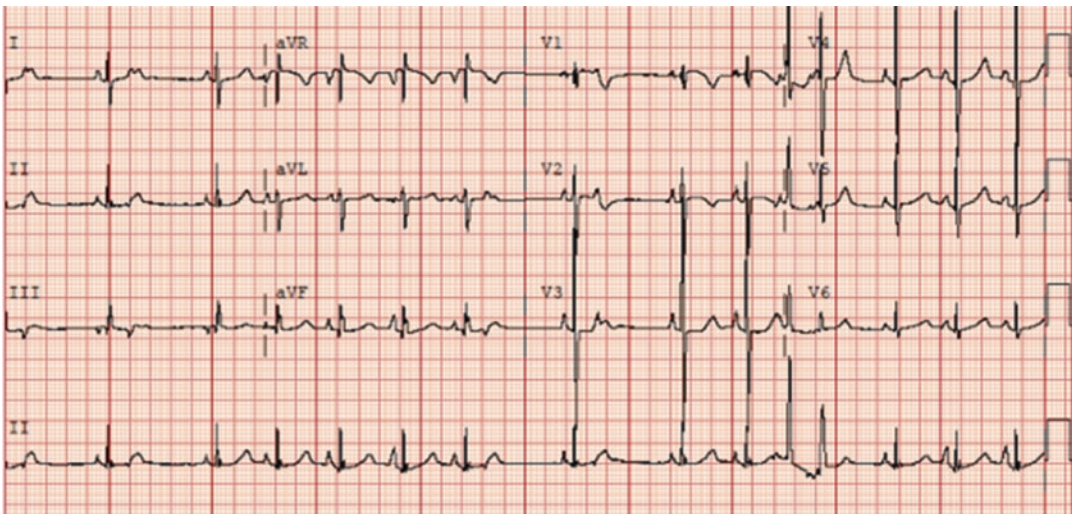

Fig. 14.1   Premature atrial contractions with aberrant conduction

Sinus Arrest

In rare cases the sinus node or, alternatively, the atrial muscle fail to induce a pacemaker impulse resulting in potentially irregular AV node or ventricular escape rhythm. Absence of atrial activity is seen on EKG for multiple cycle lengths potentially inducing severe bradycardia with occasionally irregular escape rhythm response. Atrial pauses greater than 3 sec can result in impaired cardiac output requiring cardiotropic drug therapy. Emergency use of IV atropine or epinephrine is indicated to increase escape rhythm rates prior to transfer to intensive care for assessment of need of pacemaker support.

Atrioventricular Block

An irregular or slow heart rate showing appropriately frequent and normally appearing P waves with intermittent or absent QRS ventricular conduction is the indication of impaired AV node conduction. Slowed but consistent AV node conduction is demonstrated by an increased EKG PR interval greater than 0.16 sec at birth. Intermittent nonconduction will show P waves with occasionally absent succeeding QRS complexes. In approximately 1:20,000 births, complete heart block presents with independent generation of ventricular QRS complexes at a rate not associated with P wave frequency (Fig. 14.2).

In 25–50% of cases, higher level AV node conduction abnormalities are associated with congenital heart deformities and are an indication for 2D echo screening. The most common heart abnormalities seen include congenitally corrected transposition of the great arteries, heterotaxy, seen in AV canal defects especially "polyspenia" or left atrial isomerism.

Profoundly increased vagal tone promoted by abdominal pain, airway manipulation, or CNS abnormality may temporarily impair AV node conduction. Abnormalities in electrolytes such as potassium, magnesium, and calcium need to be eliminated. Pericardial inflammation due to isolated pericarditis or pulmonary disease can induce AV node inflammation resulting in often transient AV node block.

Maternal autoimmune disorders, most prominently systemic lupus erythematosus and Sjogren's syndrome, lead to production of anti-Ro and anti-La maternal antibodies that cross the placenta and result in immune complex damage of the fetal AV node. Most cases of maternal autoimmune-induced fetal AV node injury present with second trimester fetal bradycardia and are an indication for high-risk delivery and immediate neonatal intensive care intervention.

Isolated congenital dysfunction of the AV node or distal conduction pathways can occur in
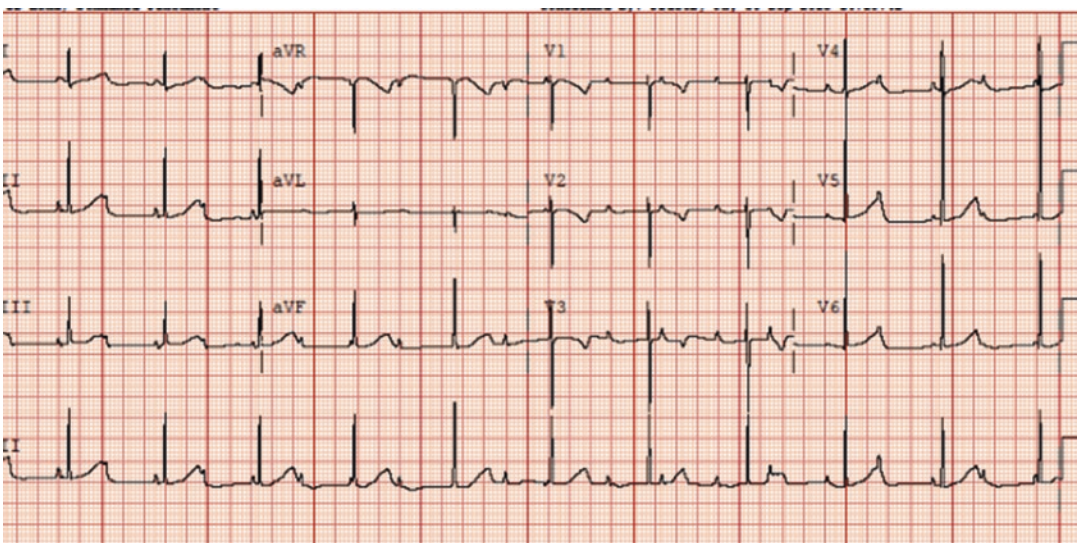

Fig. 14.2 Atrial-ventricular heart block

the absence of other structural heart abnormalities and present with fetal and subsequent neonatal bradycardia.

All cases of higher level AV node dysfunction should be transferred to an intensive care unit. Acute management with chronotropic agents is indicated if impaired cardiac output is suspected. The receiving intensive care unit should have the capability to initiate temporary pacing if an impaired cardiac output state is not stabilized on drug therapy. Permanent pacemaker placement is typically required eventually in neonates with native heart rates less than 55 beats/minute.

Premature Atrial Contraction (PAC)-Induced Bradycardia

A common cause of bradycardia seen almost exclusively in newborns is a reduced ventricular rate resulting from the nonconduction of early and frequent premature atrial contractions. Due to the generally prolonged AV node refractory period seen at birth, rapid atrial impulses occurring during the refractory period are not conducted to the ventricles. Nonconducted P waves will be seen along with abnormal-appearing conducted premature atrial beats. The resultant EKG pattern may mimic a primary AV block but typically differs upon analysis of P wave morphology and coexistence of conducted premature atrial beats. Unlike AV block where P wave axis and appearance are typically normal, the nonconducted P waves will appear similar to those seen in conducted premature atrial beats.

The bradycardia induced by nonconducted premature atrial contractions is rarely symptomatic resolving over the first month of life as atrial automaticity resolves and AV node refractory period shortens. If symptomatic bradycardia occurs, therapy with b-blockers or digoxin may be temporarily used.

Tachyarrhythmias

Case Presentation

A full-term 3.8 kg male with unremarkable prenatal course born by NSVD arrives in the nursery appearing well with initial regular heart rate 120/min and respirations 22/min with saturation of 98% by pulse oximetry. At 8 h of age, he appears mildly tachypneic with rapid pulse maintaining normal perfusion and blood pressure. A stat rhythm strip is obtained (Figs. 14.3 and 14.4).

1. What is the most likely rhythm disturbance?
   (A) Sinus tachycardia with aberrant ventricular conduction
   (B) Supraventricular tachycardia with aberrant ventricular conduction
   (C) Ventricular tachycardia
   (D) Ectopic atrial tachycardia with aberrant conduction
2. What initial intervention is indicated?
   (A) Cardioversion
   (B) Facial ice bag application or other vagal maneuvers
   (C) Propranolol 0.1 mg/kg IV
   (D) Urgent transfer to closest NICU
3. Following initial therapy the rapid heart rate persists without associated cardiovascular symptoms. What is the next treatment step?
   (A) IV adenosine 100 mcg/kg IV.
   (B) Digoxin loading dose followed by IV adenosine.
   (C) Cardioversion.
   (D) Propranolol IV followed by IV adenosine if tachycardia persists after adenosine administration; thus, rhythm strip is obtained.
4. What is the most likely underlying rhythm abnormality?
   (A) Orthodromic reentrant supraventricular tachycardia
   (B) Sinus tachycardia with AV block
   (C) Atrial flutter
   (D) Ventricular tachycardia

Answers: 1B 2B 3A 4C

Except for sinus tachycardia and atrial premature beats, many tachyarrhythmias are potentially symptomatic short-term or progress over time. Sustained ventricular tachyarrhythmias are often a medical emergency. When presenting in the

Fig. 14.3 Supraventricular tachycardia with aberrant ventricular conduction
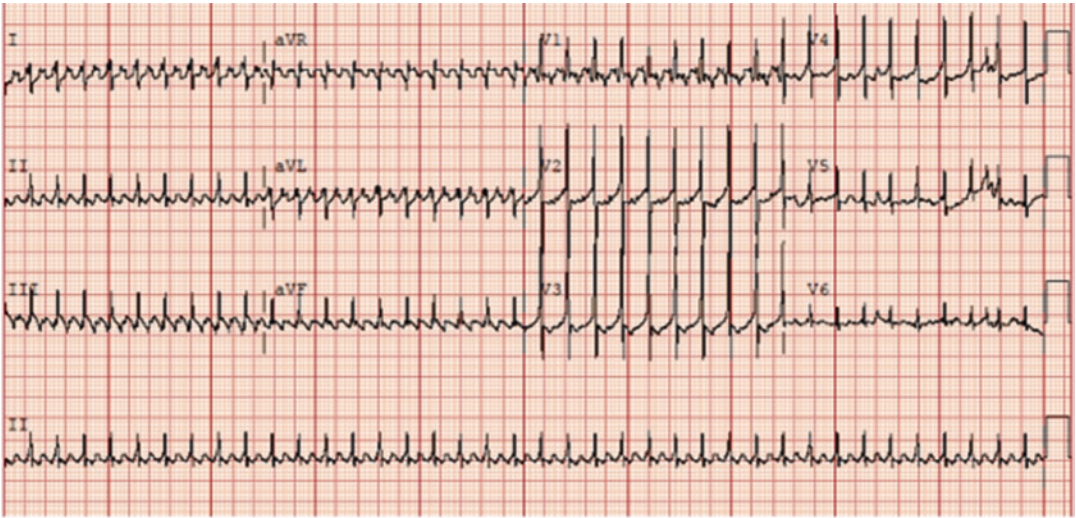

Fig. 14.4 Atrial flutter with AV block

nursery setting, cardiovascular support ranging from physical vagal maneuvers to front-line drug therapy and emergency acute life support intervention should be available.

Sinus Tachycardia

Sinus tachycardia in neonates presents as a regular rhythm with rate ranging from 160 to 230 beats/minute. Sustained heart rates progressively greater than 180 beats/minute increase the likelihood of the existance of an abnormal atrial or AV node-generated ectopic rhythm such as atrial flutter or fibrillation, reentrant supraventricular tachycardia, or an ectopic focus. Unlike most ectopic tachycardias, the EKG will show consistently present and normal P waves. Normal or abnormally oriented P waves may be seen in ectopic atrial tachycardias. Apart from increased sympathetic tone or

myocardial dysfunction, sinus tachycardia is often secondary to non-cardiac causes as fever, hypoxemia, hypovolemia, or pain. If non-cardiac factors are resolved and sinus tachycardia is sustained, a 2D echocardiographic examination is indicated to evaluate for myocardial function.

Atrial Flutter/Fibrillation

Unlike older children where atrial flutter and particularly atrial fibrillation is typically associated with structural heart deformities, isolated atrial flutter often occurs in a fetus or newborn with normal heart structure and function. Evidence of persistent fetal atrial tachycardia prior to delivery is indication for high-risk delivery and neonatal ICU support.

In both atrial flutter and re-entry atrial tachycardia, the nursery newborn will typically present

with heart rates above 200 beats/minute with atrial flutter rates more likely to approach 300 beats/minute. Higher maximal heart rates greater than 400 beats/minute are constrained by the longer AV node refractory period in neonates. The EKG in atrial flutter, like reentry tachycardia, will typically show narrow QRS complexes with no discernable P waves in the short interval between the rapid ventricular complexes. Sawtooth "flutter" waves are only seen when the ventricular rate slows with vagal maneuvers or drug therapy.

Even if a definitive diagnosis of the specific tachydysrhythmia is not yet established, vagal maneuvers are an appropriate initial response to attempt conversion to sinus rhythm. Since the mechanism of action of a vagal maneuver is to increase parasympathetic tone and incite temporary AV block, these maneuvers are more effective in converting reentrant tachycardias rather than atrial flutter arising from the atrial tissues. The most practical vagal maneuver in neonates is application of an iced cloth to the upper face, gagging maneuvers, or right carotid artery massage. If these efforts fail to convert the tachycardia, then intravenous adenosine should be administered for both diagnostic and possible therapeutic effects. Although adenosine is significantly less effective in converting non-reentrant tachycardias such as atrial flutter, the resultant transient AV block when monitored by rhythm strip will eliminate ventricular depolarization exposing the atrial flutter waves. In reentrant tachycardias no significant atrial depolarizing signal or P waves should be evident.

An initial adenosine dose of 100 mcg/kg should be rapidly infused by bolus intravenously over 1–2 sec increasing every 2 min by 50 mcg/kg until AV block is achieved. Unlike in reentrant tachycardias, further increases are unlikely to result in conversion to sinus rhythm.

If available, ongoing antiarrhythmic therapy and, if unsuccessful, cardioversion or atrial pacing should be managed in an intensive care setting. If ongoing atrial flutter is resulting in impaired perfusion, acidosis, or hemodynamic compromise, then emergency asynchronous cardioversion should be performed in the nursery prior to transfer.

Reentrant Supraventricular Tachycardia

Reentrant SVT is the most common symptomatic arrhythmia seen in neonates with incidence of up to 1:200 live births. Initial presentation after birth is identical to atrial flutter with somewhat lower ventricular rates typically less than 300 beats/minute. First-line management is no different than atrial flutter with use of vagal maneuvers and intravenous adenosine. 2D echocardiogram evaluation is indicated given the possible association with structural heart deformity. Specific defects are, most commonly, Ebstein's anomaly with preexcitation and right-sided accessory pathways in 10% of cases, L=transposition of the great arteries, and atrial septal defects.

The involvement of the AV node in the reentrant circuit increases the dose-dependent effectiveness of intravenous adenosine. As in atrial flutter, an initial IV bolus of 100 mcg/kg of adenosine is administered increasing by 50 mcg/kg sequentially up to 300 mcg/kg until conversion is achieved. At adequate adenosine dosage, up to 90% of reentrant tachycardias involving the SA or AV nodes will convert to sinus rhythm. Following termination of tachycardia, beta-blocker therapy is indicated to prevent recurrence during early infancy. If maximal adenosine dosage does not produce sinus rhythm, then pretreatment with IV beta-blocker can be administered with propranolol 0.1 mg/kg IV at 6-h intervals before repeat adenosine bolus infusion. Continued persistence of tachycardia after blocker treatment and adenosine infusion requires alternative antiarrhythmic drug treatment in an intensive care setting also equipped with atrial pacing and cardioversion capability.

Ventricular Tachyarrhythmia

Tachyarrhythmias of ventricular origin are rare in neonates. Widened QRS complex beats either in isolation or sustained typically represent a supraventricular focus with aberrant conduction though the ventricles. Isolated premature ventricular contractions are rare, asymptomatic, and self-limiting within the first month. In rare cases frequent isolated PVCs increase the risk of sustained tachycardia.

A sustained ventricular tachyarrhythmia in the neonate is a medical emergency capable of inducing progressive impairment in cardiac output and potential cardiac arrest. All tachycardia rhythms with a widened QRS duration greater than 120 ms and typically an abnormal axis should be considered of ventricular origin and trigger emergency management. In rare cases ventricular tachycardia displays a narrow but abnormal QRS configuration. Widened QRS complexes of rapidly varying morphology are seen in "torsade de pointes," with an underlying QT interval prolongation and imminent deterioration to ventricular fibrillation.

Fortunately the majority of wide complex tachycardias are of atrial origin with associated aberrant ventricular conduction due to relative refractory AV node and bundle branch conduction. Although normal perfusion suggests supraventricular origin, it does not eliminate the possibility of a ventricular focus. Adenosine administration is useful in differentiating SVT with aberrant conduction from ventricular tachycardia. Since the AV node does not participate in the ventricular tachycardia pathway, no rate response is seen upon adenosine infusion. A blockade of ventricular conduction with adenosine indicates a supraventricular origin.

Sustained ventricular tachycardia with hemodynamic compromise requires immediate intervention with synchronized cardioversion at 0.5 joules/kg increasing if ineffective to 2 joules/kg. If sinus rhythm is not achieved, then asynchronous cardioversion should be performed. 2D echocardiography is indicated to evaluate cardiac structure and assess for persistent myocardial dysfunction. Lidocaine or procainamide can be used under cardiologist supervision to prevent recurrence of ventricular tachycardia.

Case Presentation

A healthy-appearing full-term female newborn is noted to have a slow pulse on examination with HR 70–80/min; physical examination is normal with good perfusion and no respiratory distress, full oxygen saturation on RA. The parents state that an older sister recently died suddenly at 8 years of age while swimming (Fig. 14.5).

1. Initial management should include:
   (A) Transfer to NICU for electrophysiology consultation
   (B) CBC, electrolytes including NA K Ca Mg
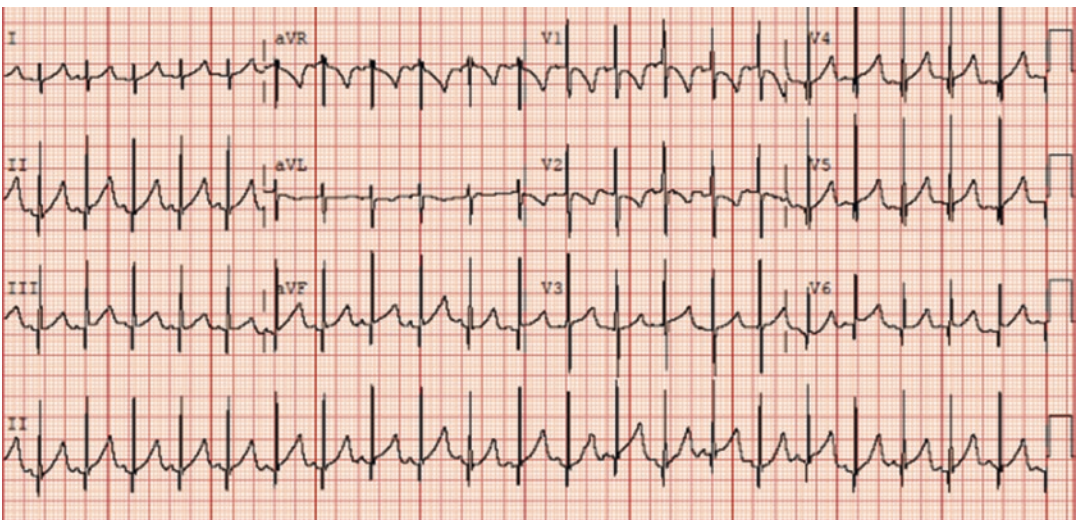   (C) Head CT scan
   (D) Holter monitoring

Fig. 14.5 QT interval prolongation

2. Which of the following drugs are contraindicated for future use?
   (A) Digoxin
   (B) Erythromycin
   (C) Gentamicin
   (D) ASA

3. What management is now indicated?
   (A) Start beta-blocker therapy and refer for cardiology evaluation.
   (B) Cardiac monitoring in NICU until discharge home.
   (C) Transfer for immediate cardiology evaluation prior to discharge home.
   (D) Start oral amiodarone and refer for cardiology evaluation.

Answers: 1B 2B 3A

QT Interval Prolongation

Abnormal ionic transport across the cardiomyocyte membrane may manifest as an electrocardiographic prolongation of the QT interval. A high risk of life-threatening tachyarrhythmia exists in neonates when AV block and extreme QT prolongation is evident.

Every EKG obtained in the nursery whether performed due to the existence of arrhythmia, congenital heart defect, or family history of sudden death should be evaluated for QT interval prolongation. Given the frequent inheritance of QT interval abnormality, a family history of every neonate with arrhythmia should include assessment of familial sudden death and QT interval prolongation.

Most neonates with abnormal QT intervals will be asymptomatic at birth. An underlying QT interval prolongation is more likely in the presence of sustained bradycardia or ventricular ectopy.

The QT interval is measured as the time duration from the onset of the QRS complex to the intersection of the T wave downslope with the EKG baseline. A trailing U wave is excluded from the measurement unless its magnitude is equally prominent as the T wave peak. A corrected QT interval is standardized for heart rate with division by the square root of the R-R interval.

Symptomatic arrhythmias are significantly more likely if the corrected QT interval exceeds 0.60 milliseconds or in the presence of 2:1 AV block, T wave alternans, or sustained bradycardia. Higher risk is seen with coexistent sensory neural hearing loss. In these high-risk cases, propranolol should be started at dosage of 1 mg/kg orally every 8 h. Additional antiarrhythmic medications may be needed if sinus rhythm is not achieved.

QT intervals greater than 0.50 milliseconds or coexistent arrhythmia require beta-blocker therapy. More precise individualized drug therapy may be later substituted under pediatric cardiology supervision with genetic subtyping of the underlying channelopathy. Temporary self-resolving QT prolongation can be seen in the first week of life with initial corrected QT duration greater than 0.44 milliseconds. Electrolyte abnormalities such as hypocalcemia and hypokalemia, macrolide antibiotics, and CNS abnormalities can produce "acquired" QT prolongation. An initial corrected QT interval between 0.44 and 0.50 milliseconds should be re-evaluated by follow-up EKGs in the first week of life before drug therapy is considered.

Case Presentation

A full-term clinically well appearing male infant is noted to have a heart murmur on arrival to the nursery. Prenatally an intermittent tachycardia was suspected with no specific documented arrhythmia. The initial vital signs show a regular heart rate of 120/min, respiratory rate 32/min with right arm blood pressure of 80/60, and pulse oximetry 94% on RA (Fig. 14.6).

1. What is the first initial management indicated?
   (A) Transfer to ICU for murmur evaluation
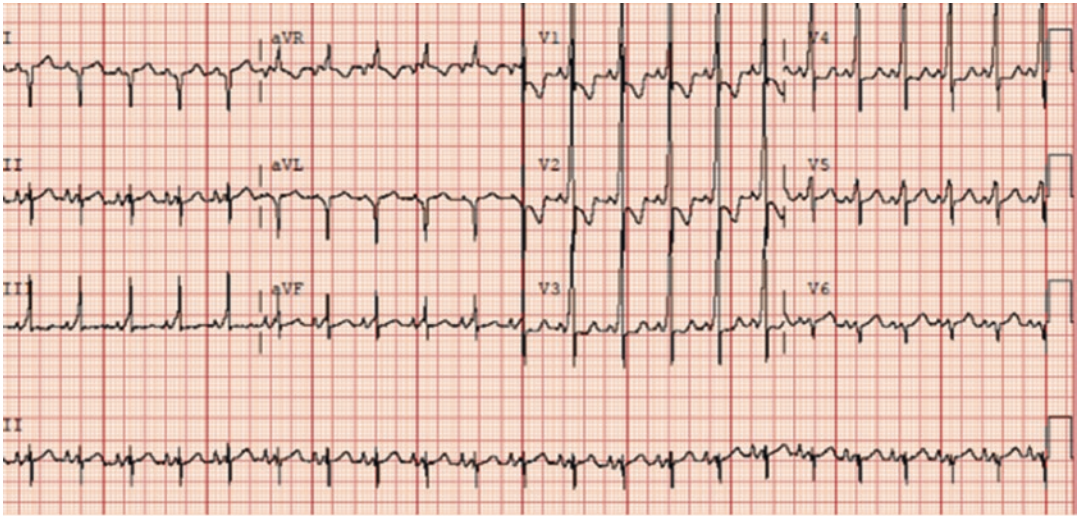   (B) Physical examination including four-extremity pulses and blood pressure and pre- and postductal pulse oximetry

Fig. 14.6 Ventricular preexcitation

    (C) EKG and rhythm strip
    (D) 2D echocardiogram

The EKG displayed the rhythm as seen, and 2D echocardiogram shows moderate right heart enlargement with a redundant tricuspid valve and moderate tricuspid valve insufficiency.

2. What structural heart abnormality is suspected?
    (A) Ebstein's anomaly
    (B) Congenitally corrected L-transposition of the great arteries
    (C) Pulmonary hypertension
    (D) ASD

3. What cardiac arrhythmia should be anticipated?
    (A) Ventricular tachycardia
    (B) Supraventricular tachycardia
    (C) Premature atrial contractions
    (D) Complete heart block

Answers: 1B 2A 3B

Neonatal Arrhythmia in Congenital Heart Disease

Infants with structural heart defects are more likely to exhibit coexistent cardiac arrhythmias triggered by an abnormal conduction system, altered hemodynamics, or hypoxic stress. Most significant newborn congenital heart disease will present with abnormal physical findings such as heart murmur, abnormal respiration, impaired perfusion, or desaturation. Some asymptomatic neonates with underlying heart defects may initially present with a wide spectrum of arrhythmias. A 2D echo should be obtained in the nursery on recognition of sustained cardiac arrhythmia. Mandatory performance of echocardiography is not required for asymptomatic transient sinus bradycardia or isolated premature atrial beats which are rarely associated with anatomic abnormality.

Most urgent is the recognition of heart defects that will imminently manifest with critical or life-threatening symptoms. Complex heterotaxy is often associated with fragile malpositioned ventricular conduction pathways resulting in impaired AV conduction or heart block. Associated pulmonary outflow obstruction or

left-sided obstructive defects in these cases may result in ductal dependency and need to initiate PGE infusion. L-transposition of the great vessels in particular is associated with conduction pathway defects distal to the HIS bundle.

Of particular significance is the frequent association of Ebstein's anomaly with Wolff-Parkinson-White syndrome due to the presence of right-sided accessory AV pathways. Infrequently SVT and atrial ectopy will also be seen in isolated atrial septal defects.

In rare cases focal cardiac tumors may provide the nidus for ventricular automaticity causing frequent or incessant ventricular tachycardia.

Neonatal Arrhythmia and Systemic Disease

Hypoxemia resulting from systemic illness such as sepsis, respiratory failure, and central neurologic abnormalities may induce automaticity of cardiomyocytes with resultant ectopy. In these situations myocardial dysfunction can contribute to altered hemodynamics causing increased cardiac muscle strain and ectopy. Severe congenital hypertrophic cardiomyopathy can lead to inadequate perfusion and oxygen delivery to heart tissues also inducing myocyte irritability. Autoimmune illness such as system lupus erythematosus (SLE) and Sjogren's syndrome can be manifested through production of maternal antibodies, destruction of AV node tissue, and resultant heart block in the fetus and newborn.

Suggested References

2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 11: Pediatric Basic Life Support and Cardiopulmonary Resuscitation Quality;

Clinical Pearls
1. Neonatal QT interval prolongation may present as profound sinus bradycardia or AV block.
2. Impaired AV node conduction and resultant heart block is a presenting feature of complex congenital heart disease.
3. Bradycardia in the presence of PACs is typically indicative of nonconduction of ectopic atrial beats rather than primary AV node dysfunction.
4. The underlying ectopic basis of sustained tachycardia rhythm is often unmasked upon adenosine-induced AV node blockade.
5. If hemodynamic stability is maintained, sustained wide complex tachycardia is typically of supraventricular rather than ventricular origin.

Part 12: Pediatric Advanced Support; Part 13: Neonatal Resuscitation.

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