PEDIATRIC ADVANCED LIFE SUPPORT

REFERENCE GUIDE

JOE JONES, NREMT-PARAMEDIC

**BLS Considerations During PALS**

High quality CPR is a must in the attempts in successful resuscitation of a Pediatric patient suffering from cardiopulmonary arrest. One should always attempt to identify the cause and treat it accordingly.

**ESTABLISH UNRESPONSIVENESS**

**CHILD PULSELESS AND APNEIC**

**BEGIN CPR WITH QUALITY CHEST COMPRESSIONS (**rate at least 100/minute)

( one rescuer begins chest compressions while second prepares to ventilate)

 ( other rescuers now should be retrieving the monitor/AED)

 ( compress at least 1and ½ inches on an infant and 2 inches on the pediatric)

**ONCE THE DEFIFTILLATOR ARRIVES THEN ATTACH TO PATIENT, STOP COMPRESSIONS AND IDENIFY THE RHYTHM OR FOLLOW THE PROMPTS ON THE AED…..**

**FROM THIS POINT TREAT ACCORDING TO THE PROPER ALOGORHYTHM RECOMMENDED BY THE AMERICAN HEART ASSOCIATION.**

**ALWAYS MAKE SURE PATIENT IS ON A FIRM SURFACE WHILE PERFORMING CPR AND WHEN ENOUGH PERSONEL ARE PRESENT SWITCH COMPRESSORS EVERY 2 MINUTES TO PREVENT TIRING WHICH WOULD LEAD TO POOR COMPRESSIONS.**

**COMMON CAUSES OF ARREST IN THE PED PT.**

In contrast to adults, cardiac arrest in infants and childrendoes not usually result from a primary cardiac cause. More oftenit is the terminal result of progressive respiratory failureor shock, also called an asphyxial arrest. Asphyxia begins witha variable period of systemic hypoxemia, hypercapnea, and acidosis,progresses to bradycardia and hypotension, and culminates withcardiac arrest.[1](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B1)

Another mechanism of cardiac arrest, ventricular fibrillation(VF) or pulseless ventricular tachycardia (VT), is the initialcardiac rhythm in approximately 5% to 15% of pediatric in-hospitaland out-of-hospital cardiac arrests it is reportedin up to 27% of pediatric in-hospital arrests at some pointduring the resuscitation. The incidence of VF/pulseless VTcardiac arrest rises with age. Increasing evidence suggeststhat sudden unexpected death in young people can be associatedwith genetic abnormalities in myocyte ion channels resultingin abnormalities in ion flow

**Hypoxemia is a common cause of cardiopulmonary arrest in the pediatric patient. Treat respiratory problems aggressively in order to prevent respiratory distress or failure will lead to respiratory arrest rapidly.**

**If a child in intubated monitor them closely for any problems with ventilator or the endotracheal tube, these can cause detrimental problems for the patient.**

**Perform a good physical exam on any child with respiratory complaints. Always obtain a good history and physical exam.**

**Pneumonia, Bronchitis and Asthma is common in the child. Treatment for these would be to:**

**Maintain A – B – C’s**

**Monitor oxygen saturation**

**Administer Oxygen**

**Wheezing Present then:**

**Beta Agonist Medications**

**Consider Steroids**

**Chest X-Ray**

**Consider asthma attacks which can not be broken with Beta Agonist Medications alone a life threatening problem.**

**Keep in mind Subcutaneous Epinephrine .01 mg/kg**

**Monitor heart rhythm, hypoxemia will lead to bradycardia which will lead to cardiopulmonary arrest.**

**Correct problems such as these early to prevent child from deteriorating.**

**SHOCK**

**Shock**
Shock results from inadequate blood flow and oxygen deliveryto meet tissue metabolic demands. The **most common type of shockin children is hypovolemic**, including shock due to hemorrhage.**Distributive, cardiogenic, and obstructive shock occur lessfrequently**. Shock progresses over a continuum of severity, froma compensated to a decompensated state. Compensatory mechanismsinclude tachycardia and increased systemic vascular resistance(vasoconstriction) in an effort to maintain cardiac output andperfusion pressure respectively. ***Decompensation occurs whencompensatory mechanisms fail and results in hypotensive shock***.

**Typical signs of compensated shock include**

* Tachycardia
* Cooland pale distal extremities
* Prolonged (>2 seconds) capillaryrefill (despite warm ambienttemperature)
* Weak peripheralpulses compared with central pulses
* Normal systolic bloodpressure

**As compensatory mechanisms fail, signs of inadequate end-organperfusion develop. In addition to the above, these signs include**

* Depressedmental status
* Decreased urine output
* Metabolic acidosis
* Tachypnea
* Weak central pulses
* Deterioration in color (eg,mottling, see below)

Decompensated shock is characterized by signs and symptoms consistentwith inadequate delivery of oxygen to tissues (pallor, peripheralcyanosis, tachypnea, mottling of the skin, decreased urine output,metabolic acidosis, depressed mental status), weak or absentperipheral pulses, weak central pulses, and hypotension.

Learn to integrate the signs of shock because no single signconfirms the diagnosis. For example:

* Capillary refill time aloneis not a good indicator of circulatoryvolume, but a capillaryrefill time >2 seconds is a usefulindicator of moderatedehydration when combined with decreasedurine output, absenttears, dry mucous membranes, and a generallyill appearance.Capillary refill time is influenced by ambienttemperature,[25](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B25)site, and age and its interpretation can be influencedby lighting.[26](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B26)
* Tachycardia is a common sign of shock, but it can alsoresultfrom other causes, such as pain, anxiety, and fever.
* Pulses are weak in hypovolemic and cardiogenic shock, butmaybe bounding in anaphylactic, neurogenic, and septic shock.
* Blood pressure may be normal in a child with compensated shockbut may decline rapidly when the child decompensates. Like theother signs, hypotension must be interpreted within the contextof the entire clinical picture.

There are several sources of data that use large populationsto identify the 5th percentile for systolic blood pressure atvarious ages.[27](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B27),[28](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B28) For purposes of these guidelines, hypotensionis defined as a ***systolic*** blood pressure:

* <60 mm Hg in termneonates (0 to 28 days)
* <70 mm Hg in infants (1 month to12 months)
* <70 mm Hg + (2 x age in years) in children 1to 10 years
* <90 mm Hg in children 10 years of age

Treatment for Shock would include:

Maintain A –B – C’s

Oxygen high flow

Cardiac Monitor

Monitor saturation levels

Hypovolemia non-hemorrhagic: IV isotonic solution at 20 ml/kg may repeat up to 4 times if needed. Make sure to reassess after each bolus of fluid.

Hypovolemia due hemorrhage: IV isotonic solution at 20 ml/kg initially then consider 10 ml/kg of packed red blood cells or 20 ml/kg of whole blood. In certain cases one may consider hypertonic solutions, i.e. Hextend.

Anaphylaxis is a highly deadly, be aggressive with airway treatment, Epinephrine 0.01 mg/kg of a 1 – 1000 solution subcutaneous or I.M. or if hypotension is present then 0.01 mg/kg of a 1 – 10,000 solution IVP, Benadryl 1 – 2 mg/kg, consider steroids. Hypotension present then 20 ml/kg of a isotonic solution bolus then reassess, consider use of vasoactive drugs such a dopamine in severe cases at 2 – 20 mcg/kg/min.

Neurogenic Shock protect the C-Spine and administer IV isotonic solutions at 20 ml/kg consider IV steroids.

Cardiogenic shock Access lung sounds prior to fluid. Remember these children can fill up fast when the pump is not pumping well. Consider vasoacive medications such as Dopamine, Dobutrex, or even possibly Levophed. Remember to seek expert consultation.

Septic Shock: IV isotonic solution at 20 ml/kg may repeat then consider the use of vasopressors such as dopamine. Work to finding out what’s causing the infection and treat it aggressively.

After dopamine has been used and not effective then a patient who is experiencing cold shock use epinephrine and the patient with warm shock use levophed.

**CARDIAC ARRHYTHMIAS**

 The primary cause of bradycardia in the pediatric patient comes from hypoxemia. One must be aggressive with the airway and breathing and maintain good oxygen saturation. In certain cases the pediatric patient will experience a vagal maneuver which will cause bradycardia to occur. Treatment should include the following.

A – B – C’s be sure to provide high concentrations of oxygen. In cases where breathing is inadequate be aggressive with ventilators. If unsuccessful then:

Monitor/EKG observe closely

IV isotonic solution at a to keep open rate do not overload the patient remember the pump is not pumping.

Epinephrine IVP 1 – 10,000 solution 0.01 mg/kg

Continue to monitor the patient and assure that airway and ventilation is being carried out correctly.

Repeat Epinephrine IVP 1 – 10,000 solution 0.01 mg/kg and repeat q-3 – 5 minutes

Seek Expert consultation if not already done so, also consider chronotropic meds such as dopamine at 2 – 10 mcg/kg/min.



With tachycardia one must assess for all causes before beginning treatment. Assure this is a cardiac problem rather than something which can be corrected with a more simple approach. Remember the response to pain, fever, Hypovolemia are causes of tachycardia, rule out these causes first. A child is not considered to be in Supraventricular Tachycardia until the rate is greater than 180 and the infant must be greater than 220. Always assess the patient and determine if he/she is unstable or stable.

STABLE SVT (NARROW COMPLEX)

A – B- C’s provide high concentration of oxygen

EKG monitor closely

Seek Expert consultation

Vagal Manuvers: blow into a closed straw, ice water to the face etc. Do not attempt to massage eyelids due to this may cause retina problems. If unsuccessful then move to more aggressive rx.

IV at kvo rate remember do not overload the patient.

Adenosine 0.1 mg/kg do not exceed 6 mg. repeat in 2 – 3 minutes

Adenosine 0.2 mg/kg do not exceed 12 mg. repeat in 2 – 3 minutes

Adenosine 0.2 mg/kg do not exceed 12 mg. max dose given

If Adenosine is unsuccessful then go to synchronized cardioversion if time permits sedation is recommended.

Start at .5 – 1 joule per kg may repeat at 2 joules per kg

Consideramiodarone 5 mg/kg IO/IV or procainamide 15mg/kg IO/IV for a patient with SVT unresponsive to vagalmaneuvers andadenosine and/or electric cardioversion; for hemodynamicallystable patients, expert consultation is strongly recommendedprior to administration. Both amiodaroneand procainamide must be infused slowly (amiodarone over 20to 60 minutes and procainamide over 30 to 60 minutes), dependingon the urgency, while the ECG and blood pressure are monitored.If there is no effect and there are no signs of toxicity, giveadditional doses avoid the simultaneous use of amiodaroneand procainamide without expert consultation.

**WIDE COMPLEX**

**SEEK EXPERT CONSULTATION IMMEDIATELY**

* Adenosine 0.1 mg/kg do not exceed 6 mg.
* Adenosine 0.2 mg/kg do not exceed 12 mg
* Cordarone 5 mg/kg, may repeat x 1 dose
* Cordarone not available Lidocaine .5 – 1 mg/kg
* Consider Procainamide if Lidocaine not successful however if Cordarone has been administered then do not give Pronestyl
* Consider synchronized Cardioversion

 **UNSTABLE TACHYCARDIA**

* Evaluate the rhythm, wide complex vs. narrow complex
* Identify and treat the underlying cause of the tachycardia, (Sinus Tachycardia, hypovolemia, fever, pain, shortness of breath, etc.
* Supraventricular/Ventricular Tachcardia
* Serious Signs and Symptoms present: Decrease LOC, Hypotension etc.
* Prepare for immediate cardioversion
* A – B – C’s
* Apply Oxygen
* Time Permitting 12 Lead EKG
* IV/IO NaCl at kvo rate
* Consider Sedation
* Synchronize Cardiovert at .5 – 1 joule/kg
* Serious Signs Still Present
* Synchronize Cardiovert at 2 joules/kg.
* 

ASYSTOLE/PEA

* As soon as the child has been identified as being in arrest then begin compressions immediately.
* Call for monitor/AED and as soon as it arrives attach to the patient stop CPR and check rhythm.
* CONFIRM RHYTHM – ASYSTOLE/PEA PRESENT THEN:
* Continue CPR while 2nd rescuer establishes IV
* Administer Epinephrine 0.01 mg/kg and repeat every 3 – 5 minutes. High dose Epinephrine has not showed any benefit other than a Beta Blocker overdose…
* Intubate the patient and confirm placement of tube. BBS/Rise and fall of the chest/End tital CO2 detector/Waveform Capnography.
* Once intubation has been achieved then continue CPR without interruptions at a rate of at least 100/minute and ventilations at **8 – 10** times per minute
* Continue the administration of Epinephrine
* Attempt to figure out the reason for arrest and treat accordingly.
* Hypovolemia administer fluids at 20 ml/kg, Drug overdose, attempt to reverse the drug responsible for the arrest, Tension Pneumothorax decompress at the second intercostal space above the third rib, Metabolic Acidosis treat with Bicarbonate at 1 – MEQ/kg. Cardiac Tamponade perform pericardiocentesis

**VENTRICULAR FIBRILLATION**

**PULSELESS VENTRICULAR TACHYCARDIA**

* Call for help activate the emergency response team.
* Assess unresponsiveness
* Child Pulseless and Apneic
* Begin Chest Compressions at least 100 times per-minute
* Call for AED or Defibrillator
* Continue chest compressions until Defibrillator is connected
* Stop compressions (rhythm above)
* Defibrillate at 2 joules/kg
* Continue CPR
* Initiate IV/IO
* Defibrillate at 4 joules/kg (not to exceed 10 mg/kg or adult dose)
* Administer Epinephrine 0.01 mg/kg
* Continue CPR for 2 minutes then
* Defibrillate
* Continue CPR
* Administer Cordarone 5 mg/kg (repeat x1 dose)
* Cordarone not available then Administer Lidocaine 0.5 – 1 mg/kg (max 3 mg/kg)
* Advanced Airway in place
* Once airway is in place one rescuer should give continuous chest compressions while the other rescuer delivers ventilations at a rate of 1 breath every 6 to 8 seconds or about 8 to 10 per minute.

**PALS Pulseless Arrest Algorithm**



Copyright ©2010 American Heart Association

**Kleinman, M. E. et al. Circulation 2010;122:S876-S908**

|  |  |
| --- | --- |
| * | **Medications** |

**Adenosine**
Adenosine causes a temporary atrioventricular (AV) nodal conductionblock and interrupts reentry circuits that involve the AV node.The drug has a wide safety margin because of its short half-life.Adenosine should be given only IV or IO, followed by a rapidsaline flush to promote drug delivery to the central circulation.If adenosine is given IV, it should be administered as closeto the heart as possible. (See also "Arrhythmia.")

**Amiodarone**
Amiodarone slows AV conduction, prolongs the AV refractory periodand QT interval, and slows ventricular conduction (widens theQRS). Expert consultation is strongly recommended prior to administrationof amiodarone to a pediatric patient with a perfusing rhythm.(See also "Arrhythmia.")

***Precautions***
Monitor blood pressure and electrocardiograph (ECG) during intravenousadministration of amiodarone. If the patient has a perfusingrhythm, administer the drug as slowly (over 20 to 60 minutes)as the patient's clinical condition allows; if the patient isin VF/pulseless VT, give the drug as a rapid bolus. Amiodaronecauses hypotension through its vasodilatory property, and theseverity is related to the infusion rate; hypotension is lesscommon with the aqueous form of amiodarone.[207](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B207) Decrease theinfusion rate if there is prolongation of the QT interval orheart block; stop the infusion if the QRS widens to >50%of baseline or hypotension develops. Other potential complicationsof amiodarone include bradycardia and torsades de pointes ventriculartachycardia. Amiodarone should not be administered togetherwith another drug that causes QT prolongation, such as procainamide,without expert consultation.

**Atropine**
Atropine sulfate is a parasympatholytic drug that acceleratessinus or atrial pacemakers and increases the speed of AV conduction.

***Precautions***
Small doses of atropine (<0.1 mg) may produce paradoxicalbradycardia because of its central effect.[208](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B208) Larger than recommendeddoses may be required in special circumstances such as organophosphatepoisoning[209](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B209) or exposure to nerve gas agents.

**Calcium**
Calcium administration is not recommended for pediatric cardiopulmonaryarrest in the absence of documented hypocalcemia, calcium channelblocker overdose, hypermagnesemia, or hyperkalemia (Class III,LOE B). Routine calcium administration in cardiac arrest providesno benefit[210](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B210)[–](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B211)[221](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B221) and may be harmful.[210](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B210)[–](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B211)[212](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B212)

If calcium administration is indicated during cardiac arrest,either calcium chloride or calcium gluconate may be considered.Hepatic dysfunction does not appear to alter the ability ofcalcium gluconate to raise serum calcium levels.[222](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B222) In criticallyill children, calcium chloride may be preferred because it resultsin a greater increase in ionized calcium during the treatmentof hypocalcemia.[222A](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B222A) In the nonarrest setting, if the only venousaccess is peripheral, calcium gluconate is recommended becauseit has a lower osmolality than calcium chloride and is thereforeless irritating to the vein.

**Epinephrine**
The -adrenergic-mediated vasoconstriction of epinephrine increasesaortic diastolic pressure and thus coronary perfusion pressure,a critical determinant of successful resuscitation from cardiacarrest.[223](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B223),[224](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B224) At low doses, the β-adrenergic effects maypredominate, leading to decreased systemic vascular resistance;in the doses used during cardiac arrest, the vasoconstrictive-effects predominate.

***Precautions***

* Do not administer catecholamines and sodium bicarbonate simultaneouslythrough an IV catheter or tubing because alkaline solutionssuch as the bicarbonate inactivate the catecholamines.
* Inpatients with a perfusing rhythm, epinephrine causes tachycardia;it may also cause ventricular ectopy, tachyarrhythmias, vasoconstriction,and hypertension.

**Glucose**
Because infants have a relatively high glucose requirement andlow glycogen stores, they may develop hypoglycemia when energyrequirements rise.[225](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B225) Check blood glucose concentration duringthe resuscitation and treat hypoglycemia promptly (Class I,LOE C).[226](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B226)

**Lidocaine**
Lidocaine decreases automaticity and suppresses ventriculararrhythmias,[227](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B227) but is not as effective as amiodarone for improvingROSC or survival to hospital admission among adult patientswith VF refractory to shocks and epinephrine.[228](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B228) Neither lidocainenor amiodarone has been shown to improve survival to hospitaldischarge.

***Precautions***
Lidocaine toxicity includes myocardial and circulatory depression,drowsiness, disorientation, muscle twitching, and seizures,especially in patients with poor cardiac output and hepaticor renal failure.[229](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B229),[230](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B230)

**Magnesium**
Magnesium is indicated for the treatment of documented hypomagnesemiaor for torsades de pointes (polymorphic VT associated with longQT interval). There is insufficient evidence to recommend foror against the routine administration of magnesium during cardiacarrest.[231](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B231)[–](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B232)[233](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B233)

***Precautions***
Magnesium produces vasodilation and may cause hypotension ifadministered rapidly.

**Procainamide**
Procainamide prolongs the refractory period of the atria andventricles and depresses conduction velocity.

***Precautions***
There is limited clinical data on using procainamide in infantsand children.[234](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B234)[–](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B235)[236](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B236) Infuse procainamide very slowly (over30 to 60 minutes) while monitoring the ECG and blood pressure.Decrease the infusion rate if there is prolongation of the QTinterval, or heart block; stop the infusion if the QRS widensto >50% of baseline or hypotension develops. Do not administertogether with another drug causing QT prolongation, such asamiodarone, without expert consultation. Prior to using procainamidefor a hemodynamically stable patient, expert consultation isstrongly recommended.

**Sodium Bicarbonate**
Routine administration of sodium bicarbonate is not recommendedin cardiac arrest (Class III, LOE B).[212](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B212),[237](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B237),[238](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B238) Sodium bicarbonatemay be administered for treatment of some toxidromes (see "ToxicologicalEmergencies," below) or special resuscitation situations suchas hyperkalemic cardiac arrest.

***Precautions***
During cardiac arrest or severe shock, arterial blood gas analysismay not accurately reflect tissue and venous acidosis.[239](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B239),[240](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B240)Excessive sodium bicarbonate may impair tissue oxygen delivery;[241](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B241) cause hypokalemia, hypocalcemia, hypernatremia, and hyperosmolality;[242](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B242),[243](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B243) decrease the VF threshold;[244](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B244) and impair cardiac function.

**Vasopressin**
There is insufficient evidence to make a recommendation foror against the routine use of vasopressin during cardiac arrest.Pediatric[245](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B245)[–](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B246)[247](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B247) and adult[248](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B248),[249](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B249) case series/reportssuggested that vasopressin[245](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B245) or its long-acting analog, terlipressin,[246](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B246),[247](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B247) may be effective in refractory cardiac arrest when standardtherapy fails. A large pediatric NRCPR case series, however,suggested that vasopressin is associated with lower ROSC, anda trend toward lower 24-hour and discharge survival.[250](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B250) A preponderanceof controlled trials in adults do not demonstrate a benefit.[251](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B251)[–](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B252)[256](http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S876#B256)