Pattern recognition in paediatric ECGs: the hidden secrets to clinical diagnosis

The aim of this article is to encourage a fresh look at the routine ECG.

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In general, medical practitioners (and paediatricians in particular) are trained to recognise common patterns of abnormalities in order to derive clues to disease diagnosis. This is typical of most clinical settings, especially in history taking and clinical examination, but also for investigations such as X-rays and common routine blood tests. It is therefore curious that even though there are a limited number of typical ECG *patterns* commonly found in routine paediatric cardiac examination, ECG interpretation is often viewed with trepidation and the common patterns are not readily recognised. Rather, the practitioner (or student) faced with an ECG blindly runs through the mantra of '*rate-rhythm-axis*', ignoring (or not recognising) the blatant abnormalities that in clinical context may supply clues to an immediate cardiac diagnosis. A cyanosed newborn with a left axis has tricuspid atresia until proven otherwise; a 6-year-old boy with weakness and large R waves in lead V1 probably has Duchenne muscular dystrophy; a child with Down syndrome and a left axis has an AV canal defect; a child with a history of syncope and unusual T waves may have long QT syndrome. It is not difficult to recognise, within clinical context, a diagnostic ECG pattern. The technicalities of rate, rhythm and axis remain as important as ever, but may play only secondary roles in the diagnostic value of the ECG.

The aim of this article is to encourage a fresh look at the routine ECG: to firstly place it firmly within clinical context, to confidently recognise abnormal from normal, and then to use the common abnormal ECG *patterns* as a diagnostic window into the heart. As with all clinical investigations, the common abnormalities are readily recognisable and the more complex ones are best left to the experts.

This article begins by reinforcing the recognition of the normal ECG patterns of children and then, by a series of clinical examples, places the commoner abnormal ECG patterns in context. Some important 'red flags' are highlighted and a structured categorisation of common paediatric ECG abnormalities is offered.

With this we hope that the ECG will become demystified and in doing so, that ECG patterns will once more regain their place in the routine clinical examination of the paediatric heart.

A few reminders

- In a normal heart the right ventricle (RV) is anterior to the left ventricle (LV) and is normally dominant in newborns and infants and is therefore also reflected in a normal ECG with a dominant R wave inV1.
- The LV gradually becomes more dominant with age.
- The P wave reflects atrial depolarisation, the QRS complex reflects ventricular depolarisation and the T wave reflects repolarisation.

ECG 1 is a normal ECG of an 8-year-old child, showing sinus rhythm, a heart rate of 90 beats/min, with a normal QRS axis of 30 degrees.

ECG 1. A normal ECG of an 8-year-old child.

Rhythm

Sinus rhythm is the normal rhythm characterised by a P wave preceding each QRS complex and a normal P axis. A normal P axis is 0 to +90 degrees, which translates to an upright P wave in leads I, II, aVF. A normal P axis indicates that the rhythm originates in the sinuatrial node in the right atrium. Tip: glance at lead aVR – all the waves should deflect away from this axis in a normal ECG.

The rhythm strip in ECG 2 shows a sinus arrhythmia which varies with respiration and is entirely normal.

ECG 2. The rhythm strip shows a sinus arrhythmia.

As with all clinical investigations, the common abnormalities are readily recognisable and the more complex ones are best left to the experts.

ECG 3 shows a patient with a low atrial rhythm, characterised by an abnormal P wave axis, indicating that the atrial impulse is not generated as it usually does in the SA node (inverted in II and aVF, only lead II shown) but at an ectopic focus low in the right atrium.

ECG 3. A patient with a low atrial rhythm.

Heart rate

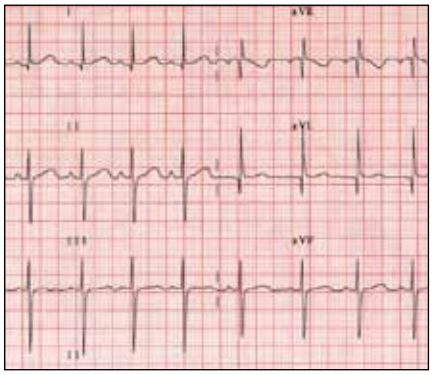
At the usual paper speed of the ECG at 25 mm/s, 1 mm (little square) = 40 ms and 5 mm = 200 ms.

There are multiple ways to calculate the heart rate. Two examples are given:

- count the number of QRS complexes in the rhythm strip in a standard 12-lead ECG, multiply that number by 6, which will give the heart rate
- count the number of large divisions (5 mm square) between two sequential R waves, divide that into 300, which will give you the average heart rate:
  - using the rhythm strip of ECG 1, the heart rate is 15 x 6 = 90
  - using ECG 3 as an example, the average heart rate is 300/3 = 100 (3 large divisions between two R waves).

QRS axis

ECG 4 is that of a child with Down syndrome presenting with cardiac failure and clinical signs of a left-to-right shunt. The QRS axis is superior with left axis deviation, suggesting an endocardial cushion defect.



ECG 4. A child with Down syndrome.

Determining the QRS axis is essential when evaluating children with potential congenital cardiac abnormalities. Newborns and infants normally have a more rightward axis with a gradual shift to the left with increasing age. The normal mean frontal QRS axis in infants and children is +60 degrees (range +10 to +110 degrees).

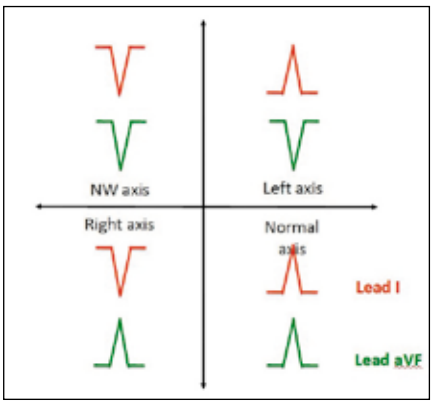


Fig.1. How to derive the mean frontal QRS axis of a 12-lead ECG at a glance by reference to limb leads I (in red) and aVF (in green).

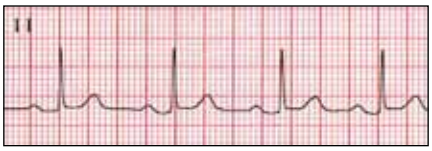
The easiest way to determine the axis is to look at the deflections of lead I and aVF. If they are both positive (R waves dominant), the axis lies between 0 and +90 degrees. It is simple to learn to recognise the 4 basic quadrants from the QRS deflections in leads I and aVF. Another way is to identify an equiphasic limb lead; the QRS axis will then be the lead that is orthogonal to that lead (Fig. 1).

Intervals

Atrioventricular conduction is assessed via the PR interval, QRS duration and QT interval.

The PR interval varies with age and heart rate. Prolongation of the PR interval (first-degree heart block) can be seen in normal individuals or is associated with myocarditis, acute rheumatic fever disease, cardiac anomalies such as Ebstein's anomaly, drug toxicity, etc.

ECG 5 is of a child presenting with a monoarthritis and a murmur, and showing a prolonged PR interval on her ECG. A diagnosis of acute rheumatic fever was made.



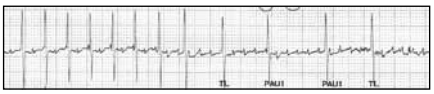
ECG 5. A child presenting with a mono-arthritis and a murmur.

A short PR interval is seen in Wolff-Parkinson-White syndrome (WPW), where there is an anomalous pathway between the atrium and ventricle. It is important to recognise WPW because there is a risk of sudden death. Parents may give a history of palpitations in their children which are episodes of supraventricular tachycardia (SVT). In any SVT it is important to do a 12-lead ECG after the patient has converted back to sinus rhythm. Only then will one be able to diagnose underlying rhythm abnormalities (see ECG 6).

ECG 6. A child with WPW syndrome.

ECG 6 is of a child with WPW syndrome: a short PR interval, a delta wave (initial slurring or QRS), and a wide QRS complex are seen. This child presented with an SVT.

ECG 7 is that of a neonate who presented to the day hospital with poor feeding; the nursing staff noted a tachycardia. He had a SVT which was terminated with adenosine, diagnosed with atrial flutter and needed cardioversion (see 'More About…' in this journal). Once cardioverted, atrial flutter hardly ever recurs.



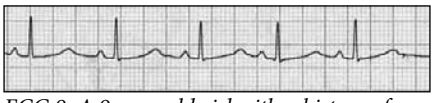
ECG 7. A neonate who presented with poor feeding and a tachycardia.

ECG 8 is of an asymptomatic 8-year-old girl with congenital complete heart block. In a complete (third-degree) heart block the atria and ventricles contract independently of one another, with the ventricular rate slower than the atrial rate: count the P waves and the QRS complexes (as above) and if the rates are different, then the child has heart block. If encountered in an infant, the child's mother should be investigated for systemic lupus erythematosus by doing Ro and La antibodies. Heart block may also be seen after corrective surgery for congenital heart disease.

ECG 8. An asymptomatic 8-year-old girl with congenital complete heart block.

QRS duration varies with age and is prolonged when there are ventricular conduction disturbances. Average QRS duration is less than 0.08 s in children, and less than 0.10 s in adults.

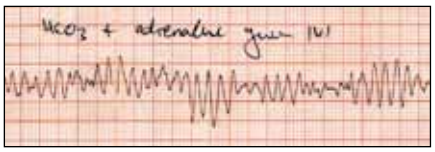
The QT interval also varies with heart rate, therefore the corrected QTc is calculated by using Bazett's formula. QTc should be less than 440 ms in children older than 6 months. A convenient rule of thumb: the QT interval should not be more than 50% of the previous RR interval, and the upslope and downslope of the T wave should be the same. Congenital long QT syndrome, a channelopathy, should be taken note of, as it is associated with torsades de pointes, which may result in sudden death. Triggers for the arrhythmia may be exercise, swimming, emotion, loud noise, but may also occur without these triggers. ECG 9 is of a 9-year-old girl with a history of sudden collapse, showing a prolonged QT interval.



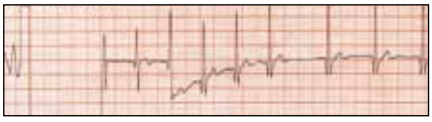
ECG 9. A 9-year-old girl with a history of sudden collapse, showing a prolonged QT interval.

ECG 10a is of a child who presented to the emergency department in severe shock. He was found to be pulseless and during chest compressions received adrenaline

boluses and bicarbonate. When the ECG tracing revealed intermittent torsades de pointes he was defibrillated and successfully returned to sinus rhythm (ECG 10b). He was subsequently diagnosed with a dilated cardiomyopathy, without long QT.



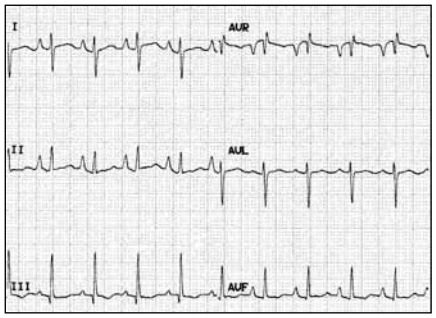
ECG 10a. A child who presented to the emergency department in severe shock.



ECG 10b. Patient defibrillated and successfully returned to sinus rhythm.

P waves

P waves are best viewed in leads II and VI. Their morphology is useful to assess right and left atrial size. ECG 11 is of a 4-year-old child with severe kyphoscoliosis and pulmonary hypertension. His ECG has as rightward mean frontal QRS axis and shows right atrial hypertrophy. Right atrial enlargement is seen as tall P waves in lead II (3 mm upper limit of normal in infants younger than 6 months and 2.5 mm as upper limit of normal in older children). Left atrial enlargement is seen as a wide or notched P wave in lead II (>0.1s) and/or a diphasic P wave morphology in V1.

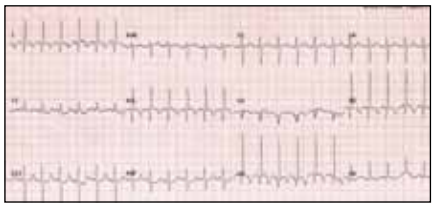


ECG 11. A 4-year-old child with severe kyphoscoliosis and pulmonary hypertension.

QRS amplitude/Q waves

As mentioned before, the RV is the dominant ventricle at birth and regresses gradually with the LV becoming the dominant ventricle. The former is reflected in the amplitude of the R wave in V1, V2, V4R (in paediatric ECGs, a lead is placed to the *right* of the sternum in a mirror position to the usual lead V4 position), aVR, relative to the smaller amplitude of the S wave in the above mentioned leads. With age, the amplitude of the R wave increases in the left precordial leads (V5 and V6).

Q waves represent the depolarisation of the interventricular septum. They are normally visible in lead I, II, III, aVF, V5, V6, but their duration should be less than 0.03 s, and they should be less than 5 mm deep. ECG 12, of a 4-month-old child who presented in congestive cardiac failure, reveals deep Q waves in leads I, aVL, V2 and V3, indicating an anterolateral myocardial infarct pattern. Deep Q waves are also seen in V5. This patient, who had cardiogenic shock secondary to cardiomyopathy, had an anomalous left coronary artery from the pulmonary artery (ALCAPA). The condition, diagnosable by ECG, is surgically correctable. It is therefore imperative that all children presenting with dilated cardiomyopathy have an ECG as part of their routine initial investigation.



ECG 12. A 4-month-old child who presented in congestive cardiac failure.

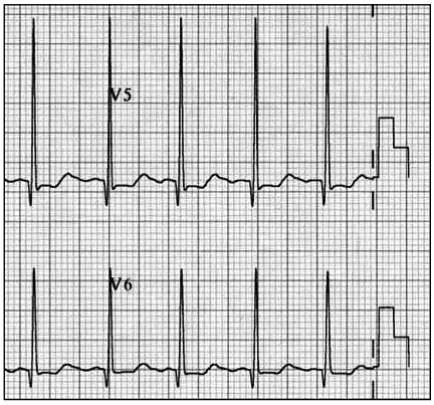
ECG 13 is of a patient who presented to a GP with a mild upper respiratory tract infection. The patient was cyanosed, but was otherwise well. The ECG is in sinus rhythm, a heart rate of 120 bpm (20 x 6), and right axis deviation, with right ventricular hypertrophy (R wave in lead V=16 mm as precordial leads are done at half standardisation. Also note this in the V4R lead). A clinical diagnosis of tetralogy of Fallot was correctly made.

ECG 13. A patient who presented to a GP with a mild upper respiratory tract infection.

RVH may be present in a number of conditions such as pulmonary hypertension, tetralogy of Fallot and pulmonary stenosis. In RVH all or some of the following criteria are met:

- right axis deviation
- R wave amplitude greater than the 98th percentile for age in lead V1
- S wave amplitude greater than the 98th percentile for age in lead V6
- R/S ratio in leads V1 or V2 higher than the upper limit of normal or a R/S ratio in lead V6 <1 after 1 month of age
- Upright T waves in lead V1
- Q waves in lead V1.

ECG 14 is of a 2-year-old child with aortic stenosis, showing left ventricular hypertrophy (LVH), reflected by tall R waves in leads V5 and V6 (precordial leads are done half standardisation as reflected by the calibration marker).



ECG 14. A 2-year-old child with aortic stenosis.

In LVH all or some of the following criteria are met:

- left axis deviation
- R waves amplitude in lead V6 >98th percentile for age
- S wave amplitude in leads V1 and V2 >98th percentile for age
- R/S ratio less than the lower limit of normal in leads V1 and V2 (towards the LV)
- Q waves more than 5 mm in leads V5 and V6
- T wave abnormalities (inversion of T waves in leads II, III, aVF, V4, V5, V6 is a strain pattern, suggestive of LVH).

In biventricular hypertrophy, the Katz-Wachtel phenomenon may be present: equiphasic QRS complexes present in two or more limb leads and in mid-precordial leads.

Ventricular conduction disturbances

The most common ventricular conduction disturbance in children is a right bundle branch block (RBBB), as seen in ECG 15.

ECG 15. A right bundle branch block (RBBB).

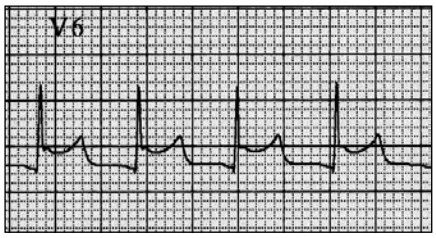
In a RBBB there is delayed conduction through the right bundle, leading to delayed depolarisation of the right ventricle. This causes lenghtening with typical terminal slurring of the QRS complex. The depolarisation of the ventricles happens at different times, so the right and left ventricular forces are therefore unopposed, with large voltages on the ECG, which cannot be interpreted as hypertrophy (slurred R wave in leads V1, V2, slurred S wave in leads V5, V6).

A RBBB can typically be seen in patients after cardiac surgery involving a right ventriculotomy, e.g. repair of tetralogy of

Fallot. An incomplete RBBB in an ASD is the result of a dilated RV and not disruption of the bundle as such. LBBB is rare in paediatric ECGs.

ST segments/T waves

ECG 16 shows ST segment elevation in a child who developed a pericardial effusion after VSD repair.



ECG 16. ST-segment elevation in a child who developed a pericardial effusion after VSD repair.

Pathological ST segments (elevation or depression) are seen in electrolyte disturbances, digitalis effect, myocarditis, pericarditis, etc. T-wave changes are associated with conditions that involve the ST segments as well. T waves in V1 are normally upright after birth, invert after the first week of life, and become upright again later in childhood (after 6 years of age).

Conclusion

The ECG is an invaluable tool when evaluating children with potential cardiac disease, but should be placed in clinical context and never be read in isolation. Some common ECG patterns are easily recognisable and may aid in making a bedside clinical diagnosis which, at times, may be of great benefit to your patient.

Further reading available at www.cmej.org.za

In a Nutshell

- It is often possible to adopt a pattern recognition approach in paediatric ECGs.
- This approach should be complemented with a systematic approach to avoid missing subtle abnormalities.
- ECGs should be evaluated in the clinical context, i.e. never forget the patient!

Single suture

Green tea and red laser attack Alzheimer's plaques

It may sound like a strange brew, but green tea and red light could provide a novel treatment for Alzheimer's disease. Together, the two can destroy the rogue 'plaques' that crowd the brains of people with the disease. The light makes it easier for the green-tea extract to get to work on the plaques.

Andrei Sommer at the University of Ulm in Germany, and colleagues, have previously used red light with a wavelength of 670 nanometres to transport cancer drugs into cells. The laser light pushes water out of the cells and when the laser is switched off, the cells 'suck in' water and any other molecules, including drugs, from their surroundings.

Now, Sommer's team have found that the same technique can be used to destroy the beta-amyloid plaques in Alzheimer's. These plaques consist of abnormally folded peptides, and are thought to disrupt communication between nerve cells, leading to loss of memory and other symptoms.

The team bathed brain cells containing beta-amyloid in epigallocatechin gallate (EGCG) – a green-tea extract known to have beta-amyloid inhibiting properties – at the same time as stimulating the cells with red light. Beta-amyloid in the cells reduced by around 60 per cent. Shining the laser light alone onto cells reduced beta-amyloid by around 20%.

It can be difficult getting drugs into the brain, but animal experiments show that the green-tea extract can penetrate the so-called blood-brain barrier when given orally together with red light. The light, which can penetrate tissue and bone, stimulates cell mitochondria to kick-start a process that increases the barrier's permeability, says Sommer.

There is no reason why other drugs that attack beta-amyloid could not be delivered to the brain in the same way, he adds.

'This important research could form the basis of a potential treatment for Alzheimer's, with or without complementary drug treatment,' says Mario Trelles, medical director of the Vilafortuny Medical Institute in Cambrils, Spain.

'The technique described could help to regulate and even stop the appearance of this disease,' he adds.

New Scientist, 3 November 2011.