ECG Quiz

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Figure 1a: ECG tracing of lead I

Figure 1b: ECG tracing of lead aVF

Electrocardiogram I.

Questions

1. Describe the rhythm seen in the electrocardiogram (ECG) in Figure 1a and b.
2. What are the possible mechanisms responsible for the production of this rhythm?
3. Is this indicative of a disease process?

Answers and discussion

There are a few basic principles that should be followed when interpreting any ECG: evaluate rate, rhythm and shape. There are two distinctly different QRS complexes present in ECG 1, waves A and B. Each wave “A” is followed by (coupled with) wave “B” at a fixed R-R interval of 560ms. This makes the rhythm a fixed bigeminal rhythm. The A-A rate is 33.3 beats per minute, as is the B-B rate. When combined, the rate is 70 bpm. The rhythm is irregular, with unequal A-B vs. B-A intervals. However, the A-B intervals are constant, as are the B-A intervals, making this a regularly irregular rhythm. Finally, the two waveforms are obviously of different morphologies, B being considerably wider and taller than A, and never preceded by a P wave. These characteristics indicate that wave B is of ventricular origin. Therefore, the rhythm seen in the ECG strip is a regular, fixed coupling, ventricular bigeminal rhythm. Additionally, all the ventricular QRS-T complexes in a given lead are identical (monomorphic). Finally, there is a long interval between the abnormal T wave and the following normal P wave.

There are two mechanisms that are thought to be involved in the generation of premature ventricular complexes (PVCs). They are automaticity and re-entry. All myocardial cells have the potential to be pacemakers for the heart. The sinoatrial (SA) node normally predominates because it has the fastest rhythm, thereby suppressing all other pacemakers. If the SA node were to cease firing, another focus, such as the AV node, would take over the role of pacemaker. Automaticity means that there is an abnormally active pacemaker site in the heart, which is firing and causing myocardial excitation despite a normally functioning SA node. The underlying problem is impulse formation. This situation results in irregular ectopic ventricular beats. Anything which brings resting myocardiocyte membrane potential towards threshold will increase the automaticity of these potential pacemakers; they will be more likely to fire abnormally. Things that increase the automaticity of myocardial tissue are hypothermia, hypoxemia, hypokalemia, and hypercalcemia.

A re-entrant mechanism is one in which the normal current originating in the SA node passes through the ventricular tissue multiple times per impulse due to either auxiliary conduction pathways or an area of abnormally depressed conduction, such as that created by infarction. Since the generation of the second (or third etc.) ventricular impulse is dependent on the normal SA node-generated current, the abnormal complex is linked to the normal complex. There is a repeated fixed interval between the normal and abnormal complexes. Thus, the rhythm is regular in its irregularity. Circus rhythm, which can cause atrial fibrillation or atrial flutter, is an example of a re-entrant mechanism arrhythmia. In such cases there may be multiple sites of ischaemic tissue usually secondary to coronary artery disease.

If the PVCs are identical in form (monomorphic), they are said to originate at one site (unifocal); if they vary in form (polymorphic), they are said to have more than one site of re-entry (multifocal). Re-entrant rhythms are usually sudden in onset (paroxysmal), regular in rhythm, and respond well to parasympathetic or DC shock stimulation. It has additionally been shown that re-entrant rhythms and bigeminy, being dependent on a necessary interval between SA impulses, are often suppressed by brady- or tachy-cardia.

The long interval between the abnormal T wave and the following normal P wave is known as a compensatory pause. The impulse generated at the SA node is conducted in a normal fashion through the atria and is evident as a P wave. But the impulse is blocked at the ventricles since the ventricular myocytes are either currently excited by the re-
following the re-entrant current, rendering them incapable of being further excited by the SA current. Since the rhythm is regular, it is possible to predict the position of these nonconducted P waves as being half way between the two adjacent conducted P waves. They are apparent as low-voltage depolarizations in the otherwise smooth contour of the PVC (not apparent in this ECG tracing).

Bigruminal rhythms can be physiological and do not require treatment unless they are symptomatic.

**Electrocardiogram 2.**

**Questions**

1. What are the two main findings on this ECG?
2. What is the most likely underlying diagnosis?
3. At what temperature was this tracing taken?

**Answers and discussion**

The two main findings on this ECG are the violently unstable baseline and the presence of J waves (Osborn waves) on the QRS complexes (arrowheads). The jagged baseline tracing is due to rapid skeletal muscular contractions. The patient is shivering.

The J wave is an “extra deflection at the end of the QRS complex”3. Also known as the Osborn wave, the J wave was first described in the early 1950’s and was thought to be pathognomonic for hypothermia. Indeed, it is present in the ECGs of 80% of hypothermic patients; however, it is also seen in Chagas’ disease (*Trypanosomiasis cruzi* infection), Brugada syndrome (characteristic ECG changes of idiopathic origin with high association with ventricular fibrillation/sudden death)5, and hypercalcemia. It has also been described in post-myocardial infarct patients and persons with no known pathophysiology. The most likely diagnosis in this patient is hypothermia, given the apparent shivering.

Hypothalamic control of core body temperature, as manifested by shivering, is absolutely lost below 29°C, and severely impaired below 34°C. Therefore, this ECG was taken when the patient was warmer than 29°C.

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The J wave is caused by an exaggeration of the transmural voltage gradient between the epicardial and endocardial layers of the heart, which is created in such conditions. The amplitude and duration of the J wave is inversely related to the core body temperature. The J wave does normally disappear with proper rewarming or treatment of the underlying disease but the danger of J wave appearance is the increased risk of potentially fatal ventricular arrhythmias and sudden cardiac death.

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**References**