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Multiple electrocardiographic anomalies during anaesthesia in an athlete

A 22-year-old athlete was scheduled for a minor surgical procedure under general anaesthesia. During anaesthesia, his electrocardiogram demonstrated multiple episodes of dysrhythmias including complete bundle branch block, atrioventricular (AV) block, isorhythmic atrioventricular dissociation with junctional rhythm. Administration of atropine 1.0 mg IV terminated the last episode of dysrhythmias. Postoperatively, a resting 12-lead electrocardiogram showed first degree AV block, ST-segment elevation and prominent U waves. A 24 hour Holter recording demonstrated first degree atrioventricular block, episodes of marked sinus arrhythmias and one episode of sinus tachycardia at a rate of 152 beats·min⁻¹. Treadmill stress testing revealed peak achieved heart rate of 200 beats·min⁻¹ without ischaemia. These findings collectively indicated athletic heart syndrome. Implications of athletic heart syndrome for the anaesthetist are reviewed and discussed.

The athletic heart syndrome was recognized as early as 1892.¹ The characteristic features of athlete's heart is that the heart is physiologically larger with above-normal stroke volume and has slower rates, longer sinus pauses, an increased incidence of atrioventricular (AV) block and early repolarization.²⁻⁴

Athletes can be divided into two general categories,⁵ those who perform dynamic (isotonic) exercise (isotonic athletes – e.g., long distance runners) and those who

perform isometric exercise (isometric athletes – e.g., weight lifters). In the former category, the heart is exposed to a chronic volume overload while in the latter group there is chronic pressure overload.

Morganroth *et al.*⁶ found that isotonic exercise tends predominantly to increase left ventricular cavity size, whereas isometric exercise increases wall thickness.

This report describes an athlete who developed multiple electrocardiographic abnormalities, without haemodynamic alterations, during general anaesthesia.

Case report

A 22-year-old professional soccer player, weighing 66 kg, was scheduled for septorhinoplasty under general anaesthesia. Physical examination revealed a well-developed, well-nourished male with a heart rate (HR) of 56 beats·min⁻¹ and arterial blood pressure of 110/60 mmHg. Examination of the heart, chest, abdomen and central nervous system was normal. There was no history of previous anaesthesia and preoperative chest x-ray, serum electrolytes and haemoglobin concentration were within normal limits. Diazepam 10 mg orally was given for premedication.

Anaesthesia was induced with fentanyl 150 µg, thiopentone 350 mg and atracurium 30 mg. Following intubation, anaesthesia was maintained with 70 per cent nitrous oxide in oxygen supplemented with one per cent enflurane. Ventilation was adjusted to maintain normocapnia and end tidal CO₂ was monitored by a Datex infra-red CO₂ analyzer. The ECG (Lead II) was continuously monitored using a Medishield M1 monitor. Arterial blood pressure was measured every five minutes using an electronic oscillotonometer (Dinamap). For the first 20 minutes after induction of anaesthesia, heart rate varied from 62 to 64 beats·min⁻¹ and arterial blood pressure ranged from 115/65 to 120/73 mmHg.

Ten minutes after the start of surgery, the ECG (Figure 1 "A") demonstrated complete bundle branch block with isorhythmic atrioventricular dissociation. The junctional rate was 56 beats·min⁻¹. This abnormal rhythm spontaneously changed to sinus bradycardia (58 beats·min⁻¹) with normal QRS configuration and first degree atrioven-

Key words

SPORT: heart, anaesthesia; ANAESTHETIC COMPLICATIONS: dysrhythmias; SYNDROME: athletic heart syndrome.

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FIGURE 1 Intraoperative ECG rhythm changes. See text for details.

tricular block. The P-R interval was 0.26 sec (normal 0.12–0.20 sec). Six minutes later the patient sustained another episode of isorhythmic atrioventricular dissociation with complete bundle branch block (Figure 1 "B"). This episode lasted for three minutes then abruptly changed to junctional rhythm and complete bundle branch block (Figure 1 "C"). Ten minutes later (Figure 1 "D") the ECG demonstrated a normal sinus rhythm (63 beats·min⁻¹), with persistent complete bundle branch block and first degree atrioventricular block (P-R 0.46–0.52 sec). Throughout these episodes of dysrhythmias the patient remained haemodynamically stable and the blood pressure ranged from 117/65 to 124/69 mmHg. No vasoconstrictors were used locally and the onset of dysrhythmias was not related to surgical stimulation. The first three episodes of dysrhythmias were self-limiting and lasted for two to four minutes only. However, the last one was persistent. Administration of atropine 1 mg IV during the last episode of dysrhythmias was associated with return of sinus rhythm (Figure 1 "E"). No further ECG abnormalities were noted during the course of surgery which lasted for one hour. Arterial blood samples obtained during anaesthesia showed that blood gases, acid–base status and potassium values were normal. At the end of the procedure the heart rate was 66 beats·min⁻¹ and the residual

neuromuscular blockade was reversed with atropine 1.2 mg followed by neostigmine 2.5 mg IV. No ECG abnormalities occurred during reversal.

Postoperative clinical cardiological evaluation did not reveal any abnormalities. Further assessment included a resting 12-lead ECG (Figure 2), 24 hour Holter recording and treadmill stress testing.

The 24 hr ambulatory Holter recording demonstrated first degree atrioventricular block, infrequent episodes of marked sinus dysrhythmias including one episode of sinus tachycardia at a rate of 152 beats·min⁻¹ which occurred during sleep. Treadmill stress testing (Bruce Protocol) revealed peak achieved heart rate of 200 beats·min⁻¹ after 15 min and 36 sec which exceeded the predicted maximum for his age (197 beats·min⁻¹). The exercise tolerance was graded as very good and no ischaemic changes were noticed. Echocardiography, chest x-ray, serum electrolytes, antistreptolysin O titre and sedimentation rate were all within normal limits.

In view of the patient's history, clinical examination, ECG, Holter monitor recording, treadmill stress testing and other laboratory investigations, the diagnosis of athletic heart syndrome was made. No further management was required and the patient was instructed to continue his normal life activities and regular training.

Discussion

The athlete's heart is likely to show a wide range of ECG abnormalities.⁴ Our patient demonstrated multiple episodes of dysrhythmias including bradycardia, complete bundle branch block with isorhythmic atrioventricular dissociation and junctional rhythm with prolonged P-R interval, the longest was 0.52 seconds. In addition, these dysrhythmias were fluctuating during anaesthesia.

Resting sinus bradycardia is the most frequent finding in more than 50 per cent of dynamic (isotonic) athletes.^{7–9} The current evidence suggests that resting bradycardia and associated dysrhythmias are due to an intrinsic cardiac component,^{10,11} not as previously attributed to an increase in vagal tone or to an alteration in neural input to the heart.^{12–14} Bullock and Hall¹⁵ reported an athlete who manifested bradycardia (heart rate fell to 30 beats·min⁻¹) during anaesthesia for a minor surgical procedure.

Complete bundle branch block has been described in six athletes,¹⁶ and it is considered to be rare.⁵ Our patient, however, had more than one episode of complete bundle branch block under anaesthesia. Nakamoto¹⁷ reported a ten per cent incidence of Wenckebach block (Mobitz I) (which invariably disappeared with exertion) in marathon runners. This prevalence is highly significant than 0.003 per cent found in an asymptomatic male population.¹⁸ Similarly, the prevalence of first degree atrioventricular block is higher in athletes (10 to 33 per

FIGURE 2 A resting 12-Lead ECG (postoperatively) showing normal sinus rhythm, heart rate $70 \text{ beats} \cdot \text{min}^{-1}$, first degree atrioventricular block (P-R interval 0.26 sec.), elevated S-T segment (early repolarization) in Leads II, III, aVF, V₂-V₆ and prominent U waves in Leads V₁-V₄.

cent)^{17,19} compared to 0.65 per cent found in the general population.

Alterations in repolarization consist of ST-segment elevation and prominent U waves was present in this case. The prevalence of ST-segment elevation varies widely in athletes. Lichtman *et al.*²⁰ reported a ten per cent incidence in over 500 athletes. Prominent U waves are occasionally observed in the athlete's heart. Elevation of ST-segment is assumed to be benign and not indicative of myocardial disease in athletes.⁵ The normal athlete does not suffer from any discomfort correlated with bradycardia, pauses or both. The athlete's heart is "at its best" under load and unlike a diseased heart does not show progressive abnormality with exercise,⁴ as demonstrated in this case with the treadmill stress testing.

Cardiac dysrhythmias occur in 60 per cent or more of anesthetized patients.²¹ Rhythm changes such as sinus bradycardia, first degree AV block and AV dissociation with a junctional rhythm are relatively common with deep levels of enflurane and may very well have contributed to this patient's rhythm disturbances.²² The role of

enflurane in producing complete bundle branch block is much less clear.

It is unlikely that 150 µg of fentanyl would have contributed to the observed rhythm disturbances which occurred 20 minutes after induction. It is also unlikely that dysrhythmias were due to atracurium or its breakdown products.^{23,24}

Athletic heart syndrome is a benign condition and posed no threat to life for the patient. This case demonstrated that athlete's heart may demonstrate alterations in the ECG simulating those of organic heart disease. Under anaesthesia, these dysrhythmias can be so confusing, that anaesthetist must be reminded of this phenomenon.

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Résumé

Un athlète de 22 ans était cédulé pour une procédure chirurgicale mineure sous anesthésie générale. Lors de l'anesthésie, l'électrocardiogramme a démontré de multiples épisodes d'arythmie incluant un bloc de branche complet, un bloc AV, une dissociation atrioventriculaire isorythmique avec un rythme jonctionnel. L'administration d'atropine 1.0 mg IV mit fin à la dernière épisode de dysrythmie. En période postopératoire, un électrocardiogramme à 12 dérivationes a démontré un bloc AV du premier degré, une élévation du segment ST ainsi qu'une onde U proéminente. Un Holter de 24 heures a démontré un bloc AV du premier degré, des épisodes marqués d'arythmie sinuale et un épisode de tachycardie sinuale avec une fréquence de 152 battements/minute. Un électro d'effort n'a pas démontré d'ischémie avec une fréquence maximale de 200 battements/minute. Ces trouvailles nous indiquent collectivement la présence d'un syndrome du cœur athlétique. Les implications de ce syndrome du cœur athlétique pour l'anesthésie sont revues et discutées.