THE WOLFF-PARKINSON-WHITE SYNDROME AND GENERAL ANAESTHESIA

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SUMMARY

The significance of the Wolff-Parkinson-White syndrome for the anaesthetist is illustrated by a case report. There is a tendency to paroxysmal supraventricular tachycardia and there may be associated congenital cardiac abnormality. In the presence of a rapid heart rate, arising spontaneously or after atropine, the distinctive W-P-W electrocardiographic pattern usually disappears and only when the pulse rate slows will the W-P-W pattern reappear. Thus in the absence of a previous electrocardiogram the diagnosis of W-P-W syndrome cannot usually be made when a patient presents, in the anaesthetic room for instance, with a very rapid pulse rate.

The electrocardiographic syndrome of short P-R interval and wide abnormal QRS complex was first described in 1930 by Wolff, Parkinson and White, who reported eleven cases. They drew attention to its occurrence in young people in good health apart from a tendency to paroxysmal tachycardia. They stressed that the electrocardiogram could become normal spontaneously, following exercise or administration of atropine. They concluded that vagal influences were largely responsible for the condition and that no serious heart disease was apparent. Following this definition of the syndrome many similar cases were described by others and the related literature became extensive. The syndrome is commonly called after Wolff, Parkinson and White, but other names used are: accelerated conduction syndrome, ventricular pre-excitation syndrome, and syndrome of short P-R interval with bundle branch block.

With more information to hand the syndrome could not be explained only in terms of vagal influences and it was thought that the condition was due to the presence of accessory conduction tissue between the atria and ventricles as described by Holzmann and Scherf (1932) and Wolfert and Wood (1933, 1941). Prinzmetal and his colleagues (1952) have suggested that the syndrome results from accelerated conduction through an irritable part of the atrioventricular node itself. Accessory conducting tissue, sometimes called the Bundle of Kent, has been found at autopsy in some, but not all, cases of the W-P-W syndrome (Lev et al., 1961, 1963). Support for the congenital nature of the syndrome is given by its familial incidence (Averill, 1956) and its association with other congenital cardiac lesions; for example Ebstein’s anomaly of the tricuspid valve.

The W-P-W syndrome is of particular interest to the anaesthetist because of the electrocardiographic and clinical changes which can occur suddenly and unexpectedly. Fear or premedication with atropine can cause disproportionate tachycardia distressing to the patient and puzzling to the anaesthetist. Furthermore, the electrocardiogram usually ceases to show the typical pattern of W-P-W syndrome and may either be normal or show S-T changes suggestive of cardiac infarction. On the other hand, pathological electrocardiographic changes masked by the W-P-W syndrome may be revealed for the first time.

A case is here described which illustrates many of the points made in previous accounts of the W-P-W syndrome and serves to emphasize the variation in the electrocardiogram which can occur in relation to anaesthesia.

CASE REPORT

A fit girl, aged 16 years, was presented in July 1967 for surgical enlargement of an auditory meatus and tonsillectomy. Weight 51 kg; height 155 cm; BP 120/80 mm Hg; pulse rate 82 beats/min; sinus arrhythmia; not anaemic; no history of palpitations, fainting or shortness of breath. There were no chest deformities;
Electrocardiogram during anaesthesia showing right axis deviation (+100°) with partial right bundle branch block as shown by rSr' in V1. T waves are inverted in leads II, III, AVF and V6, and there is slight S-T depression in leads II, III and AVF. Rate 94 beats/min.

the apex beat was within the mid-clavicular line; heart sounds were normal apart from a split second sound in the pulmonary area which remained split on expiration; no heart murmurs; all pulses were present and normal.

Anaesthesia was induced with thiopentone 200 mg, a cuffed endotracheal tube 8.0 mm was passed after suxamethonium 40 mg, and a pack was placed in the pharynx. Anaesthesia was maintained with nitrous oxide, oxygen, halothane 0.5 per cent, tubocurarine 20 mg, and intermittent positive pressure ventilation with a Manley ventilator (expired tidal volume 600 ml, minute volume 8 L/min, applied pressure 25 cm H2O).

Shortly after induction of anaesthesia an electrocardiograph was connected to the patient and it was noted that there was right axis deviation (+100°) with partial right bundle branch block as shown by rSr' in V1. T waves were inverted in leads II, III, AVF and V6, and there was slight S-T depression in II, III and AVF (fig. 1). In normal patients in this age group S-T depression and T inversion in posterior leads may occur, but here it was very marked and probably abnormal. The T wave inversion in V6 was certainly abnormal. The pulse rate was 94 beats/min. The patient's general condition was excellent and it was decided to proceed with the surgery which occupied 2 hours. The electrocardiographic pattern remained unchanged.

At the end of the operation the muscle relaxant was reversed with neostigmine 1.25 mg given 3 minutes after atropine 0.6 mg; this procedure was repeated a few minutes later with the same doses of neostigmine and atropine. The pulse rate did not vary by more than 10 beats/min following each of these drugs and no change was observed in the e.c.g. pattern. The patient left the theatre breathing adequately and responding to commands. Oxygen was administered for a period postoperatively as a routine precaution.

An electrocardiogram 48 hours later in the ward showed Wolff-Parkinson-White phenomenon (fig. 2), i.e. the P-R interval was short (0.08–0.10 sec) and the QRS complex was widened by an initial shirring called the delta wave. The rate was 80 beats/min. The change of the electrical axis to the left (−45°) can be seen in leads I and III. Electrocardiograms taken 5 months and 6 months later were identical and it is likely, therefore, that the W-P-W pattern was a constant feature in this patient. An interesting finding in the electrocardiogram taken at 5 months was a ventricular extrasystole, as it is thought that attacks of supraventricular tachycardia begin with a ventricular or atrial premature beat which sets up a circus movement between the atria and the ventricles, involving the main and accessory bundles. No tachycardia followed in this instance.

When it seemed likely that the patient would need a general anaesthetic to allow change of a surgical pack, it was decided to administer hyoscine intravenously as premedication under electrocardiographic observation. Hyoscine can produce adequate inhibition of secretions without producing pronounced tachycardia and for this reason was chosen in preference to atropine. The initial effect of 0.25 mg of hyoscine intravenously was to slow the pulse rate from 75 to 60 beats/min, but within 3 minutes the rate had returned to 75 beats/min. Figure 3 shows the electrocardiographic findings in the limb leads before and 20 minutes after the hyoscine. The complexes were unchanged and this dose of hyoscine produced satisfactory inhibition of secretions and a drowsy patient. The change of pack was accomplished without general anaesthesia.

Chest X-rays showed no abnormality of the cardiac outline or of the pulmonary blood vessels. Because of the familial incidence of the W-P-W syndrome, electrocardiograms were recorded in the two siblings and both parents. Father is the only member of the family who has palpitations and it is interesting
Fig. 2
Electrocardiogram 48 hours after operation showing W-P-W pattern. The electrical axis is now to the left (−45°). Rate 80 beats/min.

Fig. 3(A)
Electrocardiogram before hyoscine.

Fig. 3(B)
Electrocardiogram after hyoscine 0.25 mg i.v.
that he showed one complex with a slurred initial upstroke of the R wave. This suggests that father has latent W-P-W syndrome which could account for his occasional episodes of palpitations. The electrocardiograms of other members of the family were normal.

DISCUSSION

Duthie (1946) used atropine as a diagnostic agent in W-P-W syndrome and comparison with his findings suggests that the changes produced in this patient's electrocardiogram during general anaesthesia were probably due to atropine given in the premedication. That the syndrome can give rise to complications during surgery is shown in a case report by Burchell and his colleagues (1967), in which they described the onset of frequent short paroxysms of supraventricular tachycardia during the repair of an atrioseptal defect in a patient with W-P-W syndrome. Supraventricular tachycardia arising postoperatively in a case of unsuspected W-P-W syndrome has been described by Meyer and Greenberg (1966) and only after a normal sinus rhythm had been established by direct current cardioversion was the W-P-W pattern revealed. This patient had given a history of palpitations but no pre-operative electrocardiogram had been recorded. Occasionally the W-P-W phenomenon has been seen for the first time during cardiac catheterization (Kossman et al., 1950) or during induction of anaesthesia (Kay, 1967). Sudden death has been known to occur during tachycardia in the W-P-W syndrome (Fox, Weaver and March, 1952). It should be of interest to the anaesthetist to know how often he is likely to meet cases of W-P-W syndrome and what incidence of associated cardiac abnormalities is to be expected. The syndrome occurs in all ages, but more often under the age of thirty and more commonly in males. Hiss and Lamb (1962) found an incidence of 0.15 per cent in a very large study of Air Force flying personnel. Schiebler, Adams and Anderson (1959) found that 24 of 83 individuals with W-P-W syndrome had Ebstein's anomaly of the tricuspid valve and 13 of 28 infants and children with W-P-W syndrome had congenital cardiac lesions.

Five to 10 per cent of individuals who suffer from recurrent paroxysmal tachycardia have W-P-W syndrome and 25-50 per cent of those with W-P-W phenomenon get recurrent paroxysmal tachycardia (Kay, 1967). The management of paroxysmal tachycardia associated with W-P-W syndrome is that of any other supraventricular tachycardia and treatment is required when there are signs of decompensation such as fall in blood pressure. The administration of oxygen should be the first-aid measure and massage of the carotid sinus may terminate the attack without recourse to drug therapy or direct current cardioversion.

In the selection of drugs for general anaesthesia in cases of W-P-W syndrome careful consideration should be given to all drugs which affect the heart. It is suggested that hyoscine is preferable to atropine for premedication as discussed in the case report. Perhaps gallamine should be avoided for fear of precipitating a disproportionate tachycardia in these cases. Little change in pulse rate occurred and no change in electrocardiographic complexes was noted following administration of atropine and neostigmine during the reversal of muscle relaxant in the case described here. Fox, Weaver and March (1952), however, in a diagnostic investigation of a subject with W-P-W syndrome, found that neostigmine accentuated the W-P-W pattern after its modification by atropine.

It is suggested that all patients should be questioned as a routine about palpitations and an electrocardiogram recorded when their presence is reported.

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REFERENCES


LE SYNDROME DE WOLFF-PARKINSON-WHITE ET L'ANESTHESIE GENERALE

SOMMAIRE
La signification pour l'anesthésiste du syndrome de Wolff-Parkinson-White est illustrée par la description d'un cas. Il existe une tendance à la tachycardie supraventriculaire paroxysmale, et la présence d'une anomalie cardiaque congénitale associée est possible. En présence d'une tachycardie, spontanée ou consécutivement à l'administration d'atropine, l'électrocardiogramme W-P-W caractéristique disparaît habituellement et ne réapparaît que lorsque le pouls devient plus lent. Il n'est donc d'habitude pas possible d'établir, en absence d'un électrocardiogramme préalablement enregistré, le diagnostic d'un syndrome W-P-W lorsqu'un patient manifeste, p. ex. dans la salle d'anesthésie, un pouls très rapide.

DIE BEDEUTUNG DES WOLFF-PARKINSON-WHITE SYNDROMS FÜR DIE ALLGEMEINE ANÄSTHESIE

ZUSAMMENFASSUNG

BOOK REVIEW


At first sight a volume with this title might appear to be of negligible interest to anaesthetists. It is, however, a reflection of how wide the limits of the specialty now appear to be in this volume quite a number of articles which have bearing on our work. Perhaps of greatest interest are the many investigations of the usefulness of EACA in as diverse fields as after prostatectomy, in dental surgery, in the treatment of epistaxis and haematuria. The use of the analgesics both in surgery and in obstetrics also is mentioned in a number of the contributions. A valuable section on the corticotrophin treatment of myasthenia gravis which every anaesthetist ought to read. The same holds for the sections on the treatment of status epilepticus—with diazepam and a thiopentone drip. One abstract draws attention to the fact that adrenaline dropped into the eye can cause extrasytlesos in conscious patients. The authors concerned comment on the risk of this procedure in the hyperthyroid patient but anaesthetists will be more interested in the possible risks with halothane or agents with a similar action on the cardiac irritability.

The discussion on antibiotics and their side effects should be read by everyone tempted to use these for the prophylactic prevention of infection. A somewhat pungent comment from the Editor at the end of an account describing the uselessness of this therapeutic procedure asks how many times this has to be proved before it is accepted as fact.

There are also abstracts of articles dealing with the treatment with digitalis and with isoprenaline, which also bear on the work of the anaesthetist. There is one group of drugs which is not mentioned, namely the vasopressors. This, however, reflects not an omission on the part of the Editor but the fact that useful papers on the clinical use of these agents are so few and far between. On the whole, however, this is a most interesting volume into which every anaesthetist should dip occasionally.

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