on their own. The treated group were given oxyphenbutazone 250 mg twice daily in a suppository and had their legs bandaged with Lohmann's Dauerbinde (strong, or *kräftig*) for 14 days after the operation. Both legs were bandaged from toe to knee immediately after operation. The bandages were 10- or 12-cm wide, depending on the size of the patient's feet and legs. They were changed twice daily, or more often if necessary, and were worn at night. The bandaging significantly increased the velocity of the venous blood.[2]

Blood coagulation, blood in faeces, white cell count, packed cell volume, and electrolytes were recorded for 18 days postoperatively to detect any adverse reaction to treatment, especially to oxyphenbutazone.

Ascending phlebography[4] was performed 14 days or more postoperatively, or earlier when indicated clinically. The average was 19 days. Altogether 18 patients developed deep vein thrombosis. Of these, 5 were in the treated group (5/27) and 13 were controls (13/23). The difference was significant ($P<0.01$) according to Fischer's exact statistical test. No thrombosis in the treated group advanced beyond the popliteal fossa. No pulmonary emboli appeared in either group. One patient developed a rash, which disappeared after the withdrawal of oxyphenbutazone. No other side effects were noted. No change in the coagulation time was found.

Conclusion

The prophylactic effect of this regimen is comparable to that of conventional treatment with anticoagulants. It is simple to carry out and to supervise; it involves no risk of overdosage; the side effects are few and, as a rule, slight; it is well tolerated; and it calls for no unusual laboratory facilities.

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Pre-excitation and mitral valve prolapse

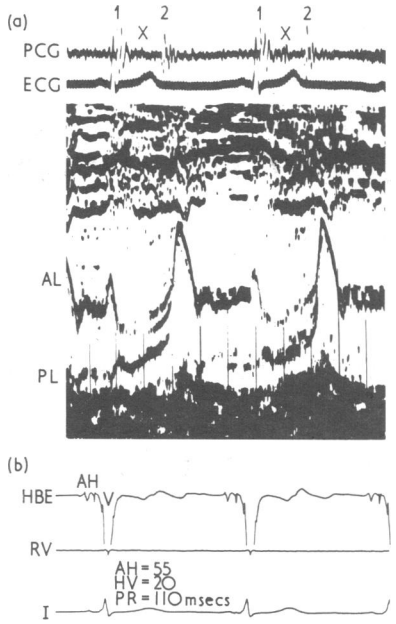
The syndrome of mitral valve prolapse is associated with a high incidence of both supraventricular and ventricular arrhythmias, which occasionally cause sudden death. Since the arrhythmias may be caused by pre-excitation it seems relevant that a case of Lown-Ganong-Levine (short P-R interval) syndrome, with an apical late systolic murmur and angiographic evidence of mild mitral regurgitation, had infranodal pre-excitation (so-called Mahaim fibres),[1] and that out of 68 patients with the Wolff-Parkinson-White syndrome seven (all with left-sided bypasses) had mitral valve prolapse.[2] We present here a further case of pre-excitation associated with mitral valve prolapse which we think is relevant to the understanding of both syndromes.

Case report

A 24-year-old primipara was referred nine weeks after confinement for investigation of an asymptomatic cardiac murmur found during her pregnancy. There was no relevant family history and no history of rheumatic fever. On examination the only abnormality was an apical mid-systolic click and murmur. An electrocardiogram showed a P-R interval of 0·10 s with normal QRS complexes. An echocardiogram showed a prolapsed posterior mitral valve leaflet. Intracardiac electrography with programmed electric stimulation showed a short HV time of 20 ms (normal 35-55 ms). This did not lengthen with rapid right atrial pacing or with induced right atrial premature beats—that is, once the impulse had traversed the AV node it entered the ventricle more quickly than would have been possible via the normal His-Purkinje system (pre-excitation due to the presence of Mahaim (nodoventricular) fibres).[3] The murmur was more obvious on auscultation than on phonocardiography. The findings of investigations are shown in the figure.

Discussion

The syndrome of a prolapsing mitral valve seems to have many possible causes, of which one may be an abnormality of the mitral valve annulus.[4] Such a defect has also been implicated in the abnormal persistence of atrioventricular connections that produces the Wolff-Parkinson-White syndrome.[5] We think that an abnormal mitral valve annulus may occasionally be the anatomical causative factor in both syndromes. That it is unusual is suggested by the absence of echocardiographic abnormalities in a further eight patients with left-sided pre-excitation we have studied and a lack of clinical evidence in a further 47. The relatively high incidence in one series[2] may be due to the severity of rhythm disorders in the patients referred for consideration of surgical treatment of the Wolff-Parkinson-White syndrome.



Case of mitral valve prolapse with pre-excitation. (*a*) Phonocardiogram (PCG) shows mid-systolic click (x) (much more obvious in second cycle); vibrations of systolic murmur are only barely perceptible. Corresponding ECG shows short P-R interval and echocardiogram shows separation of anterior (AL) and posterior (PL) leaflets of mitral valve in systole. Time markings = 0·20 s. (*b*) His bundle electrogram (HBE), with simultaneous right ventricular electrogram (RV) and ECG lead I, shows normal conduction through AV node (AH represents conduction time from low right atrium to bundle of His). HV time, which reflects conduction between proximal intraventricular conducting system and ventricle, shortened to 20 ms, indicating unduly rapid transmission of impulse from bundle of His to ventricular myocardium. Paper speed 100 mm/s.

Even if uncommon, the association is important since sudden death may unexpectedly occur in either syndrome. Therefore intracardiac electrophysiological investigations are indicated in patients with mitral valve prolapse whose ECG suggests pre-excitation. As mitral valve prolapse may not present auscultatory abnormalities we are examining patients with left-sided pre-excitation by echocardiography to explore this possible association.

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