Are there any useful investigations that predict which patients with bifascicular block will develop third degree atrioventricular block?

Syncpe is a common symptom, particularly in the elderly. In patients with cardiovascular disease, it is associated with a high mortality within the first year. Therefore, patients with syncpe that might be of cardiac origin need thorough investigation, and, if possible, should be offered a reliable treatment. One such treatment for bradyarrhythmias is pacing. The mortality in patients with documented heart block without pacing is about 50% during the first year. Second degree heart block has a similar prognosis.

Heart block arises most often in patients with conduction tissue fibrosis, and less often in those with coronary or other heart disease. A less serious form of conduction disease is intraventricular conduction delay in the bundle branches and their various fascicles. None the less, in some cases this form of “minor” conduction disease progresses to complete heart block, often accompanied by syncpe or attacks of the type described by Adams-Stokes-Morgagni. Furthermore, the association of newly acquired bifascicular block and some acute myocardial infarction is related to complete heart block (when a long HV interval is present) and to increased cardiovascular death.

There are many problems associated with bifascicular block. First, because the electrocardiogram often shows normal atrioventricular conduction after syncpe we can only be certain that the syncpe was caused by heart block. Nor can we predict which patients with bundle branch block (left bundle branch block or right bundle branch block with left hemiblock) will progress to complete heart block. Electrophysiological measurements are often abnormal, but seldom to the extent (in the case of the HV interval prolonged to 80–100 ms) that is associated with a high incidence of progression. Finally, even in patients with severe distal conduction disease, syncpe may be caused by other, coexistent abnormalities.

Electrophysiological studies to predict the development of complete heart block
Scheinman et al proved in 1973 that His bundle recordings could be used to distinguish between those who had neurological symptoms caused by transient high grade atrioventricular (AV) block and those with other causes. However, it was soon recognised that not all patients with transient AV block had considerable HV prolongation at the time of study, so that this marker was regarded as specific but not sensitive. A prospective study of a large group of patients showed that another way of predicting the development of AV block was to record from the His bundle during atrial pacing. However, few patients (10/496) in this observational study progressed to distal AV block, and only six out of 10 patients had block during atrial pacing, making such study unrewarding in most patients.

Pharmacological stress tests
In 1978 intravenous procainamide was reported to produce high degree heart block in patients with syncpe. Some groups regarded this pharmacological test as a useful method of assessing the need for pacing. Englund et al have recently used an intravenous disopyramide test as a pharmacological stress test (like procainamide, ajmaline, and flecainide) to assess the reserve of the His Purkinje system. It is clear that the stress test has a higher sensitivity than a test in the baseline state, and this is the merit of the present study. None the less the disopyramide stress test has specific limitations. What advantages does disopyramide have over procainamide, a drug which can be used in a standardised way? Disopyramide does indeed have favourable electrophysiological characteristics, but its haemodynamic profile is not as good as Englund et al suggest. They had only one case of hypotension. Their experience seems to be better than the general experience with disopyramide, which when taken by mouth has a depressant effect on the myocardium. Like others who have studied intravenous disopyramide, Englund et al seem to have been lucky. Disopyramide can cause severe hypotension in patients with arrhythmias: some patients have to be resuscitated. It seems premature to suggest that such a test is safe and can be performed outside an electrophysiology laboratory or without a temporary pacemaker.

Should pacemakers be implanted without further study?
Some advise implantation of a permanent pacemaker without additional studies in patients with symptomatic bifascicular block, or when syncpe remains unexplained. This seems reasonable, but Englund et al’s arguments for rejecting this option are valid too. The concept of a pacemaker that detects bradycardia is interesting, but bradyarrhythmias (for example, those caused by sinus node disease) may be sufficient to trigger ventricular pacing. Subsequent retrograde conduction into the AV node might cause anterograde AV block and hence reinforce the need for pacing. Therefore, we must be cautious about relying on information obtained by telemetry of pacemaker data, as was done in Englund et al’s study. Prophylactic implantation of a pacemaker in patients with bifascicular block is only appropriate if we can prove that
these patients need a pacemaker to avoid recurrent symptoms or sudden death. The occurrence of syncope in some patients with a pacemaker in the series reported by Englund et al is a striking finding.

The real causes of syncope in bifascicular block

Ventricular tachyarrhythmias are one of the leading causes of syncope. Monitored deaths were caused by ventricular fibrillation rather than heart block. Poor left ventricular function can also be important in the pathogenesis of syncope. Therefore, the (logical) exclusion of patients with a left ventricular ejection fraction of less than 35% is a major limitation of Englund et al’s study. Earlier studies had already shown that distal conduction disease in combination with congestive heart failure is an indicator of high risk for sudden death. Thus if patients are studied they should be those with poor ventricular function. A complete study includes not only provocation of heart block with a drug but also programmed electrical stimulation of the ventricle. If ventricular arrhythmias are induced, there are other treatments. I would be reluctant to implant a pacemaker without additional protective measures in a patient with inducible ventricular tachycardia. β-blockers which protect against sudden death and are promising in congestive heart failure might be useful. However, it is tempting to consider a transvenous pectoral pacemaker-defibrillator for syncope and inducible ventricular tachycardia. This will prevent sudden death caused by both bradyarrhythmias and tachyarrhythmias, but will not protect the patient against congestive heart failure.

Conclusions

In summary, patients with bifascicular block and syncope require further investigation. A disopyramide test may help to avoid unnecessary pacemaker implantations. However, other non-invasive tests (baroreflex sensitivity, tilt table testing) should be considered. When left ventricular function is poor, the risk of sudden death is high. A disopyramide or propranolol test does not seem to be the investigation of first choice in these patients, but the best strategy to investigate and treat these patients remains unclear. Treatment with β-blockers (or a pacemaker-defibrillator) is worthwhile considering, even in patients with poor left ventricular function.

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