Community Clinics in Clinical Pharmacology

Treatment of second-degree heart block

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British Medical Journal, 1977, **2**, 309

Case history

Mr E F, who is now 77, has been under the care of this practice all his life. He had no serious illness until 1972, when I found that he had pernicious anaemia, which responded well to treatment with vitamin B₁₂. Seven months ago he developed severe chest pain that lasted several hours and characteristic electrocardiographic changes of an inferior myocardial infarction. He was managed at home but subsequently developed congestive cardiac failure, which was controlled by frusemide, 40 mg daily, together with potassium supplements. He has had two episodes of acute left ventricular failure, however, over the past two weeks, both of which responded to a combination of intravenous frusemide and intravenous morphine. He is now breathless on the slightest exertion, and has severe ankle oedema. He is chair-bound, and his life is miserable. Can we help him?

Advice

I saw Mr F this morning at home. As you say, he is breathless on the mildest exertion, and he has bilateral ankle oedema, a raised jugular venous pressure (3 cm), an enlarged heart, a left-sided third sound, and crepitations at both lung bases. His blood pressure is 160 mm Hg, his pulse rate is 74/min, and an electrocardiogram shows second-degree heart block and "ischaemic" changes.

Under normal circumstances I would increase his frusemide dosage and digitalise him. This patient, however, has second-degree heart block. Cardiac glycosides not only increase myo-

cardial contractility but also impair atrioventricular conduction (both directly by acting on conducting tissues, and indirectly by reflex vagal stimulation). Digitalisation, therefore, carries the very serious risk of precipitating complete heart block. One of the morals from this patient is that we should always do electrocardiography before prescribing digitalis. I think that he should have a demand pacemaker inserted before you start digoxin.

This is not such a major undertaking in a man of 77 as one might think. The pacing electrode can be inserted through a peripheral vein and the pacemaker implanted in the axilla. The procedure causes minimal trauma, and the patient need only stay in hospital for a few days. Once Mr F has had his pacemaker inserted I would suggest that he should be started on digoxin but not more than 125 µg a day. Digoxin is eliminated by renal excretion. Because renal function declines with age, elderly patients are liable to develop digoxin toxicity at "conventional" doses. Estimates of renal function (such as serum creatinine concentration or, better still, creatinine clearance) may be useful in deciding the initial dose, but it is important to titrate the dose against the response.

Anorexia, nausea, vomiting, and sinus bradycardia are signs of digoxin poisoning. If any of these occur the dosage should be reduced. But it should be remembered that some patients may develop other toxic effects without these classic prodromal symptoms and signs. Firstly, digoxin may not only precipitate almost every type of arrhythmia, it may also actually *worsen* congestive cardiac failure: this is particularly important because of the temptation to increase the dose in these cases. Secondly, patients may develop headache, drowsiness, confusion, and even psychoses as a result of over-digitalisation. Several studies have shown that by taking the sort of precautions we have discussed already, you can reduce the incidence of digoxin toxicity by 80%.

Postscript

Mr F was admitted to hospital for insertion of a demand pacemaker. Although his recovery was complicated by a respiratory tract infection, the operation was highly successful. On discharge, he was digitalised with a daily digoxin dose of 125 µg and he is sufficiently mobile to go shopping.

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