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MEDICAL MEMORANDA

Cardiac Standstill due to Digoxin Poisoning Successfully Treated with Atrial Pacing

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British Medical Journal, 1973, 2, 526-527

Atrioventricular (A.V.) block due to digoxin toxicity is well recognized, but sinus arrest or sinoatrial block leading to atrial standstill may also occur (Bernstein *et al.*, 1965). We report a case of self-poisoning with digoxin where complete atrial and ventricular standstill occurred, and electrical pacing was required for 72 hours after ingestion of the drug.

Case Report

A 64-year-old man was found to be hypertensive in 1970. He also had ischaemic heart disease with congestive cardiac failure and renal failure (creatinine clearance 40 ml/min). He was treated with digoxin 0.25 mg/day, frusemide 120 mg/day, slow K 600 mg three times a day, and methyl dopa 250 mg four times a day. He was admitted to Glasgow Royal Infirmary as an emergency in July 1972 after taking an unknown quantity of these drugs.

On examination he was drowsy, confused, and had severe diarrhoea. The initial pulse was 48/min and regular. Blood pressure was 200/120 mm Hg. Examination was otherwise unremarkable. Investigations at this time showed plasma sodium 142 mEq/l, potassium 8.0 mEq/l, chloride 107 mEq/l, and urea 42 mg/100 ml. E.C.G. showed sinus bradycardia with pronounced S-T and T wave changes (fig. 1). Despite intravenous atropine the heart rate dropped to 25/min and he developed Cheyne-Stokes breathing and became deeply unconscious. A transvenous pacemaker was therefore passed to the apex of the right ventricle and satisfactory pacing at 80/min was achieved.

The initial hyperkalaemia was treated with soluble insulin and glucose and did not recur, the serum potassium two hours after admission being 6.1 mEq/l. and 36 hours later 5.0 mEq/l. Although the patient became hypotensive (90 mm Hg systolic), with the maintenance of the ventricular rate by the pacemaker peripheral perfusion was fairly satisfactory and a diuresis was established and maintained with mannitol and later with frusemide.

Owing to the patient's restless state the ventricular pacemaker was dislodged about four hours after insertion but the catheter

tip came to rest in a position high in the right atrium allowing successful atrial pacing for the next 72 hours. During this period a variety of conduction disturbances were noted: atrial standstill with complete ventricular standstill; atrial standstill with a very slow junctional escape rhythm unresponsive to atropine; atrial pacemaker with pronounced first degree A.V. block (P-R interval 0.48 sec), with transient improvement in atrioventricular conduction following short periods of asystole produced by switching off the pacemaker (fig. 2); atrial pacemaker with type I second degree A.V. block (Wenckebach) (fig. 3); and a junctional escape rhythm responsive to atropine (fig. 4). Neither ventricular ectopics nor ventricular tachyarrhythmias were seen.

FIG. 1—Lead II, continuous record, on admission shows sinus bradycardia and pronounced S-T and T wave changes (plasma digoxin 10.2 ng/ml).

FIG. 2—Lead II (5 p.m., day 3). Continuous record which shows atrial pacing with pronounced lengthening of P-R interval and asystole when pacemaker switched off. A transient improvement in atrioventricular conduction occurred after the asystolic period (plasma digoxin 4.5 ng/ml).

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Almost complete cessation of spontaneous cardiac activity persisted for over 48 hours, until a slow junctional rhythm responsive to the positive chronotropic effect of atropine was recorded some 50 hours after ingestion of the drug. After a further 24 hours sinus rhythm was restored and pacing discontinued. Recovery thereafter was uneventful and the patient was discharged after physical and psychiatric rehabilitation.

FIG. 3—Lead II (1 a.m., day 4). Continuous record showing atrial pacing with varying atrioventricular block (Wenckebach). (Plasma digoxin 3.6 ng/ml).

FIG. 4—Lead II (3.40 p.m., day 3) after atropine sulphate, 1.2 mg intravenously. Probable junctional rhythm with a ventricular rate of about 45/min. (Plasma digoxin 4.2 ng/ml).

FIG. 5—Plasma digoxin levels and regression line. Plasma samples after day 7 contained digoxin levels less than 0.25 ng/ml.

PLASMA DIGOXIN LEVELS

Plasma levels of digoxin were measured using the Wellcome Lanoxitest B digoxin radioimmunoassay kit (Ojala *et al.*, 1972) and are shown in fig. 5. The peak plasma digoxin level was 10.2 ng/ml. The plasma half-life was 38.4 hours, a value at the upper limit of normal (Doherty, 1968). The blood levels obtained were of the

same order as those previously described in cases of self-poisoning (Smith and Willerson, 1971; Citrin *et al.*, 1972) and confirmed exposure to a large quantity of the drug.

Comment

A high blood potassium has been previously reported in cases of severe poisoning (Smith and Willerson, 1971; Citrin *et al.*, 1972). This is probably due to inhibition of the membrane ATP-ase pump (Glynn, 1964) and responds readily to glucose and soluble insulin which is the most appropriate treatment (Scottish Medical Journal 1972).

CONDUCTION DISTURBANCES

Digitalis poisoning may produce atrial standstill or A.V. block (Smith and Willerson, 1971) and these disturbances are commonly associated with a junctional escape rhythm (Pick and Langendorf, 1968). Such a rhythm was not present in our patient initially and therefore artificial pacing was required. The fortuitous occurrence of atrial pacing in the circumstances described above permitted the maintenance of a satisfactory ventricular rate. Atrial pacing is not, however, recommended for digitalis-induced bradyarrhythmias because of the difficulty in ensuring satisfactory electrode position in the atrium and also because digitalis-induced high grade A.V. block would result in loss of pacing despite successful atrial capture.

Apart from transient 2:1 A.V. block 60 hours after ingestion of the drug, no serious A.V. conduction disturbances were encountered and pacemaker exit block was not seen. In the context of such severe depression of cardiac automaticity it is remarkable that conductivity remained satisfactory, especially as toxic doses of digitalis depress not only the automaticity of the sinoatrial node but also the conductivity of the atrium and A.V. node (Hoffman and Singer, 1964).

The transient shortening of the P-R interval after short periods of asystole produced by switching off the pacemaker is clearly shown (fig. 2). Similar improvement in A.V. conduction is seen in second degree (Wenckebach) A.V. block after failure of A.V. conduction.

The A.V. junction was the first area of the heart to regain automaticity some 50 hours after ingestion of the drug, when the plasma digoxin was about 5 ng/ml. The return of spontaneous cardiac activity was accompanied by recovery of the chronotropic response to atropine, which was a striking feature of recent cases of digoxin self-poisoning (Smith and Willerson, 1971; Citrin *et al.*, 1972).

We wish to thank Professor E. M. McGirr for permission to report the details of this case.

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