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MEDICAL MEMORANDA

Imipramine Poisoning: Survival of a Child after Prolonged Cardiac Massage

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British Medical Journal, 1974, 4, 508

Poisoning with tricyclic antidepressants is common and the clinical features are well known (*British Medical Journal*, 1974; Goel and Shanks, 1974). The present case illustrates that children who develop a critically low cardiac output from tricyclic poisoning or any other potentially reversible cause should have resuscitation procedures continued for long periods before hope is abandoned.

Case Report

A girl aged 3 years 8 months and weighing 13·6 kg was admitted to the casualty department about one hour after ingesting 18 25-mg imipramine tablets. She was unconscious and convulsing. A monitoring oscilloscope showed a ventricular tachycardia of 120/min. She was given two 50-joule D.C. shocks, which caused transient return to sinus rhythm followed by reversion to ventricular tachycardia. As no peripheral pulse could be felt external cardiac massage was begun and she was intubated and ventilated by hand with 100% oxygen. Internal jugular vein cut-down was performed. Central venous pressure was 2 cm above the sternal angle.

Diazepam, phenobarbitone, and phenytoin were given to control the convulsions, with good effect, and 20 mEq sodium bicarbonate and 10 mg lignocaine were injected intravenously. She remained in ventricular tachycardia and an infusion of lignocaine 0·5 mg/min. and glucagon 1 mg/hr was started.

Whenever massage was stopped no peripheral pulse could be felt, and the E.C.G. monitor continued to show ventricular tachycardia. While she was receiving external cardiac massage a stomach tube was passed and a washout performed. Moderate quantities of tablet fragments were recovered. A frusemide and mannitol diuresis was started at this stage.

A fine Teflon-coated, stainless-steel pacemaker wire was then inserted via the internal jugular vein cut-down in an attempt to overdrive and thus suppress the ventricular tachycardia. As the wire entered the right atrium there was a spontaneous return to sinus rhythm and a pulse could be felt peripherally. Cardiac massage was discontinued, having been performed continuously for two and a half hours. Manual ventilation was stopped and the patient was connected to a Bennett ventilator. Five hours later her conscious level had improved to the extent that she was actively "fighting" the ventilator. Ventilation was discontinued. She was extubated and given humidified oxygen by mask.

Though initially her condition was satisfactory she developed stridor with intercostal recession. This was suggestive of laryngeal oedema, probably due to the movement of the endotracheal tube during the period of cardiac massage. Later that evening she reverted to ventricular tachycardia, possibly due to the combined effects of hypoxia and an irritable myocardium, and required further cardiac massage and D.C. cardioversion before returning to sinus rhythm. She was reintubated and ventilated overnight under the control of tubocurarine and diazepam. She was also given dexamethasone, to alleviate laryngeal oedema, and ampicillin and cloxacillin.

Next day she was allowed to breathe spontaneously; she was extubated and put in a Croupette. She made an uneventful recovery and was discharged home lively and completely well four days after admission. Though she had no obvious mental deficit at the time of discharge follow-up will be necessary to confirm that she suffered no permanent cerebral damage.

Comment

This case is particularly important because it emphasizes the value of prolonged external cardiac massage in children who develop a critically low cardiac output from tricyclic poisoning or any other potentially reversible cause. We found that several attendants performing cardiac massage for about five minutes each were able to maintain an effective output for two and a half hours. Because of the immaturity of the child's skeleton this could be achieved without rib fracture or other serious complication apart from laryngeal oedema caused by movement of the endotracheal tube.

The dual effect of imipramine on the myocardium is demonstrated. This drug not only causes arrhythmias but also has a direct toxic effect on the myocardium, reducing the force of contraction (Steel *et al.*, 1967). These two effects can combine to produce a reduction of cardiac output incompatible with survival even in the absence of asystole or ventricular fibrillation.

The turning point in this child's recovery was the restoration of sinus rhythm, which must have been beneficial in two respects. Firstly, the return of co-ordinated atrioventricular activity restored the contribution of atrial systole to ventricular function (Kosowsky *et al.*, 1968). Secondly, the return of normal intraventricular conduction in the transmitted beats improved the power of ventricular contraction (Scherlag *et al.*, 1967).

We wish to thank Dr. D. A. Chamberlain for help in the preparation of this paper, and also Dr. M. E. Fielding and Dr. G. W. Hatcher for permission to report this case. In addition we thank the nursing staff of the intensive care unit, without whose care this child would not have survived.

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