Lesson of the Week

Tricyclic antidepressant poisoning and prolonged external cardiac massage during asystole

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Cardiac arrest occurs in a few cases of self-poisoning by a tricyclic antidepressant and is often extremely difficult to treat.[1] Standard methods of resuscitation are frequently useless in restoring a stable rhythm with adequate cardiac output. The primary role of external cardiac massage and assisted ventilation should not be forgotten in such cases. Prolonged external cardiac massage will buy time for the toxic effect on the myocardium to lessen, and primary resuscitation should not be given up when reversible signs such as asystole or fixed dilated pupils appear. Two cases of self-poisoning with imipramine illustrate this.

Cardiac arrest due to poisoning with a tricyclic antidepressant drug may be reversed by prolonged external cardiac massage

Case reports

Case 1—A 28-year-old man was admitted to the casualty department about one hour after ingesting an unknown quantity of imipramine tablets. He was agitated, with a sinus tachycardia of 120/min and blood pressure of 100/80 mm Hg. The pupils were moderately dilated, reacting to light, and both plantar responses were upgoing. Because he was rapidly losing consciousness and had a grand mal convulsion an endotracheal tube was passed and he was transferred to the intensive therapy unit. No gastric lavage was performed. The concentrations of urea and electrolytes were normal except for a low bicarbonate concentration of 15 mmol (mEq)/l, and arterial blood gas analysis confirmed a moderate acidosis: pH 7.11; P_{CO_2} 4·5 kPa (33·8 mm Hg); P_{O_2} 21·7 kPa (163 mm Hg); standard HCO_3^- 11 mmol (mEq)/l; base excess −19 mmol (mEq)/l. Despite intravenous 8·4% sodium bicarbonate the patient went into ventricular tachycardia, rate 150/min, with no discernible cardiac output. Assisted ventilation was continued manually and external cardiac massage started. Cardioversion at 50 and 100 joules produced asystole. Isoprenaline, intravenous calcium chloride, and intracardiac adrenaline had no effect. Transthoracic pacing also failed to achieve any ventricular response. Resuscitation was abandoned after 40 minutes, as all measures had failed to restore cardiac output.

Case 2—A 19-year-old girl was admitted to the casualty department 40 minutes after ingesting 1000-1250 mg of imipramine. She had no medical or psychiatric history. She had not ingested any other drugs. She was drowsy but talking. On examination her pulse was 150/min, blood pressure 170/100 mm Hg, and the pupils widely dilated. Gastric lavage was attempted but abandoned when she had a 30-second grand mal convulsion. She was transferred to the intensive therapy unit, where an endotracheal tube and stomach tube were passed. Gastric lavage was performed but there were no remnants of the tablets. She had a further grand mal fit, followed by a circulatory arrest. The electrocardiogram showed ventricular fibrillation, which rapidly changed to asystole (before DC shock). Cardiac output was maintained with external cardiac massage and assisted ventilation. Arterial blood gas concentrations were pH 6·9; P_{CO_2} 4·47 kPa (33·6 mm Hg); P_{O_2} 23·2 (174 mm Hg); HCO_3^- 6·2 mmol (mEq)/l; base excess −24 mmol (mEq)/l. The metabolic acidosis was corrected with 8·4% sodium bicarbonate: pH 7·32; P_{CO_2} 5·59 kPa (42 mm Hg); P_{O_2} 20·43 kPa (153·6 mm Hg); HCO_3^- 21·1 mmol (mEq)/l; base excess −4·2 mmol (mEq)/l. Urea and electrolyte concentrations were normal: Na^+ 145 mmol (mEq)/l; K^+ 3·9 mmol (mEq)/l; Cl 93 mmol (mEq)/l; HCO_3^- 16·0 mmol (mEq)/l; urea 5·9 mmol/l (35·5 mg/100 ml). Adrenaline, calcium chloride, and isoprenaline were given intravenously without benefit.

The patient remained asystolic for 90 minutes, while external cardiac massage was performed continuously. The electrical complexes and femoral pulses then returned spontaneously. For a further 90 minutes the electrocardiogram showed intermittent supraventricular and ventricular trachycardia at rates of 150-180/min. The cardiac output fluctuated and external cardiac massage was often required. Lignocaine, disopyramide, and prostigmine failed to control the ventricular tachycardia. Three hours after the cardiac arrest the patient's cardiac rhythm became stable with a sinus tachycardia of 150/min and blood pressure 70 mm Hg systolic. She was breathing spontaneously with paradoxical sternal movement. A chest radiograph showed gross pulmonary oedema but no rib fractures. The patient remained on the ventilator for a further 36 hours with resolution of the pulmonary oedema, and she was given ampicillin, gentamicin, and methylprednisolone. After extubation her recovery was complicated by fever (40°C), urinary retention lasting eight days, and paralytic ileus (six days). Cardiac enzyme activities 12 hours after cardiac arrest were creatinine phosphokinase 15 000 IU/l and serum aspartate transaminase 446 IU/l, gradually returning to normal over five days. Serial electrocardiographs showed right bundle-branch block and right axis deviation, which lasted for one week. Sinus tachycardia (> 110/min) persisted for eight days and widespread ST depression was evident throughout her stay in hospital.

She was discharged home two weeks after admission and two weeks later she was well, with a normal ECG. Despite some amnesia in hospital her memory had improved, though some impairment of short-term memory persisted.

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Discussion

These two cases highlight an extremely important aspect of managing patients who have taken an overdose of a tricyclic antidepressant. Both patients developed asystole shortly after admission to hospital, and in neither case was a cardiodepressant drug given. Despite being given inotropic drugs both patients remained asystolic, and resuscitation was abandoned in the first case after 40 minutes. The second case was equally unresponsive although external cardiac massage and assisted ventilation were continued for three hours, when cardiac rhythm was reestablished and cardiac output maintained.

The lesson to be learnt from these two cases is that asystole caused by a drug overdose does not carry the same prognosis as asystole secondary to acute myocardial infarction. Most patients who deliberately poison themselves have a healthy myocardium capable of returning to normal function despite prolonged external cardiac massage. In case 2 it must be presumed that sufficient metabolism and redistribution of imipramine took place during the three hours of external cardiac massage for the toxic effect on the myocardium to lessen and function return. Prolonged cardiac massage has also been successful in a child[2] and an adolescent[3] both with circulatory collapse after imipramine overdosage. Although our second patient was older, it was possible to maintain an adequate cardiac output by prolonged cardiac massage, as shown by her subsequent recovery.

External cardiac massage should be given as long as necessary; recovery after five hours of external cardiac massage has been reported.[3] All junior doctors dealing with medical admissions should be aware of the treatment of severe tricyclic self-poisoning[4] and remember that the ventricular tachycardia and asystole induced by these drugs have the same clinical consequences. Because most patients with tricyclic self-poisoning have a healthy myocardium before the event, the "intensive supportive treatment" should include continuing external cardiac massage and assisted ventilation for several hours rather than accepting the apparent hopelessness highlighted in case 1. This may help save the lives of many young adults who are otherwise given up for dead.

We thank Dr S J Pearce for allowing us to report on case 2.

References

1 Serafimovski N, Thorball N, Amussen I, Lunding M. Tricyclic antidepressive poisoning with special reference to cardiac complications. *Acta Anaesthesiol Scand* 1975; suppl 57:55-63.
2 Southall DP, Kilpatrick SM. Imipramine poisoning: survival of a child after prolonged cardiac massage. *Br Med J* 1974;iv:508.
3 Ramsay ID. Survival after imipramine poisoning. *Lancet* 1967;ii:1308-9.
4 Crome P. Tricyclic antidepressant poisoning. *Hospital Update* 1980;**6**: 369-74.

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Alcohol and Alcoholism

Alcohol and work: a promising approach

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Worried by the increasing incidence of alcohol problems, the present Conservative Government is enthusiastic about the possibilities of responding to the problems in the workplace. Obviously, a policy that calls on employees and unions to act on their own initiative fits with Conservative philosophy and is preferred by them to intervention by central government in raising the tax on alcohol. But there are sounder reasons than mere philosophical compatibility for encouraging attempts to deal with alcohol problems at work.

As with so many other issues Britain has lagged way behind with this one. Programmes to deal with alcohol problems at work have existed in the United States since 1942,[1] and France,[2] Norway,[3] and Australia[4] have all taken initiatives that have only rarely been taken in Britain. But now, almost 40 years after the Americans, the Government has produced a pamphlet, *The Problem Drinker at Work*,[5] that might prompt employers and unions to action. The pamphlet, like the Government "think tank" report before it,[6] sets out the solid reasons why a response at work is so desirable. Firstly, most people with alcohol problems are employed, and those problems often first become apparent at work. Secondly, certain occupations are known to be associated with an increased prevalence of alcohol problems.[7] Thirdly, the costs to employers incurred through absenteeism, poor performance, lateness, sickness, and accidents are considerable.[8] Fourthly, people whose livelihood is at stake are likely to be strongly motivated to overcome their problems. For all these reasons, much can be expected from responding to alcohol problems at work, and this article will look more closely at the association between work and alcohol problems and at the various strategies available for a response.

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Occupation and alcohol problems

Most doctors are aware of the links between occupation and alcohol problems, not least because doctors themselves are one group with a high prevalence of such problems.[9 10] The evidence for these links comes from several sources, none of them entirely satisfactory. Table I shows mortality from cirrhosis of the liver in various occupations: publicans have a rate 15 times higher than the average and doctors a rate three times higher. We know that today most cirrhosis results from alcohol abuse, and so this is strong circumstantial evidence of how various occupations drink, but we must remember that this is information about dead alcoholics; live ones may be different.