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# SHORT REPORTS

# Addiction to tranylcypromine

Psychiatrists recognise that patients taking tranylcypromine may develop psychological dependence and often have difficulty weaning them off even small doses of the drug. Some patients become addicted with a pronounced tendency to increase the dose. Neither Martindale<sup>1</sup> nor Goodman and Gilman<sup>2</sup> note the addictive properties of tranylcypromine. Since 1965 three cases of addiction have been reported.3-5 We describe four others.

## Case reports

Case 1-A 33-year-old woman was admitted after prolonged abuse of Parstelin (tranylcypromine 10 mg, trifluoperazine 1 mg), culminating in an overdose of 40 tablets. Though initially lucid, 24 hours later she became hallucinated, confused, agitated, and aggressive. Over the next eight days she returned to normal. She had first suffered from depression at the age of 15, and had since had continuous psychiatric treatment. Personality disorder and depression were diagnosed. At the age of 25 Parstelin was started and before long she was taking 30 tablets daily. On at least two occasions she developed severe thrombocytopenic purpura due to the Parstelin abuse. All attempts at withdrawal from the drug were only temporarily successful.

Case 2-A 34-year-old schoolteacher was admitted complaining of addiction to Parstelin. He was taking 15 tablets daily, but had had some difficulty obtaining the drug, getting prescriptions from many different doctors. He had a history of three previous admissions for anxiety, depression, and alcoholism. On abstaining from Parstelin he suffered headaches, diarrhoea, and weakness and felt unable to cope with work. He also abused chlordiazepoxide, taking about 100 mg daily. While abusing Parstelin he did not drink alcohol. In hospital, on withdrawal, he complained of anxiety and depression. After discharge he immediately resumed Parstelin, taking up to 20 tablets daily.

Case 3—A 65-year-old man was admitted taking up to 30 Parstelin tablets daily. He had been taking Parstelin and chlordiazepoxide for 10 years and obtained prescriptions from many doctors. He stated that he felt well only when taking Parstelin. He had had treatment for alcoholism but since abusing Parstelin he had abstained from alcohol, stopped smoking, and worked effectively. After withdrawal from Parstelin and chlordiazepoxide he became tense, pacing around the ward all day. He was discharged drug free but soon resumed the Parstelin.

Case 4-A 39-year-old shopkeeper was admitted for depression and Parstelin abuse. He had obtained Parstelin and diazepam from numerous doctors and pharmacists. He took up to 20 Parstelin and 10 diazepam tablets daily, and without them felt unable to work. He was a shy, conscientious, obsessional man and had been a very heavy drinker. After admission and withdrawal from Parstelin he became restless and anxious and complained of generalised aches for 10 days, despite treatment with chlordiazepoxide and chlorpromazine. He showed no other withdrawal symptoms.

## Comment

Addiction to tranylcypromine is not widely recognised and each of our patients received many prescriptions with relative ease. The addictive properties of tranyleypromine are not surprising in view of its close structural relation to amphetamine<sup>2</sup> and its powerful effect in suppressing rapid-eye-movement sleep.4 All our patients showed a notably increased tolerance to tranylcypromine and three also showed abuse and increased tolerance of benzodiazepines. On abrupt withdrawal of Parstelin our first patient became hallucinated and confused. This was probably a withdrawal state, as in the case of Ben-Arie and George,3 but intoxication could not be completely ruled out.

We wish to emphasise that tranylcypromine should be used with great care in dependence-prone patients, and doctors should be wary of requests for prescriptions of the drug. Withdrawal from high dosage of tranylcypromine should be gradual because of the danger of withdrawal psychosis.

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# Damage to the tricuspid valve with a Swann-Ganz catheter

We report a case of damage to the tricuspid valve during preoperative insertion of a Swann-Ganz catheter. The lesion was discovered by visual inspection during the operation, which suggests that the incidence of undetected damage and its complications<sup>1-3</sup> might be greater than previously thought.

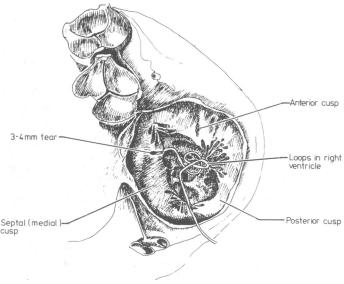
## Case report

A 59-year-old woman with a history of rheumatic heart disease was admitted for elective aortic and mitral valve replacement and insertion of a tricuspid ring. Two months previously cardiac catheterisation had shown mixed mitral valve disease, predominantly stenosis and aortic insufficiency. The murmur of tricuspid incompetence could also be heard. Chest x-ray examination showed an enlarged left ventricle, right ventricle, and left

A pulmonary artery catheter was inserted for measurement of filling pressures and thermodilution cardiac outputs during the operation. A triple-lumen 7 FG flow-directed Swann-Ganz catheter (Instrumentation Laboratories) was inserted via the right cephalic vein in the antecubital fossa using a Cordis introducer. A cut-down had previously been performed on the right basilic vein, and the catheter was inserted at the right cephalic vein after failure to enter the chest using the left cephalic vein.

The catheter entered the chest easily, and as soon as it was in the superior vena cava the balloon was inflated and the pressure at the catheter tip displayed on a Hewlett Packard monitoring system. The catheter negotiated the tricuspid valve easily to give a tracing of right ventricular pressure. Entering the pulmonary artery was difficult, and to achieve this position the balloon was deflated and the catheter pulled back into the right atrium and allowed to float back with the balloon inflated again. The catheter was eventually placed successfully in the pulmonary artery, though not in the wedge position, by the conventional method. Induction of anaesthesia was uneventful, and cardiopulmonary bypass was started after routine cannulation of the aorta, superior vena cava, and inferior vena cava.

After replacement of both the mitral and aortic valves with Bjork-Shiley prostheses the right atrium was opened for inspection of the tricuspid valve, which was suspected of being incompetent. A small tear of about 3-4 mm in the septal cusp of the tricuspid valve was obvious, and one of the associated chordae tendineae was ruptured. The catheter was curled up in the right ventricle with the tip lying in the pulmonary artery (figure). The damaged cusp and chorda tendineae were repaired with 5-0 Prolene. An attempt to insert a Carpentier ring 34 FG was abandoned because it did not fit well, and eventually surgical repair of the ring related to the anterior and posterior cups was performed without a prosthesis. Further progress was uneventful, and she was discharged from hospital without sequelae.



Tricuspid valve viewed from right atrium.

## Comment

Although distortion of the tricuspid valve causing incompetence is a complication of pacemaker insertion<sup>5</sup> and catheterisation of the heart, tears to the cusps of the tricuspid valve have not been reported. Elliott et al1 reported a case of multiple perforations in the leaflets of the pulmonary valve in a patient in whom three consecutive catheters were in place for 27 days. They suggested that the perforations might be due to the to-and-fro motion of the catheter. Smith et al2 reported a rupture of a chorda tendineae of the anterior cusp of a tricuspid valve, but in their case the catheter had been pulled back several times with the balloon inflated in attempts to enter the pulmonary artery. Their patient had pulmonary artery hypertension, low cardiac output, and biventricular failure with a large right ventricle. Several workers have reported difficulties in entering the pulmonary artery with this combination, presumably because the catheter tends to curl up in the ventricular cavity owing to the large space and reduced forward flow. In our patient, who also exhibited these four features, the catheter was found curled up in the right ventricle but with the tip in the pulmonary artery. As the catheter was never withdrawn with the tip inflated possibly the loops of catheter had become caught between the chordae tendineae and the valve cusps, so that when it was pulled back the chorda was ruptured and the tear in the septal cusp produced by traction from below the valve. Cannulation of the superior vena cava might also have encouraged to-and-fro motion of the catheter.1

We thank Professor H Huysmans for permission to report this case, and Miss J B Nichols for the illustration.

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# Ventricular fibrillation as a complication of salt-water immersion

Ventricular fibrillation is an extremely unusual complication of immersion in either fresh- or salt-water drowning¹ but has been reported as a complication during resuscitative procedures in profoundly hypothermic victims.² It is sometimes seen in profound hypothermia resulting from cold exposure in association with alcohol or barbiturate intoxication. In victims of immersion, death from drowning is believed to supervene before the cardiac temperature has been lowered below the critical fibrillatory threshold. We report on two patients with moderate hypothermia in whom ventricular fibrillation occurred after relatively short periods of immersion in salt water.

## Case reports

Case 1—An 11-year-old girl had been totally immersed in salt water at 15°C for at least six minutes. Combined external cardiac compression and expired air resuscitation were applied for 40 minutes until her arrival in hospital. During that time no pulse or spontaneous breathing developed. An electrocardiographic monitor showed coarse ventricular fibrillation, and a DC shock of 200 joules was given resulting in asystole. Ventricular tachycardia was initiated after intravenous injection of isoprenaline, and a further DC shock of 100 joules converted her to sinus rhythm. Rectal temperature on admission was 32.5°C. Blood-gas measurements (temperature corrected) were as follows: arterial oxygen pressure 18·1 kPa, carbon dioxide pressure 4·0 kPa, pH 7·04. She began breathing spontaneously 14 hours after admission and was extubated. She made a good recovery and was discharged home three days later.

Case 2—A 13-year-old boy had been immersed in salt water at 15°C for at least 20 minutes before he was pulled into a lifeboat, having been found floating face down. Expired air resuscitation was given in the boat. On admission to hospital 40 minutes later an electrocardiogram showed fine ventricular fibrillation, which failed to convert with a DC shock. Rectal temperature was 30·2°C, and blood-gas measurements were arterial oxygen pressure 13·6 kPa, carbon dioxide pressure 8·0 kPa, and pH 6·7. Despite attempts at rewarming using first peritoneal lavage and then partial bypass, ventricular fibrillation could not be terminated.

## Comment

Experimental work supported by clinical experience has shown that aspiration of a volume of water sufficient to produce ventricular fibrillation from hyperkalaemia due to haemolysis is improbable.<sup>3</sup> Ventricular fibrillation is a well-recognised complication of severe hypothermia (core temperature less than 30°C), especially during resuscitative manoeuvres.<sup>4</sup> In deaths from immersion, however, it is believed that cardiac arrest from hypoxia usually supervenes long before the core temperature of the victim has fallen to a value at which ventricular fibrillation might be expected to occur spontaneously, except when the victim is wearing a lifejacket.<sup>5</sup>

Our cases suggest, however, that some mechanism akin to the diving response maintains cardiac activity until such time as severe cooling has occurred. The total oxygen stores of man are sufficient to satisfy resting metabolic demands for only about four minutes so