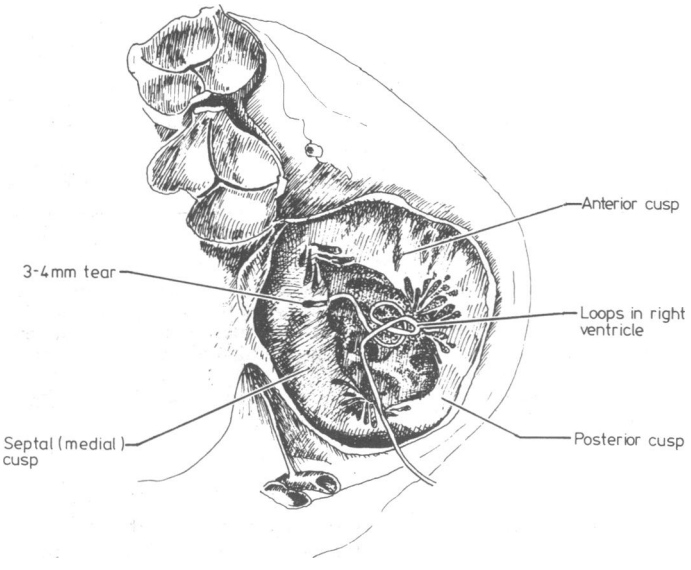
displayed on a Hewlett Packard monitoring system. The catheter negotiated the tricuspid valve easily to give a tracing of right ventricular pressure. Entering the pulmonary artery was difficult, and to achieve this position the balloon was deflated and the catheter pulled back into the right atrium and 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.[4] 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[5] and catheterisation of the heart, tears to the cusps of the tricuspid valve have not been reported. Elliott *et al*[1] 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 al*[2] 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 drowning[1] 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.[3] Ventricular fibrillation is a well-recognised complication of severe hypothermia (core temperature less than 30°C), especially during resuscitative manoeuvres.[4] 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.[5]

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

it is difficult to explain how successful resuscitation is possible after 40 minutes' submersion.[2] The reduced oxygen requirement in profound hypothermia has been suggested by some authors as the mechanism by which tissue viability is maintained in these circumstances,[2] but this alone is an incomplete explanation, as for the submerged body to cool to a degree at which oxygen requirements are sufficiently reduced, heat transfer by mass flow—that is, circulation—must be present (a dead body cools relatively slowly). The degree of cooling encountered in our cases can be explained only by the fact that circulation was maintained for some or all of the period of submersion. The persistence of cardiac activity during such a prolonged period of apnoea supports the hypothesis that some protective mechanism may be present. We do not know whether ventricular fibrillation was present before resuscitative efforts were started and must concede that these efforts alone may have initiated the arrhythmia.

Our cases show yet again that successful resuscitation is possible after long periods of immersion and emphasise the importance of performing electrocardiography early in the immersion incident, preferably at the accident site, before death is pronounced.

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Effect of nifedipine on bronchomotor tone and histamine reactivity in asthma

Secretion of chemical mediators from mast cells and contraction of bronchial smooth muscle are two major components in the pathogenesis of asthma. Both processes depend on the transmembrane passage of calcium ions, and drugs that inhibit this might therefore be expected to be of value in asthma. Cinnarizine, an antagonist to calcium transport, has been shown to exert a beneficial effect in patients with chronic asthma,[1] and verapamil, another calcium antagonist, prevents potassium- and serotonin-induced contraction of canine trachealis muscle.[2] Nifedipine is a potent inhibitor of transmembrane calcium ion flux, and results of a recent study suggest that it prevents exercise-induced asthma.[3] We therefore investigated the effect of nifedipine on resting bronchomotor tone and on histamine reactivity in patients with asthma.

Patients, methods, and results

We studied 10 patients, aged 25-60 years, with chronic stable asthma. The protocol was approved by the hospital research ethics committee, and all subjects gave informed consent.

Each subject received either nifedipine 20 mg or placebo in a randomised double-blind fashion on separate days and was instructed to bite the capsules and keep the fluid in the mouth as long as possible. Both active and placebo capsules had the same appearance and contained liquid with an identical peppermint taste.

Using a dry spirometer, we measured forced expiratory volume in one second (FEV_1), vital capacity (VC), and maximal mid-expiratory flow (MMEF) before and at 15, 30, 45, and 60 minutes after the capsules were taken. Blood pressure and heart rate were also recorded. After the measurements at 60 minutes histamine reactivity was assessed by giving the subjects serial dilutions of histamine acid phosphate solution to inhale via a Wright nebuliser. The concentration of histamine producing a 20% fall in FEV_1 (PC_{20}) was calculated. Each subject received 200 μg inhaled salbutamol at the end of the study.

The studies were performed in an air-conditioned laboratory under constant environmental conditions. Both tests were performed at the same time of day in each subject, and the subjects had not taken any medication for at least eight hours beforehand.

No subject complained of any side effects, and there were no significant changes in blood pressure or heart rate. The lung function results are summarised in the table. There was no significant difference in baseline spirometry between the days when nifedipine and placebo were taken. Although the increases in mean FEV_1 and MMEF were both greater with nifedipine than placebo, an analysis of variance failed to show any significant difference between the two. PC_{20} was significantly greater ($p<0.05$, paired t test) after nifedipine (0·42 ± 0·14 mg histamine/ml) than placebo (0·15 ± 0·03 mg histamine/ml). All subjects showed a good bronchodilator response to inhaled salbutamol at the end of the study.

Mean ± SEM changes in tests of lung function during treatment with nifedipine and placebo

	Control	Minutes after taking capsules			
		15	30	45	60
	Forced expiratory volume in one second (l)				
Active	1·63 ± 0·21	1·81 ± 0·22	1·92 ± 0·21	1·98 ± 0·22	1·93 ± 0·23
Placebo	1·61 ± 0·16	1·75 ± 0·16	1·78 ± 0·17	1·81 ± 0·17	1·86 ± 0·17
	Vital capacity (l)				
Active	3·04 ± 0·24	3·29 ± 0·22	3·38 ± 0·21	3·44 ± 0·20	3·37 ± 0·21
Placebo	2·97 ± 0·19	3·17 ± 0·19	3·24 ± 0·17	3·37 ± 0·21	3·29 ± 0·23
	Maximal mid-expiratory flow (l/s)				
Active	0·86 ± 0·15	1·01 ± 0·17	1·05 ± 0·17	1·17 ± 0·21	1·11 ± 0·19
Placebo	0·82 ± 0·12	0·88 ± 0·12	0·98 ± 0·16	0·95 ± 0·12	1·02 ± 0·15

Comment

Although we were unable to show any significant bronchodilator activity for nifedipine, we showed that this drug provides a significant protective effect against histamine-induced bronchoconstriction. This suggests that it may have an effect on the contractility of bronchial smooth muscle, but the effect is too small to make oral nifedipine therapeutically useful in asthma. Our results clearly show, however, that nifedipine may safely be given to patients with asthma. Its clinical effectiveness in the treatment of angina is well documented, and there is some evidence that it produces an improvement in FEV_1 when given to patients with angina and labile airways obstruction.[4] Beta-adrenoreceptor antagonists have been the mainstay of anti-anginal treatment in recent years, but the use of such drugs in patients with asthma may be extremely hazardous.[5] Patients with angina and coexistent asthma should be treated with nifedipine for their angina in preference to beta-blockers.

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