caused by wax or condensation may be easily identified as the tubing is transparent.

Residents who were considered to have a hearing loss that affected them socially were followed up to see whether matters had improved after their hearing problems had been dealt with by IC or their general practitioners or at the audiology clinic.

The table shows the results. Half of the hearing aids already fitted were not functioning, nine because of flat batteries and 16 because of blockage by wax. Although

Findings in 304 residents whose hearing was assessed informally

Finding	No of residents	
Already fitted with an aid		49
Aid non-functioning	25	
Aid functioning	24	
Dementia not improved by amplification		11
Referred to audiology-hearing aid clinic for first time		42
Received hearing aid	27	
Died before seen in clinic	6	
Hearing improved after wax removal	2	
Required no action	4	
Admitted to hospital for reasons unconnected with ENT	3	
Refused offer of referral to audiology department		17
Known to have hearing loss but uncooperative		13
Use of personal amplifier recommended		7*
Found to have severely disabling tinnitus		5
Required wax removal		79
Referred to general practitioner but no action taken		15
Required no action		132

* Patients too ill to travel to hospital.
ENT = Ear, nose, and throat.

eight of the 24 residents fitted with functioning aids required no action, 10 needed a new mould or aid; the hearing aids of the remaining six needed minor attention such as new tubing. Of the 42 residents referred to an audiology clinic for the first time, five were confused and their mental state noticeably improved with the use of personal amplifiers. Disabling tinnitus was found in five residents, who were referred for counselling, relaxation training therapy, and trials with a masker (maskers look similar to hearing aids that fit behind the ear and introduce a band of sound into the ear which may be more tolerable than the person's tinnitus). In 11 patients the coexistence of deafness and dementia was probably coincidental. Overall we found that 154 of the 304 residents had a loss of hearing that affected them socially.

Comment

Fisch suggested that screening all elderly people makes sense only when care is available for all those who need it.[1] We knew that we could cater for all the residents' hearing needs in this small scale study. In an audiometric study of 365 men and women aged 70 and over 60% were found to be deaf.[3] We found that about half of the residents (51%) had a hearing loss that affected them socially. Nassar found that 23% of patients admitted to a geriatric ward needed to have wax removed[4]; our results were similar (26%), and, in addition, 16 residents had hearing aids that were blocked with wax.

Our results in the five residents with confusion suggest that hearing should be assessed in confused elderly people. Ideally, an audiological technician should be available to carry out domiciliary work with elderly people, but resources are limited. We suggest that staff who care for elderly people should be trained to identify hearing difficulties and malfunctioning aids and to change batteries and blocked tubing. Every residential home should keep spare batteries as the life span of a battery may be as short as three days. As most people with hearing loss do not acknowledge their disability staff should be aware that confusion, irritableness, delayed responses, and changes in social behaviour may be signs of hearing loss that is affecting them socially.

The waiting list for our audiology clinic is short, about four weeks. Nevertheless, we recommend that the time between the initial assessment and audiological examination should be shortened to speed up the provision of hearing aids.

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Captopril for correcting diuretic induced hypotension in pulmonary oedema after scorpion sting

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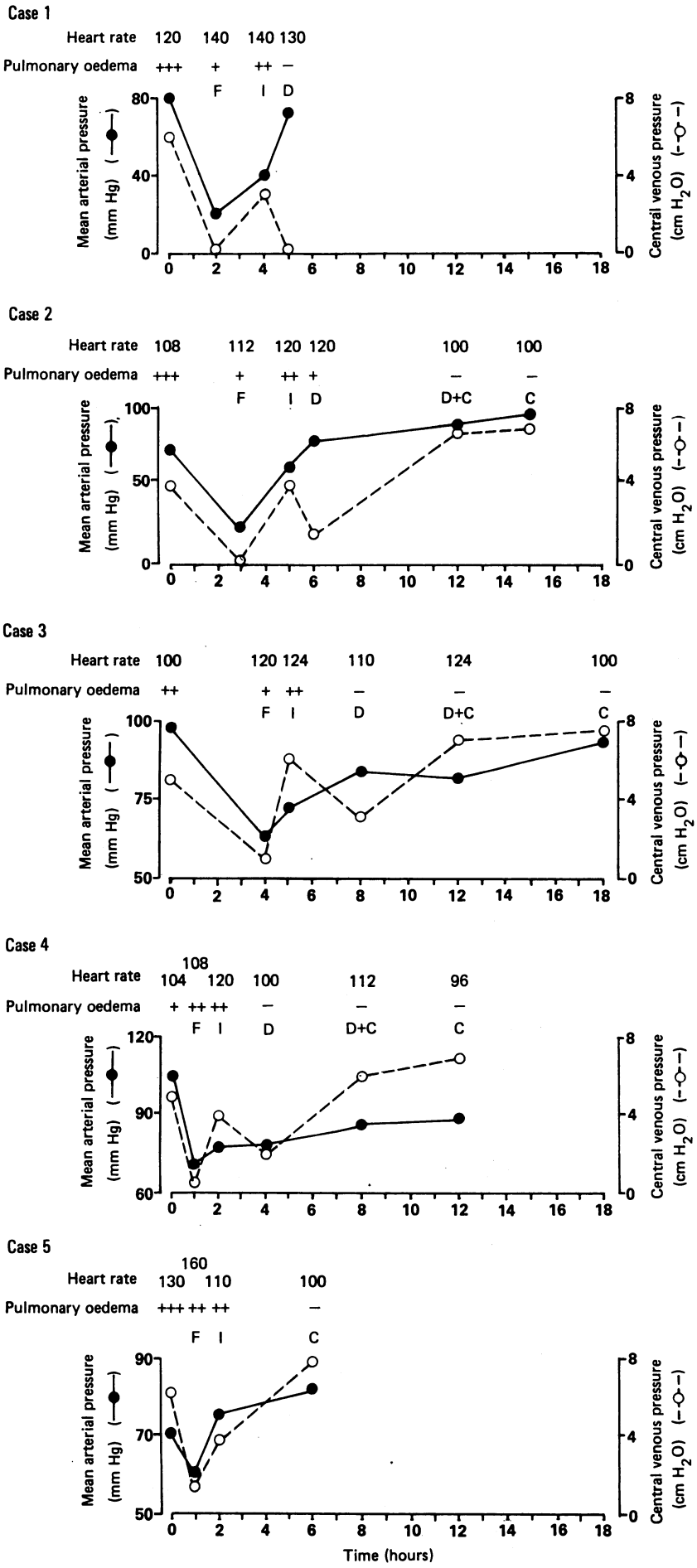
Stings from the red scorpion (*Buthus tamulus*) are common in rural western India.[1] The venom causes initial transient cholinergic stimulation followed by sustained adrenergic hyperactivity.[2] Excess circulating catecholamines released from the adrenergic neurons and adrenal medulla cause myocardial damage similar to that seen in phaeochromocytoma.[3] Hypoxia resulting from an increased myocardial requirement for oxygen is thought to be responsible. Myocardial damage and catecholamine induced increase in afterload cause pulmonary oedema after severe envenomation.[3] Conventional treatment of cardiac failure after scorpion sting consists of digoxin and diuretics. We describe five patients with severe envenomation who presented with pulmonary oedema and developed a series of haemodynamic problems after parenteral diuretic treatment, for which they were given captopril.

Patients and results

All five patients were young (12-25 years) previously healthy residents of rural western India who were admitted to the medical intensive care unit four to 12 hours after envenomation. The figure summarises their clinical course and their response to therapeutic interventions. All patients initially had pulmonary oedema with a normal blood pressure, but, while respiratory distress and rales decreased after intravenous frusemide administration, hypotension and peripheral circulatory collapse occurred one to four hours later. Central venous pressures were low, and attempts to correct these with crystalloid infusion worsened the pulmonary oedema. Inotropic drugs (dopamine and dobutamine) produced improvement in blood pressure and pulmonary oedema, but the reduced cardiac preload persisted.

The first patient continued to receive dopamine and



Changes in heart rate (beats/min), mean arterial pressure, central venous pressure, and pulmonary oedema in five patients with severe scorpion envenomation after admission (time 0). F=intravenous frusemide, I=infusion of crystalloids, D=addition of dopamine or dobutamine, C+D=addition of captopril, C=captopril alone. Pulmonary oedema was graded as: – absent, + few basal rales, ++ extensive rales with tachypnoea, +++ blood tinged frothy sputum with central cyanosis

improved steadily but developed sudden hypotension 36 hours after admission. Her condition worsened in spite of increasing doses of dopamine. Pulmonary oedema recurred as a terminal event. In cases 2, 3, and 4 pulmonary oedema worsened on fluid infusion at subnormal central venous pressures, and inotropic drugs were continued. The patients then received 12·5 mg of captopril orally and fluids were infused simultaneously. With this peripheral cyanosis disappeared, urine output increased, heart rate decreased, and the low central venous pressure was corrected with minimal change in blood pressure, and inotropic drugs were discontinued. The fifth patient had a similar initial course but was given 12·5 mg captopril along with fluid infusions after frusemide produced hypovolaemic shock; the pulmonary oedema resolved, and he did not need dopamine.

Captopril was discontinued after three to four days in all four patients. No adverse effects were seen.

Comment

All our patients followed a characteristic course, with resolution of pulmonary oedema but development of shock with a low central venous pressure after intravenous diuretics. Fluid infusion worsened the pulmonary oedema before central venous pressure could be corrected. Low central venous pressure indicates subnormal right ventricular preload and may not reflect left ventricular diastolic pressures in acute left ventricular dysfunction. Since pulmonary oedema after scorpion sting occurs only when the left ventricular end diastolic pressure is high,[3] we concluded that left ventricular preload was raised in our patients though central venous pressure was low (pulmonary wedge pressure was not measured). Inotropic drugs were tried and they did raise the blood pressure. Both dobutamine and dopamine act by stimulating cardiac β adrenergic receptors, and one could debate their value in patients with already increased circulating catecholamines. These drugs might actually add to the endogenous catecholamine induced cardiac damage. The first patient, who did not receive captopril, developed sudden hypotension and died after receiving dopamine for 24 hours.

Reduction in afterload enhances performance of the failing ventricle by decreasing myocardial wall tension and oxygen requirements. Prazosin, an α blocker, has been tried to this end in patients with scorpion stings with some success.[1] We preferred captopril because, in addition to its vasodilatory action, this angiotensin converting enzyme inhibitor also inhibits release of catecholamines from peripheral nerve terminals and reduces central sympathetic outflow.[4] Recent studies have shown that it may also prevent myocardial damage by scavenging free radicals.[5]

Diuretics should be used with caution in patients with pulmonary oedema due to scorpion sting. If hypotension does develop oral captopril with simultaneous fluid infusion corrects this readily.

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