one-third of the normal calcium. This stimulation caused inhibition of the pendular movements to about half their previous size ; they remained at this level while the stimulation continued, and while the drum was stopped for five minutes. At the arrow, calcium was added to the bath to bring the concentration up to normal, and the inhibition at once increased to become almost total.

Action of Nicotine.—If the sympathetic impulse first releases acetylcholine and this in turn releases noradrenaline as Burn and Rand have suggested, then the release of noradrenaline by acetylcholine should be affected by calcium in the same way as the release by sympathetic stimulation. This has not yet been fully studied, but it has been shown that nicotine, in the presence of hyoscine, causes an inhibition of the pendular movements of the ileum which increases in proportion to the concentration of calcium. It is therefore reasonably certain that acetylcholine will act in the same way, and indeed some evidence that it does so has already been obtained.

Effect of Magnesium.—Douglas and Rubin found that the addition of magnesium to Locke's solution used to perfuse the adrenal gland reduced the effect of raising the calcium. We likewise observed that the effect of additional calcium in increasing the inhibitory response to stimulation was antagonized by magnesium.

Role of Calcium

There are two explanations of the events which lead to the release of noradrenaline at the end of the post-ganglionic fibre. The first is that the impulse, on reaching the end of the fibre, itself creates a change in the permeability of the fibre membrane to calcium, so that calcium enters the fibre ; the extent of calcium entry will depend on the external concentration. When the calcium has entered, it then liberates noradrenaline from the granules. The second explanation is that the nerve impulse first releases acetylcholine and it is this acetylcholine which causes the change in permeability of the fibre membrane to calcium, so that calcium entry can occur.

The significance of the observations is that they indicate that the effect of sympathetic stimulation depends on the concentration of calcium ions. Thus a low concentration of calcium ions in the blood may perhaps be responsible for a low arterial tone, and intravenous infusion of a substance which removes calcium ions, such as the citrate present in blood used for transfusion, may, if given rapidly, cause a fall of blood-pressure. Similarly a high concentration of calcium may be responsible for a high arterial tone.

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Medical Memoranda

Pressure Palsy of the Accessory Nerve

Brit. med. J., 1964, **1**, 1483–1484

The following report concerns a patient who sustained an injury to the accessory nerve as a result of an unsuccessful attempt at suicide by hanging.

Case Report

A shoe-maker aged 54 who had tried to kill himself by hanging was admitted to hospital. He had been alone for 10 minutes when his wife heard his strangled breathing and rescued him. He was bleeding from the nose, pharynx, and both ears, and there were superficial haemorrhages of the subconjunctivae and skin of the head and neck above the rope burn (Fig. 1). He remained confused for about 10 hours.

There was nothing relevant in the family history. Though he was an over-protected and sickly child, his subsequent life and personality were stable. He was a moderate drinker and his past illnesses were not relevant.

Arterial hypertension was first noted when he was aged 33, and, though he had no symptoms, treatment with reserpine, 0.25 mg. thrice daily, was begun three-and-a-half months before admission. After two weeks his blood pressure was within normal limits and the dose was reduced to 0.25 mg. daily. Four weeks later he was clearly depressed, had lost weight, was sleeping poorly, and was anxious and retarded.

Physical examination after admission revealed no further abnormality. His blood-pressure was 165/105 mm. Hg, a routine urine examination was normal, the blood Wassermann reaction negative, and radiographs of the chest and cervical spine showed no lesion. His subsequent progress did not indicate intellectual deterioration or change in personality. He had a retrograde amnesia of a few minutes' duration and could not recall events of the 10 hours of clouded consciousness. The reserpine was suspended, he was given imipramine, and had fully recovered from the depressive state eight weeks after admission.

Three weeks after admission, shortly after beginning physical work, he complained of pain in his left shoulder. On examination he complained of pain at about 70° active abduction of his left arm, and could not abduct his arm further. There was asymmetry of the shoulders due to wasting of the left trapezius and lowering of the left shoulder. The body of the scapula was rotated slightly downward and outward. There was moderate weakness of elevation of the left shoulder, but no observable difference in the power of both sterno-mastoids or any other abnormality on full neurological examination.

Electromyography carried out at three-and-a-half weeks and 12 weeks after admission recorded abnormalities that were confined to the left trapezius. The muscle rapidly recovered in bulk so that by the tenth week there was no appreciable difference between the two sides, and no difference in power. The rapidity of recovery, with all areas of the muscle recovering together, indicated a first-degree injury (Sunderland, 1951) of the accessory nerve.

Discussion

Depression is not uncommon as a complication of medication with reserpine for hypertension (Jensen, 1959 ; Quetsch, Achor, Litin, and Faucett, 1959). The particular interest in this case is the unique paresis of the trapezius. The pain that drew attention to the lesion characteristically occurred when the patient began to do physical work (Hanford, 1933 ; Norden, 1946 ; Mead, 1952 ; Woodhall, 1952).

The trapezius is supplied by the spinal root of the accessory nerve, which receives communications from the second and third cervical nerves. Branches of the third and fourth cervical nerves join the accessory nerve to form a plexus on the deep surface of the muscle (*Gray's Anatomy*) and supply motor fibres to the lower part of the muscle, where their distribution

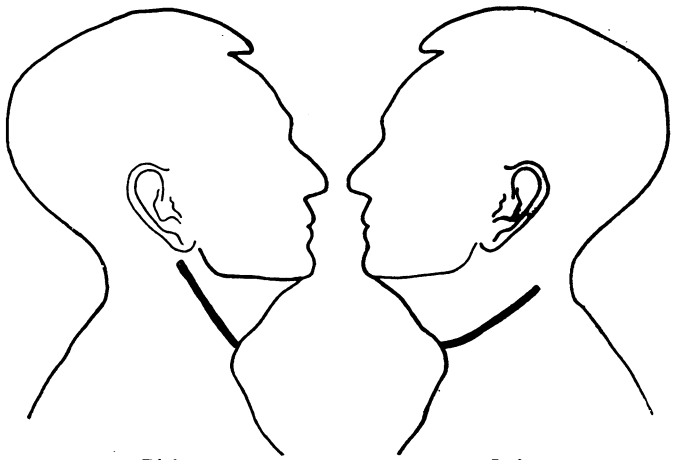

FIG. 1.—Position of the rope burns.

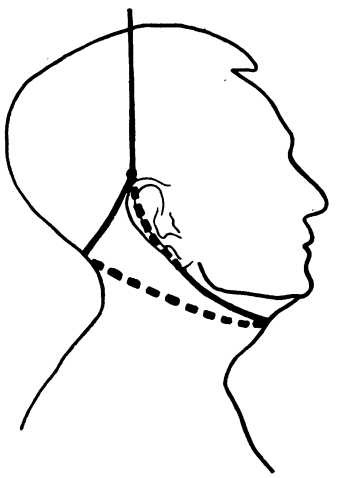

FIG. 2.—Reconstruction of the position of the noose.

is subject to considerable variation (Coleman and Walker, 1950 ; McKenzie and Alexander, 1950 ; Woodhall, 1952). The fact that other muscles were not involved indicated a lesion restricted to the accessory nerve distal to its branches to the sterno-mastoid, in all probability in the posterior triangle where the nerve is relatively superficial and more readily subject to trauma (Hanford, 1933 ; Wulff, 1941 ; Mead, 1952 ; Woodhall, 1952). Though traumatic lesions due to surgery at this site are not uncommon, pressure palsy is rare.

A reconstruction of the probable position into which the noose was forced (Fig. 2) indicates why it pressed on the left accessory nerve, but not on the right.

The unusual circumstances of this case make it possible to speculate about the mechanisms that produced the lesion. The indirect effect of pressure through localized ischaemia of the nerve may be discounted because the maximum period of compression was only 10 minutes and probably less. In experimental studies of pressure palsy both in man and cat, to produce motor paralysis the pressure had to last for at least 25 minutes (Lewis, Pickering, and Rothschild, 1931 ; Bentley and Schlapp, 1943a), and generally the clinical examples of tourniquet paralysis have been the result of much longer periods of pressure (Barlow and Pochin, 1948 ; Richards, 1954). Traction may be dismissed, as the direction of force would have tended to shorten rather than pull on the nerve. Evidence advanced by Denny-Brown and Brenner (1944) and Bentley and Schlapp (1943b) would seem to prove that pressure directly damages the nerve fibres only in circumstances where there is deformation and displacement of the nerve. The noose would have produced these mechanical effects, and consequently the lesion of this patient.

I wish to thank Dr. J. C. Cummins, Director-General of Public Health and State Psychiatric Services, for permission to publish this case, and Professor R. W. Gilliatt, Professor L. G. Kiloh, and Dr. G. Preswick for their advice.

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