the drops of which are already evaporating and are cold on reaching the skin. (Hedley No. 13 pack for general anaesthetics is the most suitable of those commercially available.) It follows that the nozzle should be held far enough away from the skin to allow the fluid to break up into cold droplets and not so far away as to produce only an attenuated blast of cold gas. With this pack 12 to 18 in. (30 to 45 cm.) is found to be a suitable distance. If these rather simple directions are borne in mind the rather elaborate technique recommended by Travell will be found to be unnecessary. The length of time the spray should be continued varies a little with the severity of the pain. The spray should not be so prolonged that skin-freezing occurs, and should be stopped at the earliest suggestion of freezing. To continue longer will produce no effect on anaesthetized skin, in addition to which a cold burn may result. Some of my colleagues find several repeated bouts of five seconds produce the required result. To obtain the mass barrage required, all the skin of the appropriate dermatome should be sprayed. In renal colic, lumbago, and dysmenorrhoea, to keep up the attack, the spray can be continued for 15 to 30 seconds, using about one-half to three-quarters of the bottle, before any sign of freezing appears.

Treatment by the spray is simple; the technique can be easily learnt and carried out by the general practitioner in the house or by co-operative lay persons. Many local general practitioners are using it with gratifying results. To give the ethyl chloride bottle to the patient for personal use in the home is not, however, without danger. Ethyl chloride is flammable, toxic, and anaesthetic. To get over these disadvantages, Dr. Travell and, later, Smith Kline and French Laboratories in Philadelphia experimented with inert liquids of low boiling-point so as to discover one with the same physical properties as ethyl chloride. Dr. Bain eventually produced a mixture of fluoromethanes for us, put up in a metal canister similar to that used in household aerosols. If this spray pack ("skefron") is held a little farther away from the skin than ethyl chloride the cold effect on the skin is almost identical and the clinical results are comparable. We have given these canisters to a number of patients experiencing various kinds of pain, for them to take away for home use. One patient with moderately severe low-back pain was relieved by the skefron spray used in this department and asked for help to continue in his newly started business as a painter and decorator. He was given a canister to take home. At the end of a week he returned asking for a further supply. He told us that, although his pain had recurred at intervals, the skefron spray had relieved it so much that he did not have to give up any of his work.

Summary

Some papers recommending the use of ethyl chloride spray in the treatment of acute pain associated with muscle spasm are reviewed.

Nine years' experience in the casualty department of the General Infirmary at Leeds with the ethyl chloride spray for the treatment of acute pain in many conditions is described. These painful conditions include lumbago, acute wryneck, fibrositis, renal colic, dysmenorrhoea, and fractured rib.

A theory is put forward to account for the many striking successes achieved and also to account for the failure in some cases. The technical points necessary

for success are discussed. A new fluoromethane product, skefron, which is non-toxic and non-flammable and gives the same results as ethyl chloride, is described.

I am indebted to Dr. W. A. Bain for his long-continued interest in this treatment. The new product, skefron, was evolved at his suggestion. To Smith Kline and French Laboratories, Philadelphia, I am indebted for liberal supplies of fluoromethane mixtures for clinical trial.

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OSTEOSCLEROTIC BONE CHANGES IN PRIMARY HYPERPARATHYROIDISM WITH RENAL FAILURE

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It is well recognized that primary hyperparathyroidism may cause a serious form of renal disease (Albright *et al.*, 1934; Richardson, 1953). It is also a well-established fact that chronic renal insufficiency may result in secondary hyperplasia of the parathyroid glands (Albright *et al.*, 1937; Castleman and Mallory, 1937; Shelling and Remsen, 1935). Bone changes are sometimes detectable radiologically in these latter patients, and they then resemble closely the changes in primary hyperparathyroidism (Dent and Hodson, 1954; Stanbury, 1957). Osteoclerosis may develop in cases of renal failure, although it is an uncommon radiological finding (Crawford *et al.*, 1954; Karani, 1955; Wolf and Denko, 1958), and the demonstration of osteosclerosis in a patient with hyperparathyroidism has been taken to show that hyperparathyroidism is secondary (Brookfield *et al.*, 1955). However, the renal damage resulting from primary hyperparathyroidism could presumably produce osteosclerosis similar to that seen complicating primary renal disease. A case was reported by Beveridge *et al.* (1959) of a patient with a parathyroid adenoma in whom renal failure was present and radiological examination showed presence of marked osteosclerosis.

We wish to report the case of a patient who we believe had primary hyperparathyroidism, osteosclerotic bone changes, and renal failure. A further unusual feature was that of calcification in a haemorrhagic parathyroid cyst.

Case History

A spinster aged 63 was first seen by one of us (R. G. P.) at the request of Mr. M. P. McCormack, to whom the patient had been referred with a fracture of the right radius. The first symptom possibly attributable to her bone disease had occurred in 1955, when she attended another hospital

on account of pain in her right knee. Clinically she was thought to have an effusion, and was treated for osteoarthritis without any improvement. By 1958 she had developed pain in both knees, the right shoulder, and the right forearm; she also complained of an "occasional" swelling in the forearm.

In July, 1959, while using a hammer, she suddenly developed pain at a site corresponding to the swelling, and radiographs revealed a fracture through a "cystic" area in the radius. At no time had there been any urinary symptoms and there was no preceding history of renal disease.

On admission here she was found to have a well-marked kyphosis together with a prominent sternum. In addition, on the right side of the neck there was a hard round irregular mass which moved on swallowing.

Biochemical findings gave the following results (the details are shown in Fig. 5). Plasma calcium, 10.5 to 10.7 mg./100 ml.; plasma phosphate, 3.1 to 3.5 mg./100 ml.; total proteins, 6.3 to 6.7 g./100 ml.; blood non-protein nitrogen was raised 62 to 81 mg./100 ml. Three consecutive 24-hour estimations of urinary creatinine and calcium gave calcium/creatinine ratios of 0.27, 0.24, and 0.23. Endogenous creatinine clearance was estimated at 19% average normal clearance. On two occasions a urine concentration test gave a maximum concentration of 1009.

FIG. 1.—Tomograph showing calcified opacity in right sternoclavicular region.

FIG. 2.—Radiograph of the hands, showing rarefaction, subperiosteal erosions, and cystic areas.

The first radiological film, that of her chest, was taken in 1950; this showed no abnormality in either the ribs or the lung fields. By 1958 well-marked skeletal changes were present, and films of the neck taken at this time showed a calcified opacity in the right sternoclavicular region (Fig. 1); this opacity had produced both tracheal and oesophageal compression.

In 1959, radiographs of the tibiae, fibulae, and forearm bones showed widespread rarefaction, while the hands also showed subperiosteal absorption and several "cystic" areas (Fig. 2). In addition "cystic" changes were present in both tibiae, and the right radius showed an expanding "cyst" at the site of the previous fracture, which was accompanied by organizing periosteal new bone. X-ray examination of the skull revealed an alteration in bone texture of the vault.

Intravenous urography showed that the kidneys were small and that the contrast medium was poorly concentrated. No nephrocalcinosis was visible. The lumbar vertebrae were seen to have the typical osteosclerotic appearances known as "rugger jersey" vertebrae (Fig. 3).

FIG. 3.—Radiograph of lumbar spine, showing "rugger jersey" appearances.

FIG. 4.—Specimen removed at operation.

The biochemical findings were considered to establish a diagnosis of primary hyperparathyroidism. This diagnosis was supported further by a plasma calcium estimation of 11.1 mg./100 ml., with the plasma phosphate 2.8 mg./100 ml. immediately prior to the operation.

On February 1, 1960, the thyroid gland was exposed through a collar incision (R.G.P.). An encapsulated and calcified swelling was at once obvious, occupying most of the lower and posterior part of the right thyroid lobe, which from its position could well have been parathyroid in origin. Macroscopically, however, it did not resemble any lesion of these glands. In addition the relatively large size of the swelling tended to cast doubt on its being a true parathyroid tumour. A second and much smaller swelling (1 by 0.5 cm.) was seen in the upper and posterior aspect of the right lobe and was thought possibly to be parathyroid, but immediate section revealed normal thyroid structure.

A complete right hemithyroidectomy was performed and a thorough search made in the exposed tissues for positive evidence of parathyroid adenoma, but without success. The question then arose whether the mediastinum should be explored, but in view of the patient's poor general condition this was not seriously considered.

The specimen removed (Fig. 4) was the right lobe of the thyroid, to which was firmly attached an oval encapsulated swelling (6 by 5 by 4 cm.). The cut surface of this swelling showed it to be haematoma with fibrous and calcified walls, with at the lower end soft yellow tissue which proved on section to be parathyroid adenoma. Section of the calcified wall, including the haematoma, showed small scattered islands of parathyroid tissue both within the main capsule and in the fibrous capsule. The encapsulated swelling showed no thyroid tissue. The conclusion was that this parathyroid tumour, which contained a large number of thin-walled blood spaces, may have bled many years ago to form a haematoma, the walls of which had subsequently become fibrotic and calcified.

After operation, the plasma calcium levels fell (Fig. 5), remaining low despite calciferol, and rising again only on treatment with high doses of calciferol and calcium carbonate powder. These, however, were stopped on March 25 in view of the rising blood non-protein nitrogen, which was considered to reflect possible further renal damage as a result of the high calcium intake; during this period the

urine calcium/creatinine ratios were of the order of 0.35 to 0.66. By the time of discharge (April 20) from hospital, further radiographs had shown that reossification was occurring throughout the skeleton, and the cystic changes were regressing. However, little change was apparent in the "rugger jersey" appearance of the spine.

Discussion

The findings of an encapsulated tumour with the histological appearances of an adenoma, together with the biochemical findings enumerated above, establish the present case as one of primary hyperparathyroidism. Also the raised plasma non-protein nitrogen and the considerable reduction in endogenous creatinine clearance show that renal damage coexisted. In the absence of any preceding history of renal disease these changes presumably arose as a result of the hyperparathyroidism. Such a complication is well recognized and is usually due to nephrocalcinosis.

In addition, x-ray examination of the patient's thoracic and lumbar vertebrae showed the changes known as the "rugger jersey" spine. Essentially, this appearance is due to a horizontal layer of less dense bone being sandwiched between an upper and a lower layer of osteosclerotic bone, the latter being due to excessive osteoblastic deposition at vertebral margins.

It has been stated by Brookfield *et al.* (1955) that "the finding of osteosclerosis in hyperparathyroidism would at least exclude the hyperparathyroidism as a primary disease." In view of the findings described above, it would appear that this contention is incorrect and that no significance should be attached to these appearances in determining the primary or secondary nature of the parathyroid activity. What does seem likely is that these changes are indicative of renal damage irrespective of whether this is the cause or the result of the hyperparathyroidism. This view is supported by the cases of Crawford *et al.* (1954) and of Karani (1955), in which renal failure had given rise to secondary hyperparathyroidism, both their cases having "rugger jersey" vertebrae. Beveridge *et al.* (1959) have also described a case which, like our own, had primary hyperparathyroidism, secondary renal failure, and a "rugger jersey" pattern.

It is known that areas of osteosclerosis may develop in the skeleton of patients with long-standing renal failure without manifest evidence of hyperparathyroidism. Crawford *et al.* (1954) described such cases with increased uniform density of spine, skull, pelvis, and long bones. It does appear, therefore, that a relationship exists between renal failure and new bone formation, but the distribution pattern and severity of the osteosclerosis are variable. Precisely what the relationship is remains obscure, although the fact that the blood-forming bones are those affected and that such patients are anaemic has given rise to the suggestion that the anaemia may be a factor of importance (Karani, 1955). In this connexion it is of interest that our patient had no marked anaemia.

Another unusual feature in this case was the large size of the parathyroid tumour. Pre-operatively it was thought, as a result of clinical and radiological investigation, to be due to a goitre, and the presence of well-marked calcification in the neck was thought to be due to calcification in a thyroid cyst. Subsequently a review of the literature has shown that, although several cases of parathyroid adenomata simulating goitres have been described, radiologically detectable calcification is

FIG. 5.—Biochemical details and treatment.

rare and we have been able to discover only one case (Bogdonoff *et al.*, 1956). It is therefore not surprising that evidence of histological calcification is also uncommon.

Summary

A patient with primary hyperparathyroidism and renal damage had the radiological picture of "rugger jersey" spine due to zones of osteosclerosis. This appearance does not, as has been stated, show that the hyperparathyroidism is secondary to renal damage, but merely that renal damage coexists.

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Medical Memoranda

Lateral Cervical and Preauricular Sinuses: Their Transmission as Dominant Characters

Lateral cervical sinus or fistula is not an unfamiliar condition to the paediatric surgeon. Gross (1953) reports 148 cases from the records of the Boston Children's Hospital, 25 of them bilateral. It is also known as "branchial sinus or fistula," a designation which appears in most of the standard surgical textbooks.

The name "fistula" is used here when referring to a "complete" lesion which communicates with both the skin and the pharynx, the name "sinus" when referring to an incomplete lesion that communicates only with one of them (nearly always the skin) (Aird, 1949).

Preauricular sinus is also not rare in a children's hospital. Gross (1943) reports 44 cases, of which 12 were bilateral.

EMBRYOLOGY

Lateral Cervical Sinus.—In the first two or three weeks of embryonic life, four (or possibly five) grooves develop in either side of the neck, running obliquely downward, forward, and somewhat medially, which are called the branchial clefts. They are matched internally by out-pocketings of the foregut which are the branchial pouches. They delimit in between them the branchial arches, which later will give rise to the neck and lower part of the face. By the fifth week an overgrowth of the second branchial arch will extend down in front of the arches caudal to it, forming the precervical sinus (the cephalic portion of which is the second branchial cleft). The lateral cervical fistula is derived from the second branchial pouch, and the precervical sinus. The lateral cervical sinus, opening in the skin, derives only from the latter. Confirmation of that origin is given by the usual findings at operation; (a) that the fistulous tract runs in between the internal and external carotid arteries, the former from the second branchial arch and the latter from the third; (b) that the pharyngeal opening, when it exists, lies in the tonsillar fossa, which corresponds to the internal end of the second pouch.

Preauricular Sinus.—The different parts of the ear arise from six small nodules, first described by His (1885), which develop around the dorsal extremity of the first branchial cleft. The three nodules from the first arch would give the tragus and helix, and the three nodules from the second arch the antitragus and antihelix.

Though no agreement exists about the origin of preauricular sinuses, according to Aird (1949) the least objectionable hypothesis regards them as the result of the inclusion of part of the angle between the base of the first tubercle and the adjacent skin. The direction and length of the sinus would be due to the growth of the mandibular process with downward and forward stretching of the overlying skin, while the bottom of each groove would remain fixed.

Photographs showing lateral cervical and preauricular sinuses.