and humeri. It is therefore difficult to account for the occasional involvement of bones such as the patella, clavicle and scapula. The small size of the patella and its dense cancellous structure make the bone an unlikely site for a metastatic deposit. Joll (1923) suggested that trauma may determine the establishment of a circulating tumour embolus. In this case, however, there was no previous history of injury.

Summary

A pathological fracture through a secondary deposit of breast carcinoma in a patella is described. As far as can be ascertained, no previous case has been recorded.

I would like to thank Miss M. D. Snelling for permission to record this case, and also Dr. P. Byers and the Photographic Department of the Institute of Orthopaedics for their help.

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HYPERTHYROIDISM COMPLICATED BY PERIODIC PARALYSIS

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PERIODIC paralysis associated with hyperthyroidism is extremely rare outside Japan. In a series of 6,333 cases of hyperthyroidism admitted to three hospitals in Japan the incidence was found to be 2% (Okinaka, 1957). Twenty cases have been reported in America (Bartels and Pouget, 1963) and four cases have been reported in England (Petch, 1964). A further case is reported below.

Case Report

H. B., a 32-year-old fitter, who had previously been in excellent health, awoke in the early hours of the morning of December 27th, 1963 to find himself paralysed from the waist down. His lower limbs gradually regained their full strength five hours later. He had no more trouble until January 9th when he awoke in the early hours of the morning to find himself unable to move his arms and legs, but able to breathe, speak and swallow normally. He had no difficulty in controlling his bladder or bowels. During this second attack he was examined by his practitioner who found that he had a flaccid paralysis with normal sensation and absent limb reflexes. This attack lasted nine hours. On several occasions after these two attacks his thighs felt weak and heavy after he had been sitting for some time, but this sensation wore off after he had walked around the room for a while. There was no family history of similar attacks.

A few weeks before his first attack of paralysis he noted that he had become severely irritable and, despite a very good appetite, he had lost two stones in weight.

On Examination he had no abnormal neurological signs, but he had a moderate-sized nodular goitre. His pulse rate was 112 per minute, he had hot

sweaty palms and a fine tremor of the outstretched fingers. There were no eye signs.

Investigations. BMR + 92% and + 75%, on two occasions. Serum protein bound iodine 12.4 $\mu\text{g}/100\text{ ml.}$, serum potassium on four separate occasions was 4.1, 4.4, 4.0, 3.3 mEq./l.

A diagnosis of hyperthyroidism associated with periodic paralysis was made and it was decided to prepare him for surgery. First he was given carbimazole. Ten days before sub-total thyroidectomy was performed he was given Lugol's Iodine.

Operation. The clinical impression of a nodular goitre was confirmed and sub-total thyroidectomy was performed. The patient's recovery from the operation was straightforward and when he last attended out-patients six months later he was found to be euthyroid and he had had no more attacks of paralysis.

Histological examination of the thyroid revealed a hyperplastic gland with scanty colloid.

Comment

Periodic paralysis is the rarest of the neuromuscular disorders which can complicate hyperthyroidism. When it is not associated with hyperthyroidism, periodic paralysis is familial; in 80% of cases, occurs usually in adolescence and spontaneously regresses at the age of thirty. The sex ratio is three males to one female. When the disease complicates hyperthyroidism it is not familial; it most commonly occurs between the age of thirty and fifty and the sex ratio is twenty males to one female (Okinaka, 1957).

The diagnosis is suggested by the history. The attacks usually occur in the early hours of the morning and last several hours before ceasing spontaneously. The muscles of the limbs are

most commonly affected and it is rare to find any disturbance of respiration, phonation or deglutition. The level of serum potassium during an attack is often low, but may be normal or even high. An intravenous infusion of potassium may speed recovery from the paralysis (Overholt, 1957).

Paralytic attacks can frequently be produced by the administration of carbohydrate and insulin. This lends support to the theory that the cause of this attack is a shift of potassium from the extracellular to the intracellular fluid compartments. Grob, Johns and Liljestrand (1957) found a marked difference in the potassium content of arterial and venous blood from the forearm during an attack and postulated that the potassium had passed into the muscle cells. Conn (1957) investigated two cases of familial periodic paralysis and found that the attacks were preceded by an increased urinary output of aldosterone associated with sodium retention, but no increase in the urinary excretion of potassium. Jones (1959) failed to duplicate Conn's results.

The role of the thyroid hormones in producing attacks of paralysis is not clear. The administration of thyroid hormones to familial cases of periodic paralysis can produce an attack (Shinosaki, 1925). Recurrence of hyperthyroidism has, on two occasions, resulted in the recurrence of attacks of periodic paralysis. (Dunlap and Kepler, 1931). When periodic paralysis complicates hyperthyroidism, successful medical or surgical treatment of the hyperthyroidism always cures the paralytic attacks.

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