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HYPOPARATHYROIDISM FOLLOWING THYROIDECTOMY

What is the true prevalence of parathyroid insufficiency after thyroidectomy? Estimates in the past have varied from less than 1% to more than 10%. These differences may depend not only upon the method or type of surgery, but perhaps just as much on the criteria used for diagnosis of hypoparathyroidism, and upon the length and thoroughness of the follow-up investigations. Clinical evidence of hypoparathyroidism may be non-existent for many years, appearing later at the time of some particular metabolic parathyroid stress, such as pregnancy and lactation. So-called 'temporary' hypoparathyroidism — minor tetany or cramp with paraesthesiae shortly following a thyroid operation, easily controlled by extra calcium, and passing off within a few days — must probably be considered as a danger signal. It is quite likely that in most if not all of such cases, the parathyroids do not ever completely recover, so that a latent parathyroid insufficiency may lurk in the background for years with eventual dire effects upon the patient. An illustrative case is noted in the *Lancet*:[1] 'In 1944 a young woman, aged 28, had her thyroid removed on account of nodular goitre. After the operation she had slight cramps and paraesthesiae which were easily controlled with calcium. In 1953 she was admitted to hospital for the investigation of convulsions and papilloedema, and in 1955 she was blind with cataract — all the result of chronic hypoparathyroidism'.

The clinical features which may eventually appear are quite varied, and enter the domains of several different specialities. Tetany is frequently absent in the chronic state, but there may be integumental changes, especially eczemas and impetigo herpetiformis, alopecia, and nail dystrophy; ocular cataracts; attacks of laryngeal crowing; features resembling the 'frozen shoulder'; fits with papilloedema; mental changes including anxiety and depression; abdominal pains; and more minor symptoms of general lassitude and vague ill health since the operation.[2]

It is thus incorrect to equate hypoparathyroidism with tetany, and the disappearance of tetany with a return to parathyroid normality. Estimations of serum calcium should certainly be made after a thyroidectomy, but even so a single reading within the low-normal range is no guarantee of continued normality, because of spontaneous variations in the calcium level and because of the possible effect of future metabolic stresses. It is today a general principle that the integrity of the function of an endocrine gland can be well tested only in conditions which apply a maximum stress to that gland. Thus we test adrenal cortical function not only by means of the estimation of the resting levels of urinary 17-hydroxycorticoids and plasma cortisol, but also by the increase in these levels in response to stimulation with corticotrophin. We test the function of the pancreatic beta cells not only by measuring the fasting blood sugar, but by observing changes in the sugar level following a glucose load. The parathyroid glands have a primary function in maintaining the levels of the plasma ionized calcium. To make them work hard we must produce a state which will tend to lower this level. Workers from Cardiff[3] have recently standardized such a test. They gave a diet containing 180 mg. of calcium daily, and added 9 G. of phytate daily two days later, continuing for 3 - 5 days. The effect of the phytate is to lower even further the available intestinal calcium by producing non-absorbable calcium-chelated-phytate. They found that in normal persons the plasma calcium did not fall, and that a figure below 8·5 mg. per 100 ml. was evidence of parathyroid insufficiency. Using this test they set out to ascertain the prevalence of partial hypoparathyroidism and to study its manifestations.[4]

In their study they examined 82 patients who had undergone thyroidectomy, none of whom had features of overt hypoparathyroidism, and 82 randomly selected control subjects. During their calcium-deprivation test they found that at least 24% of the post-operation group were unable to maintain a normal plasma-calcium level. These patients all complained of symptoms rather difficult to evaluate, including paraesthesiae, cramp in the legs, lethargy, depression and lassitude, abdominal pain, syncopal attacks, headaches, ectodermal lesions, and tightness in the throat. These symptoms appeared largely to abate with the use of additional oral calcium. It must be admitted, however, that such symptoms, apparently associated with partial hypoparathyroidism, were all of that common, non-specific variety so often seen in a general practice or hospital out-patient department. Furthermore, it is rather difficult to accept that they were caused by hypoparathyroidism with a 'non-stressed' serum calcium within the normal range.

Whatever reservations we may have about these symptoms, it is clear that, at least biochemically, the Cardiff workers have shown a very high prevalence of partial hypoparathyroidism to exist following thyroidectomy. Further analysis indicated that the frequency of (unstressed) plasma-calcium values below 9·3 mg. per 100 ml. did not differ significantly, whether the operation had been a subtotal or partial thyroidectomy, but that such low readings were more frequently found following multiple operations on the thyroid.

If we accept the high frequency with which the parathyroid glands are damaged at operation, we must next ask why? Parathyroid glands appear to be supplied by single, tiny end-arteries, almost always arising from the inferior thyroid artery.[5] The danger of ligating this artery during thyroidectomy, of carefully identifying or handling the parathyroid glands, or of haemostatic manoeuvres beneath the capsule of the thyroid are thus apparent and should presumably be avoided where possible. It would also appear that parathyroid glands are actually inadver-

tently excised far more commonly than surgeons would generally believe. In 332 thyroid specimens recently reported upon by Murley and Peters after removal by one of the authors and his colleagues, parathyroid glands were found in 14%.[5] Probably no surgeon can guarantee not to produce parathyroid damage, but he should perhaps feel obligated to follow up his patients longer and more carefully than is usually done at present.

1. Leading Article (1961): Lancet, **2,** 144.
2. Smith, J. W. G., Davis, R. H. and Fourman, P. (1960): *Ibid.*, **2,** 510.
3. Davis, R. H., Fourman, P. and Smith, J. W. G. (1961): *Ibid.*, **2,** 1432.
4. Halsted, W. S. and Evans, H. M. (1907): Ann. Surg., **46,** 489.
5. Murley, R. S. and Peters, P. M. (1961): Proc. Roy. Soc. Med., **54,** 487.