as effective observation units where the correct disposal would be worked out.

Summary

The course and outcome of patients aged 60 and over admitted to the geriatric unit and the mental hospital serving the city of Belfast have been examined. Comparisons were made between those correctly placed and those misplaced—that is, those whose principal illness was one which would have been more appropriately treated in the other hospital.

The misplaced patients had a higher mortality in hospital than was found among the correctly placed patients. This was so in both hospitals. Also, their mortality was higher than that of patients suffering from similar ailments who had been admitted to the proper unit. Among the survivors, the misplaced patients were less often discharged than correctly placed patients. These differences were not accounted for by differences in age or sex, or by the seriousness of the illnesses. Patients with mixed physical and mental disabilities did not have an especially bad prognosis. Misplacement itself seemed to be directly responsible for the poor outcome of the patients. Reasons for this poor outcome are discussed, and ways of avoiding misplacement are outlined.

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Opportunities offered by male nursing as a career for boys are outlined in *Nursing for Men*, a revised edition in the "Choice of Careers" series published by the Central Youth Employment Executive. Details are given about training and the conditions required for admission to the Index of Student Nurses. Information is also given about conditions of service, openings and prospects, opportunities for male nurses in H.M. Forces, the Prison Service, in territories overseas, and under the guidance of the World Health Organization. (H.M.S.O., 1s. 6d. net.)

SOME PROBLEMS OF HYPERPARATHYROIDISM*

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Osteitis Fibrosa

The problem posed by the only occasional coexistence of a specific bone disease, osteitis fibrosa generalisata, has not to my mind been sufficiently studied. Albright and Reifenstein (1948) clearly stated that there were two forms of primary hyperparathyroidism—that "with" and that "without" bone disease—and they further suggested that the patient's previous calcium intake was the factor determining whether he got the bone disease or not. They held that a high calcium intake could put any patient "with" bone disease into positive balance and eventually cure the bone disease, and that the patient's previous calcium intake therefore determined the state of his bones—low intake inducing bone disease, high intake preventing it. However, many clinicians have noted individual patients who did not appear to follow this rule, and the only systematic dietary survey (Dent, Hartland, Hicks, and Sykes, 1961) showed that the previous calcium intake of patients "with" bone disease did not differ from those "without" (Fig. 8). Others have argued that the duration of the disease was the significant factor, the bone disease taking a long while to develop and therefore being present only in long-standing cases, all cases being presumed to begin and, for some time, to remain "without" bone disease. A more plausible theory could be that the severity of the hyperparathyroidism was relevant, only the more severe ones—that is, with a higher parathormone production—getting the bone disease. Whichever of these theories one might favour, it is obvious that it would imply that the forms "with" and "without" bone disease must merge into one another without any sharp line of demarcation.

I have held for some while that none of these theories fit all the known facts, and that there must be some other reason for the presence or otherwise of the osteitis fibrosa. I put forward the following arguments relative to this point:

1. Most clinicians agree that it is nearly always possible to divide patients with proved primary hyperparathyroidism clearly into either the group with or into the group without bone disease on the basis of their bone x-ray films, and often purely on clinical grounds. When this is done it is found that the plasma alkaline phosphatase levels also divide into two groups—that is, a biphasic distribution—the patients with bone disease having raised levels, those without having normal levels (Dent and Harper, 1962). There is surprisingly little difficulty in making this grouping into two different "diseases." We think this would be more difficult to do if they all represented one single spectrum of a single disease of varying severity; indeed, the distribution should then be monophasic.

2. It has often been noted that patients with osteitis fibrosa tend to have a higher average plasma calcium. Many interpret this as a consequence of increased parathormone production. However, I would point out that the correlation is not a close one. Our data, for instance (Fig. 14), show

*Conclusion of the Humphry Davy Rolleston Lecture delivered to the Royal College of Physicians, London, on June 18, 1962.

that the majority of patients with the bone disease have total plasma calciums of below 13.5 mg./100 ml. Moreover, the patients with the highest plasma calcium happen in our series to have minimal signs of bone disease as seen on x-ray film or as determined by measurement of the plasma alkaline phosphatase. Indeed, a plot of plasma calcium against alkaline phosphatase shows, if anything, a negative correlation (Fig. 15). I take this to mean that there is no evidence yet that the degree of hypercalcaemia and the presence and severity of the osteitis fibrosa are due to the same immediate cause—namely, a very high production of the same parathyroid hormone.

3. Patients in the group with bone disease have a shorter total history of all symptoms than those without bone disease (Fig. 16). This is the wrong way round if it were a

FIG. 14.—Plasma calcium levels (averaged for each patient) in our first 80 patients divided into the three groups shown. Some of the highest plasma levels were in the patients with osteitis fibrosa. However, the four highest shown here had only mild bone disease as compared with some of those with much lower total calciums. Moreover, some quite high calciums are obtained in the group who had formed neither stones nor osteitis fibrosa. These nine latter patients were diagnosed by the curious methods described in Table II.

FIG. 15.—A plot of pre-operative levels of total plasma calcium against alkaline phosphatase in our 25 patients with bone disease. Phosphatase level roughly correlates with the severity of the osteitis fibrosa as visible radiologically. It is clear that there is no direct relationship between the height of the calcium and the height of the phosphatase. Indeed, it can be argued that the relationship shown is, if anything, an inverse one.

longer duration of disease that was the factor required to give time for the bones to be affected. The duration of known stone formation has not been separately shown. It is even shorter in the bone-disease group, since many do not get stones at all, most having presented only with bone pains. The exceptional patient with parathyroid cancer mentioned in the legend to Fig. 16 justifies further thought. A similar history was given by another of these patients with the rare parathyroid cancer (Cook, 1958). Her history of bone disease (an osteoclastoma of the jaw) was of only just over a year. She had had renal stones removed seven years previously and had a much longer history of dyspepsia. It seems likely that in both these cases the beginning of the bone disease followed malignant change in a parathyroid adenoma which up till then was only causing stone formation. We have not yet obtained such a history from a patient with a simple adenoma of the parathyroid.

4. There is more renal damage in the patients with bone disease than in those without

(Table III). When it occurs in those with bone disease there is not usually a simple explanation in the form of, say, an obstructive nephropathy due to renal stones. The renal failure occurring in the group without bone disease can usually be explained as the result of stone formation and is in any case not commonly severe.

It is remarkable how long the history of stone formation is in some patients in this group, and many after 30 or more years of recurrent stones have surprisingly normal renal function. It is odd, too, that nephrocalcinosis is more constantly associated with renal damage in the bone-disease group. Only three of our eight patients in the group with bone disease and nephrocalcinosis had "normal" renal function as defined in Table III, and it should be stressed that all three had marked renal tubular dysfunction as shown by a vasopressin-resistant diabetes insipidus and a renal amino-aciduria. In contrast nine out of the ten with nephrocalcinosis and without bone disease had normal renal function.

TABLE III.—Primary Hyperparathyroidism (U.C.H.). Incidence of Renal Damage and of Nephrocalcinosis Visible on Plain X-ray Film of Abdomen. We Omit Here Our Nine Patients Without Bone Disease and Without Stones

25 Patients with bone disease ± stones or nephrocalcinosis:
Normal renal function, 11 patients (3 with nephrocalcinosis)
*Abnormal renal function, 14 patients (5 with nephrocalcinosis)
46 Patients without bone disease, all having stones or nephrocalcinosis:
Normal renal function, 43 patients (9 with nephrocalcinosis)
*Abnormal renal function, 3 patients (1 with nephrocalcinosis)

*Defined as having average pre-operative plasma-urea levels of over 50 mg./100 ml.

5. Cortisone has not affected the plasma calcium in 13 of our patients without bone disease (Fig. 4). It has made it normal in one out of two with bone disease and perhaps in another not at that time under our control (Case 2 of Dent and Harper, 1962). We need to confirm this in more patients before pressing this as evidence.

6. There is a difference between secondary hyperparathyroidism following renal disease and primary hyperparathyroidism. In the renal cases there is a close correlation between the size of the hypertrophied parathyroids and the severity of the osteitis fibrosa, and patients dying with normal-sized parathyroids do not have the bone disease (Gilmour, 1947). In primary hyperparathyroidism large adenomas occur frequently in cases without bone disease.

7. Extraordinary cases are beginning to be reported in which the plasma levels of calcium and phosphorus characteristic of hypoparathyroidism (low calcium, high phosphorus) with high renal phosphate threshold occur in the presence of quite typical radiological signs of osteitis fibrosa. We have several patients with steatorrhoea and renal disease who have shown this bizarre combination.

FIG. 16.—Duration of symptoms likely to be due to the hyperparathyroidism in our first 80 patients. Note the much shorter history in the case of the patients with bone disease. In most of these patients the symptoms were related to the skeleton rather than to stone formation. It could be argued that the two odd bone-disease patients with a history of 10 to 12½ years were sufficiently atypical to justify their omission from the graph. One of them had passed a stone 12 years previously, but was quite well without further stone formation till he developed increasing bone pains and weakness over 18 months. His parathyroid tumour was carcinomatous. The other odd patient gave a 10-year history of bone pains. She was the very exceptional one with coeliac disease described by Davies, Dent, and Wilcox (1956).

Kolb and Steinbach (1962) have recently described this in two patients who also have the physical features of the pseudohypoparathyroid syndrome. We have one patient at U.C.H. who shows a similar "hypo-hyperparathyroidism" (but without pseudohypoparathyroid signs) and who is otherwise completely healthy. It appears from our patient that large doses of vitamin D can correct the signs of both hypoparathyroidism and hyperparathyroidism, but unfortunately we are not so sure of this in the cases elsewhere, as the vitamin D has not always been given as sole therapy.

A possible explanation for the above facts is that the parathyroid gland can produce two hormones—one acting on bone to produce osteitis fibrosa and a raised alkaline phosphatase and having a nephrotoxic action additional to that produced by hypercalcaemia if present, the other having only the combined calcium-raising and phosphorus-lowering action of the pure Rasmussen preparation. In ordinary primary hyperparathyroidism without bone disease only "Rasmussen hormone" would be overproduced. In cases with bone disease and in hyperparathyroidism secondary to renal disease both hormones would be overproduced. In the bizarre patients mentioned above the combination of too little "Rasmussen hormone" and too much osteitis fibrosa hormone could be assumed to occur. Another possibility could be to assume variable end-organ responses to a single parathyroid hormone having all the above actions, or it might be that circulating metabolites can block some but not all the actions of such an all-purpose single hormone. A substance such as the "anti-vitamin-D factor," supposed to circulate in the plasma of patients with renal failure, might fit this role (Dent, Harper, and Philpot, 1961; Stanbury and Lumb, 1962).

Whatever the explanation we would like to suggest that fruitful experimental work could be devised to explore the clinical anomalies. Clearly we want to know first how efficacious the pure hormone so far obtained is in causing osteitis fibrosa and in raising the phosphatase level. Then we want to know if extracts can be obtained from the parathyroid gland with different relative potency for raising the plasma calcium and the plasma phosphatase. Finally, can different hormones be extracted from human parathyroid tumours from the groups of patients with and without bone disease? It seems odd to me that many workers have spent time showing that the one hormone had both calcium-raising and phosphorus-lowering properties, a response to be expected on the clinical evidence; while nobody I am aware of has recently tackled or even considered the problem of the osteitis fibrosa, a matter with intriguing difficulties of interpretation that shout at one from the clinical evidence. Indeed, many authors describing cases of primary hyperparathyroidism have omitted to record which of them had osteitis fibrosa and raised phosphatase levels. Recent evidence suggesting the production by the parathyroid of a hormone with hypocalcaemic action has recently been published (Copp and Cameron, 1961). This does not yet seem to tie up with any of our clinical problems, but it does remind us not to be too fixed in the view that there is only one hormone produced by the one endocrine gland.

Medical Treatment of Primary Hyperparathyroidism

Three further studies of patients with primary hyperparathyroidism may be of sufficient interest to record here. Mrs. Emily P. came to us with classical radiological, clinical, and biochemical signs of primary hyperparathyroidism. She had already been explored elsewhere unsuccessfully in the neck and mediastinum for a

parathyroid tumour. In two exceptionally difficult and thorough explorations, again of neck and of mediastinum (Fig. 12), Mr. Davies was unable to find the tumour (this is his only failure in our U.C.H. series to date). We thought the patient, who by now firmly refused further surgery, presented us with an exceptional and long-desired opportunity to attempt medical means of therapy, which in other circumstances would have been entirely unethical. We accordingly treated her in 1957 with a high dose of disodium hydrogen phosphate by mouth. The remarkable result is shown in Fig. 17. It seems indisputable that the phosphate load was capable of lowering her total plasma calcium level with only a small induced rise in plasma phosphorus. Nevertheless, with this change she became symptom-free, her x-ray subperiosteal erosions healed, and her phosphatase became normal.

We wondered how far this could be a spontaneous change from damage to her parathyroid adenoma at her three explorations, so, with her full understanding and agreement, she undertook to stop taking the phosphate for a period in late 1961 and early 1962. Her symptoms returned, with corresponding worsening of the biochemical and radiological signs. Everything has now become more normal

FIG. 17.—Follow-up data on Mrs. Emily P., with primary hyperparathyroidism, on whom we have been unable to find a tumour. She has been treated only with oral sodium phosphate as shown. All her symptoms have disappeared, with healing of the subperiosteal erosions and a lowering of the alkaline phosphatase to normal levels. The calcium and phosphorus levels have not become fully normal, however. Note especially the immediate setback at the end of 1961, when we withheld the sodium phosphate, and the immediate correction when it was reintroduced. Renal function is remaining surprisingly normal throughout this time.

FIG. 18.—Mrs. Margaret B. Carcinoma of the parathyroid. Follow-up data showing the effect on her total calcium and other variables of the two surgical procedures for removal of parathyroid tissue and then, when she became inoperable, of our medical procedures.

since the phosphate has been begun again. The reciprocal changes shown with the large changes in plasma calcium and phosphorus levels were described as occurring in hyperparathyroidism some while ago (Albright, Bauer, Claflin, and Cockrill, 1932), and we have fully confirmed this in many other patients (see also Fig. 3). The Albright school seemed to have erected their original theory of parathyroid hormone action on this basis. At U.C.H., however, we have not been able to show that this is a general phenomenon; for instance, it does not occur in normal subjects (Philpot, 1958), presumably because of the greater responsiveness of the normal parathyroid glands. Experimental work could well investigate further this remarkable phenomenon. We have suggested oral phosphate as of possible use in the hypercalcaemic crisis occurring in inoperable cancer of the parathyroid, but in one trial done rather late (Cook, 1958) it was of no help at all. We have, however, further data from a recent case of our own (Fig. 18).

This patient had had two neck explorations—one for the removal of the primary tumour, found to be a parathyroid cancer, and another for removal of locally recurring secondaries. She did well for a time in each case, but on her third severe hypercalcaemic phase she was regarded as inoperable. We therefore tried her on two forms of therapy we had previously considered of possible value in such a case. The first, already in operation since her second exploration, was to render her partly myxoedematous. This was easy here, since we had asked Mr. Davies to remove her thyroid gland at her second neck operation and had since kept her on maintenance triiodothyronine at a dose of only 20 µg. a day. This was continued, the patient tolerating well the mild symptoms of hypothyroidism. The second measure was to give oral disodium hydrogen phosphate. This worked well for a month, but she developed severe vomiting and was readmitted with severe hypercalcaemia. As she could not tolerate anything by mouth the first dose of phosphate was given intravenously and had the unexpected result of lowering her calcium to tetany levels. We worked out a smaller maintenance dose which we later could give again by mouth. The ensuing biochemical remission by purely medical means was comparable to that produced by the two previous surgical explorations. Her clinical remission continued so far as the symptoms of hypercalcaemia were concerned and she was in good shape for a while. Later, however, she developed painful ectopic calcification in her fatty tissues and arteries. The slow rise in phosphatase and of x-ray signs of hyperparathyroidism clearly show that her parathyroid hormone production has not been inhibited as was the case with the patient shown in Fig. 17. It is unfortunately possible that the phosphate increased the rate of ectopic calcification.

We have one more contribution to make with regard to medical treatment of osteitis fibrosa due to "primary" hyperparathyroidism. Mrs. May B., some of whose plasma calcium levels are shown in Fig. 10, presented a problem in diagnosis in early 1957. The newly devised method for determining ionized plasma calcium twice gave the rather high figure of 6.8 mg./100 ml. (Dr. G. Alan Rose). However, her total calcium was normal (10.2 mg./100 ml.) and I (against Dr. Rose's opinion) was hesitant to advise operation. In discussion with the patient (who was very reluctant to have an operation) it was decided to treat her first with vitamin D (as dihydrotachysterol) on the assumption that she had

hyperparathyroidism secondary to her chronic renal failure. The patient was told that this would probably not help her if indeed she needed neck exploration, so that such a therapy trial would be a help in diagnosis. The main results are summarized in Fig. 19. It is seen that her signs and symptoms of osteitis fibrosa quickly improved, so much so that the patient became almost addicted to dihydrotachysterol and took it without our approval when she could. We finally decided that we could cure the bones only by producing hypercalcaemia, so the hyperparathyroidism had after all at that stage become "primary" or, more likely, that an autonomous parathyroid tumour was now functioning. We were now able to put more pressure on the patient, and at operation Mr. Davies found a

single chief-cell adenoma. She is now well without treatment in spite of continuing and very slowly progressive renal damage. We fear she may grow another autonomous parathyroid tumour in time if our theories up to now have been correct.

A Diagnostic Problem

Some remarks now on the problem of distinguishing secondary hyperparathyroidism from primary. When associated with steatorrhea the diagnostic problem is not too difficult, but when there is the more common association with renal failure then we are in much greater trouble as in the case of Mrs. May B., above. Primary hyperparathyroidism, especially the form with bone disease, often leads to the development of secondary renal failure. Long-standing renal failure, even if mild and symptomless, almost always leads to some degree of secondary hyperparathyroidism, as evidenced by bone x-ray films showing osteitis fibrosa, a raised alkaline phosphatase, and enlarged parathyroid glands (detectable only at necropsy or operation). To make things more difficult for us a mild degree of renal damage raises the plasma phosphate level and invalidates the renal phosphate studies, and this phosphate retention tends to lower plasma calcium to more ambiguous levels. Even worse difficulties arise from our belief that such patients can develop functioning autonomous adenomata, so that when one of these is found and identified it may still be the result of primary renal failure.

We have had a lot of trouble at U.C.H. with this problem. For instance, we let Miss Dorothy J. (Fig. 9)

FIG 19.—Mrs. May B. Primary hyperparathyroidism, or perhaps an autonomous parathyroid tumour, resulting from chronic hyperplasia from prolonged renal damage. We treated her with dihydrotachysterol owing to an early misdiagnosis of secondary hyperparathyroidism, and once having started we found it difficult to persuade her to let us stop it, since she felt so much better from the point of view of the bone pains and suffered no symptomatic or other notable disadvantage from the ensuing prolonged hypercalcaemia. Except for the latter, which is obviously bad for her, we can claim to have "cured" her disease during this time.

die in 1955 without much idea of what we should be doing; while Mrs. May B. (Figs. 10 and 19) we equivocated over for three years before firmly advising the neck exploration. We think we have had a near shave with Mr. Philip B., whose follow-up charts are shown in Fig. 20. He was and still is hale and hearty in spite of having constantly a marked proteinuria and plasma urea levels of around 120 mg./100 ml. He presented with pains in his feet and back, and x-ray films showed large cystic lesions in his pubic rami and only mild phalangeal erosions. His urine protein behaved on the boiling test exactly like Bence Jones protein, so our first guess was that he had myelomatosis. However, we were unable to confirm this with more specific tests, and we decided then that he must have hyperparathyroidism secondary to renal disease of unknown origin. He was given vitamin D with excellent clinical, radiological, and biochemical response, but he became rapidly hypercalcaemic and remained so for some months after we stopped the vitamin D, during which time we were seriously considering again the

FIG. 20.—Mr. Philip B. Follow-up chart showing the results of our treatment with at first vitamin D₃ and later dihydrotachysterol. His requirement for these substances is much less than usual, so we overdoed him and he had several periods of hypercalcaemia, from which fortunately he had no untoward symptoms. However, owing to these high total plasma calcium readings we were several times in doubt about whether he was all the time a patient with primary rather than secondary hyperparathyroidism. The latter is now a firm diagnosis. He slowly developed osteitis fibrosa again during the recent period without treatment, so he has just had to begin taking dihydrotachysterol again.

diagnosis of primary hyperparathyroidism. However, our follow-up shows that we have, by lowering the dose of vitamin D, managed to heal his bones without necessarily producing hypercalcaemia, and we interpret this as proof of the secondary origin of the hyperparathyroidism. In view of our experiences with these and other patients we are surprised to read in Rasmussen and Reifensstein's (1962) recent review that "primary hyperparathyroidism can be differentiated from secondary hyperparathyroidism very easily."

Features for Further Study

May I now summarize what I believe to be the main features of hyperparathyroidism that require more study and explanation? I quote them in the sincere hope that they may stimulate work by others.

1. Primary hyperparathyroidism of the type without bone disease can exist in a patient for 30 to 40 years with complications due almost only to those directly related to stone formation. In patients whose stones do not obstruct urinary flow or whose hypercalcaemia does

not cause appreciable symptoms the disease may cause surprisingly little renal damage. We have no evidence that osteitis fibrosa has occurred as a late complication in any such case except in the two patients mentioned in the text whose parathyroid tumours were found at operation to be carcinomatous.

2. Primary hyperparathyroidism of the type with bone disease seems to run a shorter course before demanding medical attention and consequent diagnosis. In spite of this, nephrocalcinosis and significant renal damage occur in most of these cases. Often renal stones do not occur, and in many cases it is quite clear that the renal damage cannot be consequent on obstruction to urine flow from stone formation. There is no adequate evidence either that the renal damage is more severe on account of a much more severe or prolonged hypercalcaemia.

3. There is no adequate explanation for the two types of primary hyperparathyroidism. The pathology of the parathyroid tumour and the calcium and phosphorus metabolism seem identical, but the clinical picture is generally so different as to justify their description as two "diseases."

4. Recent case records emphasize the existence of states in which the bone changes are those of osteitis fibrosa, while the plasma calcium and phosphorus levels are those of hypoparathyroidism. Such patients present us with a considerable theoretical challenge. There is, however, no problem in treatment, as vitamin D cures both states simultaneously.

5. The osteitis fibrosa of both primary and secondary hyperparathyroidism can be healed with vitamin D. There are strong theoretical considerations suggesting that this treatment should not be used in primary hyperparathyroidism; but in secondary hyperparathyroidism the treatment, while having some risks, is nevertheless usually most efficacious. We still do not know how vitamin D works here. A direct action on parathyroid hormone production seems possible, although the more likely explanation is that it acts by producing a small rise in plasma calcium which then leads to suppression of parathyroid hormone production.

6. In our one patient with primary hyperparathyroidism with bone disease and an undiscoverable tumour we have good evidence that a high phosphate intake cures the bone disease and makes plasma calcium and phosphorus levels less abnormal. There has been no progression of renal damage nor other discoverable complication of treatment during the four years of her follow-up on high phosphate. It should be noted here that the healing of the hyperparathyroidism while the plasma calcium is being lowered by phosphate represents a quite opposite situation from that under No. 5.

7. It seems highly probable that autonomous adenomas can rarely arise in patients with steatorrhoea or renal failure, perhaps as a result of calcium depletion. However, primary hyperparathyroidism does not usually arise as a result of a period of low dietary calcium intake, neither does the development of osteitis fibrosa seem to be related to this factor.

8. There are a remarkable number of other diseases whose association with primary hyperparathyroidism occurs too often to be due to chance. The reasons for these associations seem entirely obscure but may provide further hints on the aetiology of hyperparathyroidism. It can be recalled that the opposite state of hypoparathyroidism has a similar large and curious set of mostly different associations.

9. We need to know more about those patients with primary hyperparathyroidism whose plasma calcium levels fall with cortisone, especially whether they are mostly those who have osteitis fibrosa.

10. There seems no simple explanation for the fact that when hyperparathyroidism secondary to renal disease is finally proved to have occurred because of the finding of hyperplastic glands at post-mortem examination these patients always have osteitis fibrosa (Gilmour, 1947). Yet only about 30% of patients with primary hyperparathyroidism, even when of long duration, get osteitis fibrosa.

11. The parathyroids in renal disease from sarcoidosis need further study. Our own data and our reading of the literature suggest that secondary hyperparathyroidism from this cause is much rarer than should be expected but that "primary"—that is, autonomous tumour formation—hyperparathyroidism occurs more often than can be accounted by chance.

12. Patients with parathyroid cancer need further study in case they show a different evolution from patients with adenoma.

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RESULTS OF INTERNAL SPHINCTEROTOMY FOR ANAL FISSURE

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Anal fissure is a common and painful condition, and, though frequently dismissed by the doctor as a minor complaint, it is in fact responsible for much discomfort, so that its successful treatment results in a very satisfied and grateful patient. A variety of surgical methods are available for the management of this condition. The longest-established is most probably simple stretching of the anal sphincters, which has been practised for very many years. Excision of the fissure with a triangular piece of perianal skin, which was introduced by Gabriel in 1930, has had many supporters. Hughes (1953) modified this method by applying an immediate split-skin graft to the resulting wound. Miles (1939) treated anal fissures by "pectenotomy," or division of what he called the "pecten band" in the lower part of the anal canal. We now know that this apparent fibrous band is really the spastic, prominent, lower edge of the internal sphincter (Eisenhammer, 1951; Goligher, Leacock, and Brossy, 1955). The operation, which would thus be more accurately termed "internal sphincterotomy," has been revived under its correct designation by Eisenhammer (1951), and has become one of the most widely used methods of treatment for fissure in recent years. What has contributed to its popularity is the fact that with proper facilities and technique it is very simply performed, and can be done as an out-patient procedure if desired, thus relieving the strain on hospital beds.

Internal sphincterotomy has been warmly recommended also by Morgan and Thompson (1956), Lockhart-Mummery (1957), and Goligher (1961) as providing a reliable cure for anal fissure. These recommendations, however, appear to be based on general clinical impressions rather than on detailed follow-up studies. A most important aspect of the results on which precise information would seem to be particularly lacking is the effect of the operation on anal function. It would be natural to inquire whether such division of the internal sphincter can be accomplished without impairment of anal continence. Morgan and Thompson (1956) did not comment at all on the functional condition of their patients treated by sphincterotomy. Goligher (1961) stated that control for flatus and liquid faeces was impaired for several weeks in some of his sphincterotomy cases, but this eventually returned to normal. Brossy (1956) claimed that there was no lasting

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