Section of Proctology

President Professor J C Goligher chM

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Fistula-in-ano [*Abridged*]

Mr A G Parks (*The London Hospital and St Mark's Hospital, London*)

and

Dr B C Morson (*St Mark's Hospital, London*)

The Pathogenesis of Fistula-in-ano

There are two problems relating to the cause of fistula-in-ano. The first is the passage of organisms across the anal wall into the peri-anal tissues; the second is the persistence of this infection which results in a chronic, abnormal communication between the anal canal and perineal skin.

Peri-anal suppuration is common compared with abscess formation around other parts of the alimentary tract. This frequency must have a scientific explanation. When a perivisceral abscess does occur, for example in the intestine, it is usually caused by a diverticulum, or an ulcer perforating all coats of the bowel wall. A superficial breach in the mucosa, such as a fissure, cannot be seriously entertained as an explanation for peri-anal suppuration for, if this were so, such infection would be a common complication of hæmorrhoidectomy.

Assuming that peri-anal infection has occurred and has led to a fistula, it is difficult to explain why it should persist. The natural history of an abscess is that it discharges and heals. If it fails to do so then the suspicion is aroused that it has some specific pathogenesis such as tuberculosis or is connected with a deep-seated, poorly drained abscess, or an infected cyst. The fact that it is a fistula is not in itself an explanation for its persistence, for fistulæ also have a natural tendency to heal unless there is a good reason for their not doing so.

Comprehension of the anatomy of the anal region is essential for treatment and for understanding the pathogenesis. Before infection can set up an abscess in the peri-anal tissues it must cross the mucosa and the circular muscle layer, which is at this point the internal sphincter. There are many fibrous tissue septa intersecting the various muscles, but although these might determine the direction of spread of pus, they do not offer an explanation for the initial infection. We are driven, therefore, to seek further for a cause for the entry of infection into the peri-anal tissues.

Chiari in 1878, followed closely by Hermann & Desfosses in 1880, first described structures which are referred to as the anal glands, or anal ducts. They showed that glands ramified in the internal sphincter and submucosa of the mid-anal region, finally to discharge into the anal crypts. They suggested that the glands acted as a channel through which infection could reach the peri-anal tissues and set up an abscess. In 1914 Johnson made a study of these glands. He noted the presence of lymphoid tissue around the ducts in some cases and he referred to this tissue as the anal tonsil. Further observations supporting the glands as a cause of fistula were made by Lockhart-Mummery (1929), Gordon-Watson & Dodd (1935), Kratzer & Dockerty (1947) and Hill *et al.* (1943). On clinical evidence, Eisenhammer (1958) concluded that these glands were the cause of most fistulæ.

A previous study (Parks 1961), showed that anal glands are found in most, if not all, individuals. They are complicated structures ramifying in the submucosa of the middle and lower parts of the anal canal (Fig 1). In at least half the cases, the

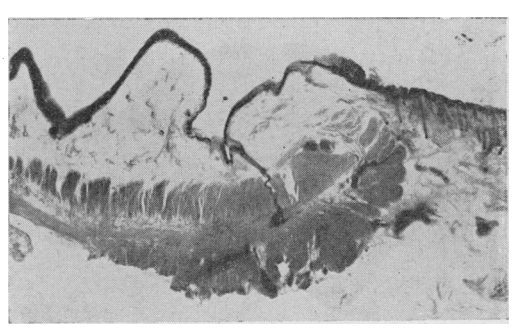

Fig 1 *Thick section of anal wall showing an anal gland penetrating the internal sphincter and terminating in the longitudinal layer* × 2·3

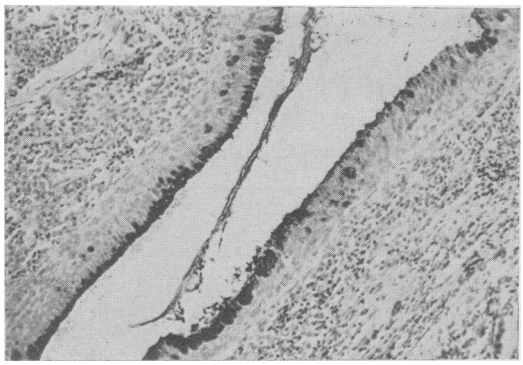

Fig 2 *Stratified epithelium of anal gland with a columnar surface layer, the cells of which secrete mucus* × 70

glands penetrate the internal sphincter, extending into the longitudinal layer. No branches extended beyond the longitudinal layer into the external sphincter. The diameter of the ducts is small, only 30–40μ, but in some specimens small cysts were found up to 0·5 cm in diameter. Histologically they are lined by stratified mucus-secreting columnar epithelium, but occasionally part is lined by squamous epithelium, presumably the result of metaplasia (Fig 2).

Normal anal glands are so minute that it is difficult to believe that infection in them is the cause of fistula. It was decided to examine pathological material obtained at operation to see if further evidence in favour of their role in pathogenesis could be obtained (Parks 1961). In 22 of 30 cases evidence that the anal glands play a causative part was found. More interesting than the statistics was the anatomy of the glands in some of the fistula specimens. They were not the simple structures found in the anatomical investigation. In 8, macroscopic cystic cavities were found, some being up to a centimetre in diameter (Fig 3). The cysts were usually full of pus, and some contained vegetable matter suggesting that fæcal material had entered the ducts. An inflammatory reaction usually surrounded the

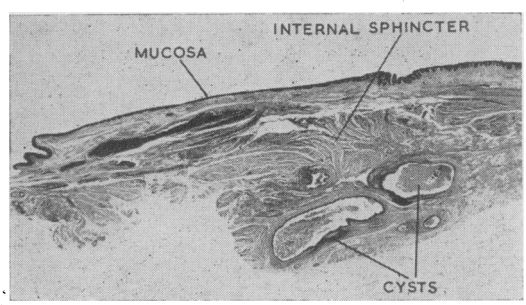

Fig 3 *Section of a fistula specimen showing infected cystic spaces deep to the internal sphincter* × 2·3

glandular spaces and in 6 cases lymphoid tissue was present. It was also found that the infected cystic spaces were always deep to the internal sphincter. Often the duct penetrating the internal sphincter showed no evidence of infection but its dilated segment in the intersphincteric layer was the site of an abscess. This is interesting because in 1958 Eisenhammer suggested that the cause of fistula was to be found in the intersphincteric zone deep to the internal sphincter and he suggested that the anal glands were the agents involved. In 22 specimens, the anal gland crossed the internal sphincter and terminated in an abscess in the longitudinal layer. This is pathological confirmation of Eisenhammer's views.

Any hypothesis of the cause of fistula-in-ano must explain the high incidence with which the internal opening is found in the anterior or posterior commissures of the anal canal. Anal glands are distributed equally around the anal circumference. If fistula is due to a congenital abnormality of the glands, such as a cyst, it is difficult to understand why this should occupy one particular site. In children under 5 the site of the internal opening is distributed at random around the anal wall and is not found with greater frequency in the anterior or posterior positions (Venturo 1953). It is therefore probable that in children the cause of fistula is a congenital cystic abnormality of the anal glands. In adults some explanation has to be found for the more frequent occurrence of cystic glands in the anterior and posterior positions. The commonest lesion in these sites is anal fissure. It is possible that inflammation from a fissure either infects an anal gland or so seals off the ducts discharging into the crypts that the deep part of the gland becomes cystic. Cysts have been found anatomically in the submucosa and also deep to the internal sphincter in the longitudinal layer. If a cyst in the latter site becomes infected an abscess will form. It may discharge into the anal canal but if this path is impeded by the internal sphincter, the obvious course for pus to take, on anatomical grounds, would be to follow the longitudinal layer down to the anal verge. This is the commonest site for fistula; about 70% occur here. Depending on circumstances, the spread of pus could be in any direction; if it penetrated the external sphincter an ischiorectal abscess would be produced; if it spread upwards in the longitudinal layer a pararectal abscess could be formed (Fig 4).

This investigation shows that in two-thirds of the patients the cause of fistula was an intersphincteric abscess originating in an anal gland. The hypothesis is advanced that the intersphincteric abscess is the primary cause of fistula in most

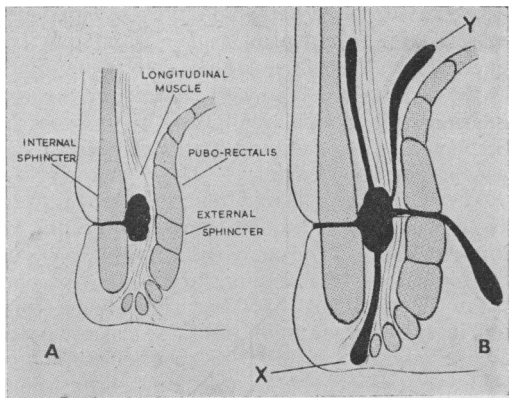

Fig 4 A, *diagram to illustrate the hypothesis that the primary infection occurs in an anal gland situated in the longitudinal layer.* B, *diagram showing the various possible routes of spread of infection. X is the commonest, Y the most rare pathway taken*

cases, though not necessarily all. On this theory any tracts extending from the intersphincteric abscess are secondary and dependent upon the primary abscess; if true, it has significance from the point of view of surgical treatment. Anatomically, anal glands have never been found to spread beyond the longitudinal layer into the external sphincter muscles. Clinically, there is evidence that this does occur as a congenital abnormality on rare occasions. Such congenitally epithelial-lined tracks could be a source of difficulty in surgical treatment, especially in the method advocated in a previous paper (Parks 1961).

Thus far, the non-specific fistula has been considered. Of the other varieties tuberculosis is perhaps the commonest cause. The anal canal is affected by this disease more than any part of the alimentary tract. This may be because the mycobacterium has a well-known affinity for lymphoid tissue. There is enough lymphoid tissue in the anal canal for Johnson (1914) to refer to it as the anal tonsil. There is other evidence that the anal canal has a capacity for forming lymphoid tissue. It is in the region of the upper anal canal, just above the line of the anal valves, that the so-called benign lymphoma is most commonly found (Cornes *et al.* 1961); these tumours are sometimes multiple and are almost certainly of inflammatory origin.

In the lower half of the anal canal lymphoid tissue is found around the ducts of the anal glands, particularly that portion lying deep to the internal sphincter in the intersphincteric layer. Lymph follicles have been found in specimens including those from stillbirths and these are less likely to be secondary to infection than a primary constituent of the region. In about 20% of our fistula cases lymphoid tissue was well developed, often surrounding cystic cavities in the intersphincteric zone. When a tuberculous fistula is explored a cavity is usually found deep to the internal sphincter containing tuberculous granulation tissue and it is tempting to suppose that the organisms primarily infect the lymphoid tissue. No anal gland epithelium has been found in the tuberculous granulation tissue, but in two cases an anal gland was seen penetrating the internal sphincter in the direction of the tuberculous tissue; perhaps the disease destroyed the deep part of the gland. It is not known whether the tuberculous organisms infect this region from the blood stream or through the gut lumen. Tuberculous fistulæ frequently affect the anterior and posterior commissures as in the non-specific variety and it is difficult to account for this; possibly tubercle bacilli have an affinity for a gland or its surrounding lymphoid tissue which has already been infected by a non-specific organism.

Patients with Crohn's disease commonly develop anal fistula. Histologically, such a fistula often contains the sarcoid-like granulomata of Crohn's disease (Morson & Lockhart-Mummery 1959). Again, an explanation must be sought for the occurrence of the disease at this site. It would appear that Crohn's disease particularly affects regions which contain aggregations of lymphoid tissue (Lockhart-Mummery & Morson 1960), for the formation of lymphoid granulomata is a consistent histological feature. It is tempting to suggest that Crohn's disease affects the anal region because of the aggregations of lymphoid tissue in the intersphincteric zone.

One rare variety of anal fistula is mentioned for completeness. This is the congenital fistula, which may be single or multiple, and has nothing to do with the anal glands. It is lined by rectal mucosa in its upper part and squamous epithelium in its lower portion. It is probably a duplication of the lower end of the hind-gut and its importance lies in the fact that malignant change is prone to occur with the formation of a mucus-secreting or colloid carcinoma within the fistulous track. A series of such cases were described by Dukes & Galvin in 1956, and several have been seen since at St Mark's Hospital.

It is important that tissue from a fistulous track should be examined histologically in every case of anal fistula. Usually the histopathologist will recognize a non-specific chronic inflammatory infiltration, with or without an accompanying foreign-body giant cell reaction. The biopsy examination confirms the non-specific

inflammation and excludes the presence of a sarcoid-like reaction of Crohn's disease, tuberculosis or a carcinoma in a congenital fistula.

Since the research of Chiari (1878) and Hermann & Desfosses (1880), evidence has accumulated that infection in anal glands plays a role in the pathogenesis of anal fistula, but this knowledge has not yet greatly influenced surgical treatment. Our knowledge of pathology has led to advances in surgical treatment of diseases of the colon, rectum and anus, particularly cancer. At present anal fistulæ do not cause death, but they cause considerable morbidity. If improvements in treatment are to be achieved they must be based on a more complete understanding of pathogenesis. This has been outlined (Parks 1961).

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Mr Henry Thompson (*London*)

The Orthodox Conception of Fistula-in-ano and its Treatment

A fistula is an abnormal communication between a hollow viscus and the surface of the body, or between one hollow viscus and another. A sinus is a granulating track leading from a deep-seated focus of suppuration to the surface. These definitions are difficult to apply precisely to the anorectum; a fistula-in-ano is an abnormal track, usually lined with granulation tissue, leading from the anal canal to the skin, but we also speak of blind internal and external fistulæ – terms which contradict our definitions.

The ætiology of fistula-in-ano depends on the understanding of the development of abscesses around the anus and rectum.

There is a group with an obvious cause recognized from the history and physical signs and a group with no clue to their origin. For years it has been known that suppuration can originate between the external and internal sphincter muscles (the intermuscular plane) and that the internal openings of fistulaæ are commonly found at the dentate line. The study of anal crypts and intermuscular glands, exemplified by the work of Stephen Eisenhammer and Alan Parks, fills in the gaps in our knowledge.

The healing of a fistula, described by Milligan as third intention healing, may fail for one of three reasons: (1) The contents of the hollow viscus draining down the fistulous track may keep it open. (2) Epithelial surfaces may join without intervening granulations. Classical examples are vesicovaginal fistulæ, gastrocolic fistulæ and some vesicocolic fistulæ. (3) The disease which has caused the fistula may prevent healing.

All must be familiar with the history of a fistula-in-ano which, judged by the cessation of discharge from the external opening, heals for a variable period, only to break down and discharge following a bout of pain and fever. The daily passage of fæces and mucus over the internal opening reinfects the fistulous tract and results in recurrence and persistence of the fistula. This may be too simple. Are there other factors preventing healing besides the continual introduction of infected mucus through the internal opening? The presence of transitional epithelium of an anal gland lining the innermost part of the track may be the answer. We have, however, to extend our intermuscular gland from internal openings at the dentate line but also up to the higher internal openings.

The track of a fistula-in-ano is lined by granulation tissue; in chronic fistulæ, squamous epithelium may line the track in the region of the external opening and transitional epithelium from an intermuscular gland may line it at the internal opening. Thus the second factor of union of the epithelial edges may be a cause of a fistula failing to heal.

Lastly, a fistula may persist because of associated disease – foreign bodies, tuberculosis, actinomycosis, Crohn's disease, ulcerative colitis, diverticulitis, carcinoma, endometriosis, infected anal intermuscular glands, syphilis and diabetes. This list, together with the health of the patient, should be considered if healing is delayed after apparently adequate treatment.

Classification

A past error in recognition between the lower end of the internal sphincter and the subcutaneous external sphincter undermined my belief and confidence in the practical value of the exact anatomical classification of fistulæ. There are fistulous tracks passing through the internal sphincter muscle alone and tracks passing through both external and internal sphincter muscles, but there are also fistulæ where I am uncertain which tissues

are traversed. The low anal fistulæ, previously described at St Mark's Hospital as passing between the subcutaneous external sphincter and the lower border of the internal sphincter, I now believe pass through the lower part of the internal sphincter or through the internal sphincter and a small part of the external sphincter. As regards orthodox treatment the issue has no *practical* importance.

The termination of the intestine is that of modified intestinal wall surrounded by voluntary muscle. Without the voluntary muscle the intestinal contents would pass through the anus involuntarily or without control. Does the thickened end of the circular muscle of the bowel (the internal sphincter) play any part in continence? Continence depends on the voluntary external sphincter. If this is true the critical determination to be made is the relationship of the fistulous track and the internal opening to the voluntary sphincter muscle. To decide this, the extent of the external sphincter must be recognized and fortunately this can be done by sight and touch.

I recognize two groups of fistulæ: (1) Complex, or fistulæ difficult to deal with (5%). (2) Simple, or fistulæ which are easy (95%).

The difficult fistulæ are those where the internal opening is above the anorectal ring (the so-called pelvirectal fistulæ) and those where the fistulous track is in relationship to three-quarters of the circumference of the sphincter muscles, although the internal opening may be relatively low placed. These are known as ischiorectal or horseshoe fistulæ.

There is no clinical difficulty in distinguishing a simple from a complex fistula. The track of a simple fistula can be palpated as a thick, fibrous cord running from the external opening towards the anal canal. A probe passed through the track of such a fistula lies transversely to the axis of the rectum. In the difficult type of fistula no fistulous track can be palpated subcutaneously and the probe passes parallel to the axis of the rectum upwards for a distance of 6 or 7 cm to the apex of the ischiorectal fossa. Digital examination of the rectum reveals a horseshoe induration just below the level of the anorectal ring.

Treatment

The orthodox treatment of a fistula-in-ano is to lay open the fistulous track in its entire length from internal to external opening or, in terms of wound healing, to convert a fistulous track that has failed to heal by third intention into a flat wound which will heal by second intention. If this alone were done in simple fistulæ and the patient isolated on a desert island, with no treatment except a swim in the sea night and morning, the fistula would heal.

Wounds of the anal canal deserve special consideration. Take, for instance, a two-inch wound through the skin and subcutaneous tissues of the surface of the body and a wound of similar length and depth through the anal margin, such as is made during an internal anal sphincterotomy or a Miles' pectenotomy. We would be surprised if the surface wound in the skin were not healed in ten to fourteen days, but I agree with the late Ernest Miles that the average healing of pectenotomy wounds is nineteen to twenty-one days. This difference in healing time is not surprising because a wound in the anal canal is kept warm and moist instead of cool and dry and at least once a day the wound edges are stretched at defæcation and fæces soil the surface. A wound in the anal canal is analogous to an imperfectly drained abscess – improve the drainage and healing will be accelerated. Because of these factors a wound of the anal canal takes twice as long to heal as a surface wound. If a surface wound is necessary to drain the wound of the anal canal then it should be twice the area of the anal wound so as to maintain drainage while the anal wound heals. Such is the basis of Salmon's backcut outwards from the external fistulous opening and the sacrifice of skin.

It is often said that if a fistula is only laid open its edges fall together and the fistula reforms and that persistence or recurrence of a fistula is due to this. The trimming of the edges of a fistula wound and Salmon's backcut are measures taken to quicken and improve the healing of the wound and not to prevent the recurrence of the fistula as stated. This misconception requires correction.

The conversion of a simple fistula into a racquet-shaped wound is simple; the difficult fistula is a different matter. To lay open a horseshoe fistula requires an incision in perianal skin involving almost the entire circumference of the anus. To flatten the wound adequately requires the sacrifice of a large amount of skin and the resulting wound may take eight to ten weeks to heal. The fistula whose internal opening lies above the anorectal ring cannot be treated by the classical method without resulting incontinence and it is the fear of dividing too much of the sphincter muscle and rendering the patient incontinent that is often responsible for the inadequate treatment of fistulæ. This is borne out by the observation that in many unsuccessful fistula operations there is no wound through the anal margin into the anal canal.

The orthodox treatment consists of the incision or laying open of a fistula from the external to the internal opening. There are, however, a variety of treatments in vogue which claim better and quicker healing than this: (1) Excision of the fistulous track. (2) Excision of the fistulous track, repair of the divided sphincters and primary suture

(Paris). (3) Division of the fistula with a diathermy current passed through a wire loop (diathermic syringotomy, Simonetti). (4) Excision of a fistula and primary skin grafting.

Discussing these methods with Continental colleagues, I have been impressed by the different meanings of the word 'healed'. It varies from a complete and final epithelialization to the 'setting' of a wound which no longer is a major discomfort to the patient, and the healing time of a fistula operation varies according to the individual surgeon's definition of 'healed'. Indeed, unless a daily examination of the wound is made it is not possible to be exact in the matter of days. Absence of discharge, however, is a reliable guide that granulations are finally epithelialized.

I would repeat that the wound from a simple incision of tissues, including the lower end of the internal sphincter, at the anal margin takes, on an average, nineteen to twenty-one days to heal and I do not believe that if excision of tissue is added to this, healing in the strictest sense can be accelerated – on the contrary, I would expect it to be retarded.

The successful management of a fistula demands a knowledge of its ætiology and recognition of its relation to the sphincter muscles. With this knowledge, healing of a simple fistula by orthodox methods presents no problems. Complex fistulæ may tax the patient's endurance and the surgeon's skill to the utmost.

Mr R C Bennett
(*University Department of Surgery, General Infirmary, Leeds*)

A Review of the Results of Orthodox Treatment for Anal Fistulæ

Having read most of the papers on fistula-in-ano, I realized that there was a lack of information regarding the results. It was decided to follow up all cases treated during the past six years, with personal interrogation and examination. The points particularly noted were: (1) The length of time required for complete healing and the time off work. (2) The incidence of recurrence or incomplete healing. (3) The functional results.

The number of patients recorded as having been treated primarily for fistula-in-ano from August 1955 to August 1961 was 129. Of these, 118 have been personally examined: 114 were treated by laying open the fistulous track; the remaining 4 had internal openings above the anorectal ring – 2 were not given any treatment and 2 were eventually submitted to rectal excision. Eleven cases were not interviewed; 6 of these had either died or moved elsewhere and the other 5 were untraceable.

Length of follow up: All have been followed up for more than three months, 28 cases for between three and twelve months and the other 90 for more than a year, half of these for over three years.

Age and sex incidence: Males were twice as common as females. The greatest incidence was in the fifth decade, the remainder being evenly distributed on either side in diminishing numbers.

Classification: The majority of low anal fistulæ traversed the internal sphincter and opened at the level of the anal valves – not at the anal intermuscular depression as reported by Milligan & Morgan (1934). All anal fistulæ with internal openings above the valves were classified as high anal. Low anal fistulæ were commonest with an incidence of 62%. The incidence of 25% high anal fistulæ is appreciably higher than the 15% reported by Milligan (1943). Anorectal, subcutaneous and submucous fistulæ were less common.

Horseshoe fistulæ were present in 19%; individually they belong to one or other of the main types, principally high anal, but because of their difficulties and the incidence of post-operative defects, they have been grouped together for comparison.

Our series showed internal openings in 63%. None was found in 28% and in 9% the notes were insufficient.

Associated conditions: Fourteen had fissures, and only 4 had proctitis or colitis. Here only those presenting with fistulæ, and being treated for this, are considered. Others complicating ulcerative colitis or Crohn's disease have been treated but are classified under these conditions. One person had a proven tuberculous fistula – a smaller incidence than the 15% reported by Gabriel (1921).

Time off work: The time off work with its attendant dressings, post-operative examinations, and economic loss, varied according to the type and complexity of the fistula. An average figure is of little use unless it is restricted to fistulæ of similar type. It increased from about five weeks in low anal fistulæ to seventeen weeks in double horseshoe fistulæ. These figures are only a guide, for the number of horseshoe fistulæ is small, but they do show the magnitude of the economic loss in complicated fistulæ.

Recurrences or incomplete healing occurred in 10 patients. Analysis of these shows that:

(*a*) One fistula recurred in a man with ulcerative colitis, which has since necessitated procto-colectomy.
(*b*) Three showed minor defects attributable to incomplete healing. Small granulations were noted in the scar, either alone or with a small pit. There were no lengthy tracks or internal openings. Discharge was not a feature and recurrent abscesses did not occur. Further treatment has not seemed justified, and when suggested in one case was refused.
(*c*) Three had left hospital but their unhealed wounds were supervised at the Rectal Clinic. Excess discharge was noted; they were readmitted and further fistulous tracks were opened. All have satisfactory results.
(*d*) Three had true late recurrences. An elderly man, treated two years previously for a high anal fistula, refused further operation and has remained virtually symptom free for four years. The other 2 had further operations with satisfactory results to date; one was originally low anal and the other submucous. These are the only cases in which the operation can be said to have failed.

When the types of fistulæ in these 10 cases are considered and their frequency expressed as a percentage of their overall incidence, it is found that only 4% of low anal cases have trouble compared with 19% of high anal and 24% of horseshoe fistulæ. The numbers are small, and the percentages are only a guide, but they confirm clinical impressions that trouble is most likely to be experienced in high anal and horseshoe fistulæ, and indicate that this can be expected in about 1 in every 5.

Functional Results

While appreciating that complete incontinence is avoided if the anorectal ring is preserved, we realized that minor defects in anal control occur after less radical division of the sphincters. We made enquiries to elucidate the frequency of such imperfections and to relate them to the type of fistula. If the patients admitted to the repeated inadvertent passage of flatus or fæces, often embarrassing and different from before operation, they were regarded as having defective anal control. Others, while not admitting inadvertent passage of fæces, did notice frequent soiling of their underclothes. This was usually a brown stain and more suggestive of a fæcal leak than a purulent discharge, and was annoying and embarrassing.

Of the 114 patients questioned, 12% had inadequate control of fæces, 16% had inadequate control of flatus and 24% had frequent soiling of their underclothes. In all 36% of patients complained of one or more defects.

A series of 100 'normal' people were asked similar questions. Of these, 3% had inadequate control of fæces, 10% had inadequate control of flatus and 2% had frequent soiling of their underclothes; 10% had one or more of these defects.

All patients mentioned in this review who had functional defects claimed they were a result of the operation.

With all defects, the incidence of poor control increases as we pass from the low anal fistula to the high anal and horseshoe varieties. The incidence of patients with one or more of these defects also increases and reaches about 55% in horseshoe fistulæ. Perhaps division of one or both inferior hæmorrhoidal nerves plays a part.

Anorectal fistulæ are not considered as a group because of the small number, and single and double horseshoe fistulæ are grouped together for the same reason.

In the majority, these defects were relatively mild, but in 12 of the 114 they were present daily, and 2 people wore pads for protection, both after low anal fistulæ.

In conclusion, I think that most patients are satisfied with their operation, and that in the majority it can be relied upon to produce healing of the fistula without recurrence. Time off work may be considerable in the complicated cases. Functional defects, even after preservation of the anorectal ring, are more common and disturbing than generally realized, and are most frequent in the high anal and horseshoe fistulæ. It is a poor consolation for the fastidious patient who, after seventeen weeks off work for treatment of his horseshoe fistula, finds that his underclothes are stained brown instead of yellow even though the fistula is healed.

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Mr M Ellis
(*General Infirmary, Leeds*)

Recurrence of Infection Following Treatment of Anorectal Abscesses by Primary Suture

Two years ago, I described the treatment of anorectal abscesses by incision and primary suture (Ellis 1960): thirty to sixty minutes after a large injection of penicillin and streptomycin the abscess is incised. All loculi are broken down; the contents, pus and slough, are evacuated. The wound is closed with deep sutures obliterating all dead space. The sutures are removed on the fourth day and the patient is discharged healed

around the seventh day. The immediate results of 200 consecutive cases were then described. I now present another 200 cases. A follow-up was carried out on 146, the last patient having had an operation nine months previously. The results of treatment are presented from a different angle to the anatomical one presented by Mr Parks in the ætiology of anorectal abscesses and fistula-in-ano.

The 124 primary cases are considered first. In 115 cases, healing was uneventful and the patients were discharged about one week later and have had no further infection. Five cases, i.e. 4%, developed a fistula-in-ano some weeks later. Four cases developed another abscess and were treated by incision and primary suture and have had no recurrence since.

In the 22 cases who had had recurrent abscesses, 19 had had one abscess previously and 3 had had two or more. In the 19 cases of a second abscess, 13 had been treated by us by incision and primary suture and 6 had been treated elsewhere by cutting skin edges, packing and healing by granulation. Of the 13 cases treated by primary suture, one had had his first operation six years before, with no trouble in the interim. Four presented with an abscess on the opposite side. Thus 8 cases had a recurrent abscess on the same site following primary suture compared with 6 cases treated by radical drainage and healing by granulation. Healing by granulation is illustrated by 2 of the 3 patients who had had two previous abscesses. (The third patient had no operations for his multiple previous abscesses.) In each of these two patients, the first abscess had been treated by primary suture and the second abscess had had radical drainage. In the first patient, dressings were carried out for six weeks after the operation and in the second dressings were required for fourteen weeks and after this a third abscess developed three and a half months later. He developed a fourth abscess on the scar some weeks later, which discharged spontaneously and has remained healed since.

Excluding recurrence of abscess, it can be seen from these two series that 5 fistulæ developed from 124 cases of first infection and 4 fistulæ developed from 22 second infections. Of the 54 cases not followed up, none is among the patients reviewed by Mr Bennett. Of the 9 cases of fistula, 4 were sent by their doctors to other surgeons and the remaining 5 were treated by primary excision and suture of the fistulous track.

The histological reports on the tissues excised are of interest. In 3 the tract is described as being lined by squamous epithelium and in one it was lined by granulation tissue. In Mr Parks' paper (Parks 1961) the histologists reported finding glandular epithelium in all specimens.

Care was taken to distinguish between the peri-anal abscess and the ischiorectal abscess. In the former there was always a roof of fascial tissue separating the abscess cavity from the ischiorectal fossa. In the ischiorectal abscess a breach in this fascia could be felt at operation and the abscess extended above it. There were 93 peri-anal abscesses from which two fistulæ developed and 53 ischiorectal abscesses after which 7 fistulæ remained.

The organisms in the original abscess show some relation to the occurrence of a fistula. In the 9 cases where a fistula followed, *E. coli* or other bowel organisms were present in the original abscess. In peri-anal abscesses 60% were due to staphylococcal infection and 40% due to *E. coli*, &c. In the ischiorectal abscesses, 55% had *E. coli*. Since *E. coli* were found in the abscesses preceding the fistulæ, we should expect less fistula from the peri-anal abscesses. If, as Mr Parks suggests, infection by the anal gland is the cause of all anorectal abscesses, the staphylococcus should rarely be the infecting organism.

One factor in fistula after abscess may be a connexion between the abscess cavity and the anal canal demonstrable at operation, with pus in the anal canal. Five fistulæ had such a connection. In 4 other cases, where a connexion was seen, primary suture, obliterating the cavity and opposing the rent in the anal epithelium, was not followed by fistulæ.

Conclusion: In 146 cases of anorectal abscesses treated by incision and primary suture, 14 cases showed subsequent infection, 5 had recurrent abscesses and 9 fistulæ-in-ano. Of the fistulæ-in-ano, 5 were in 124 primary abscesses and 4 were in 22 secondary abscesses. Seven fistulæ followed 53 ischiorectal abscesses and 2 followed 93 peri-anal abscesses. All abscesses followed by fistula formation contained *E. coli*.

REFERENCES

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