INFLAMMATION OF THE SALIVARY GLANDS WITH PARTICULAR REFERENCE TO CHRONIC AND RECURRENT PAROTITIS

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by

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THE CLINICAL FEATURES of inflammation of the salivary glands were known to the Greeks, and we find in the Hippocratic writings an accurate account of an epidemic of mumps, including the complication of orchitis, and a clear recognition of its distinction from suppurative parotitis. To get things in perspective, however, we should remember that the concept of externally secreting glands did not at that time exist, and would not exist for the salivary glands for another 2,000 years, until Wharton and Stenson in the 17th century discovered the ducts now known by their names. Mediaeval ideas on parotitis were naturally conditioned by humoral concepts of disease, and are exemplified by such statements as that of Paul of Aegina, who in the 7th century referred to " an affection of the glands about the ear, being sometimes occasioned by humours of the head which are impacted in it, and sometimes by those collected from the rest of the body during the crisis of a fever ". As late as the 16th century, Ambroise Paré called the parotid glands " the emunctories of the brain "; " for these, because they are large and spongy ", he continued, " are fit to receive any excrement thereof ". John Hunter was a man of his time in his support of the doctrine of " sympathy " as an important factor in disease processes, and by this doctrine the complication of orchitis in mumps was explained as resulting from a peculiar sympathy between the testicle and the parotid. In the earlier years of post-Listerian surgery, the doctrine of sympathy was resuscitated to explain in a reverse direction the not infrequent development of acute parotitis as a complication of ovarian cystectomy, one of the earliest intra-abdominal operations to become established. We now all know that post-operative parotitis is due to an ascending infection along the duct as a result of dehydration and inadequate oral hygiene, and that by proper attention to these factors it has been almost abolished from surgery.

To-day, the main interest of surgeons in inflammation of the salivary glands is concentrated on the often difficult and troublesome conditions of chronic and recurrent parotitis, and it is with these that I shall principally deal in this lecture, though I shall also refer briefly and chiefly for purposes of comparison to inflammation of the sub-mandibular glands. The particular contribution of the surgeon to this problem in recent years

has developed out of the modern attack on parotid tumours. Techniques have been worked out and standardized for removing satisfactorily with preservation of the facial nerve parotid tumours of all except the highest degrees of malignancy, and these techniques have been applied successfully to cases of chronic and recurrent parotitis which have failed to respond to less radical measures. As has often happened before, advances in surgical treatment have led to increased understanding of the disease processes involved, partly because of a general stimulation of interest, and partly by provision of material for pathological study. Apart from the direct clinical approach, advances in other branches of medical science have in recent years helped in our understanding of inflammation of the salivary glands. The great increase in depth of our knowledge of the physiology of salivary secretion, a scholarly survey of which has recently been given by Burgen and Emmelin (1961), has stimulated us and others

TABLE I

CLASSIFICATION OF 120 CASES OF CHRONIC AND RECURRENT PAROTITIS SEEN FROM 1952 TO AUGUST 1964

Obstructive cause	62 cases
Calculi	40 (15 parotidectomies)
Strictures	15—unilateral 10 —bilateral 5 (4 parotidectomies)
Various	7
" Punctate sialectasis—sicca syndrome " ..	40 cases (19/21 parotidectomies)
Miscellaneous	18 cases (8 parotidectomies)

(In the " punctate sialectasis—sicca syndrome " group, 21 parotidectomies were done on 19 patients.)

to study the functional aspects of salivary gland disease, which had previously been largely neglected. Again, the concept of auto-immunity, which has shown such exciting and fruitful developments since its importance in thyroid disease was first demonstrated in 1956, in this country by Roitt and his colleagues (Roitt *et al.*, 1956) and in the United States by Rose and Witebsky (1956), has been invoked as a possible explanation of one common but obscure type of inflammation of the salivary glands. Finally there are hints that a study of the possible influence of viral factors in these conditions might be rewarding.

The clinical material for my lecture consists of 120 cases of chronic and recurrent parotitis seen during the last 13 years, predominantly in the Department of Surgical Studies at Middlesex, but including also a few cases seen in private practice (Table I). There are obvious gross selective factors in this material, the first resulting from our interest in parotid disease, as a result of which we see, for example, nearly as many cases of parotid calculi as of sub-mandibular calculi. There is also an internal

selective factor in that up till about two years ago we did not keep in our nosological index cases of parotitis seen only in outpatients, which have consequently not been included in this table except recently. As a result, the material has a bias towards the more severe type of case, and this is shown by the relatively large proportion of cases coming to parotidectomy, which was performed 48 times in the 120 cases. You will also see from this table that the two main groups are, firstly, parotitis due to obstruction, with calculi the commonest cause of the obstruction, and secondly the group I have termed " punctate sialectasis " or " the sicca syndrome ". The miscellaneous group is a heterogeneous collection, some examples from which I shall be giving later.

In Table II I have classified the 48 of the 120 cases of Table I which we have seen during the last two years. Since they include all outpatients as well as inpatients, they eliminate some of the bias towards the more

TABLE II

CLASSIFICATION OF THE 48 CASES OF CHRONIC AND RECURRENT PAROTITIS SEEN DURING 1963 AND 1964 UP TILL 31ST AUGUST

Obstructive cause	20 cases
Calculi	8
Strictures	11—unilateral 8 —bilateral 3
Various	1
" Punctate sialectasis—sicca syndrome " ..	24 cases
Miscellaneous	5 cases

(One patient had both punctate sialectasis and calculi and is therefore included under both headings.)

severe case shown in Table I. The most striking differences between the two tables are that the sicca syndrome replaces obstruction as the most common cause of parotitis, and that strictures are a more common cause of obstruction than calculi. This would suggest that at any rate on a short view many patients with strictures and the sicca syndrome are reasonably well controlled without admission to hospital.

The two special investigations which best help to determine the nature of parotitis are radiography, particularly sialography, and studies of secretory function. Sialography is a well established procedure, and I would only emphasize once again the harm that may result from injecting large quantities of the radio-opaque medium, particularly in the parotid. Ranger (1957) has shown that even normal parotid ducts easily rupture, and Thackray (1955) has described and illustrated the histological features of the severe iatrogenic parotitis that used frequently to result from the extravasation of the medium around the weakened ducts of disease. Pattinson (1957) advises that only 0.5 ml. should be injected in the first instance, and more injected only if the duct system is shown to be dilated

and inadequately filled. The secretory function test that we use was worked out by Curry (1963), and has been the subject of a recent paper (Curry and Patey, 1964). Briefly, in an adult 5 mgm. of pilocarpine are injected intravenously, and the secretion from each parotid collected separately for five minutes following the injection. The amount secreted varies widely in different individuals, but is constant for the same individual, and is the same on both sides. In 31 normal patients the amounts varied between 3 ml. and 13 ml. except for two patients in whom it was between 17 and 18 ml. Amounts below 3 ml. may therefore be regarded as indicating deficient secretory function, and above 14 ml. as unusually high. In unilateral disease, the unaffected side provides a normal control, and changes in amounts of secretion in repeated tests are also significant. In bilateral disease, however, we have

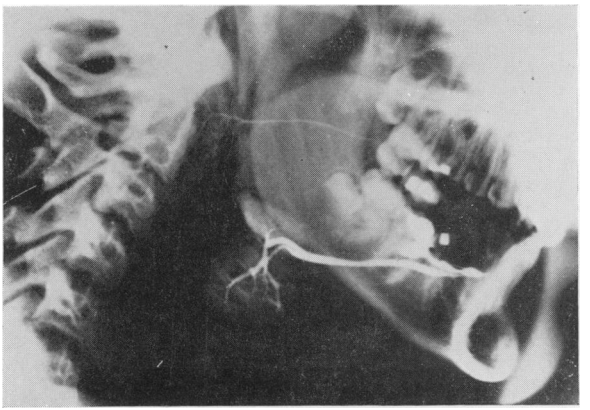

Fig. 1. Sialograms of parotid and sub-mandibular glands to show relative size of ducts.

no means of determining the normal for the particular patient, though the figures are significant if as often happens they are well outside the normal range. The test is also useful in these cases in showing the difference in the degree of functional impairment on the two sides.

Before analysing our material in greater detail, I would like to say a word on infection. Infection may occur in all types of disease under consideration and, if pyogenic organisms are concerned, the infection may be the most important element in the clinical picture. But the salivary glands possess such an efficient mechanism against ascending infection, chiefly through the regular flushing out of their ducts with saliva but partly also through the presence of weakly anti-bacterial substances in the saliva, that one should always search for the primary cause of the break-down of this protective mechanism. Both the main groups of our series

have such causative factors, the first in obstruction to the salivary flow, the second in a breakdown in salivary secretory function.

Coming to the detailed analysis, the frequency of parotid calculi is generally under-estimated, and their presence often missed. During the 13 years of the present investigation, the total number of all sub-mandibular calculi admitted to all departments of the Middlesex Hospital was 137, and of parotid calculi 44. This gives a relative proportion of three sub-mandibular calculi to one parotid calculus. While there is still some degree of fallacy due to selection, this is the nearest we can get to a relative incidence figure. There are several reasons for the failure to diagnose the presence of parotid calculi. Because the parotid duct is much

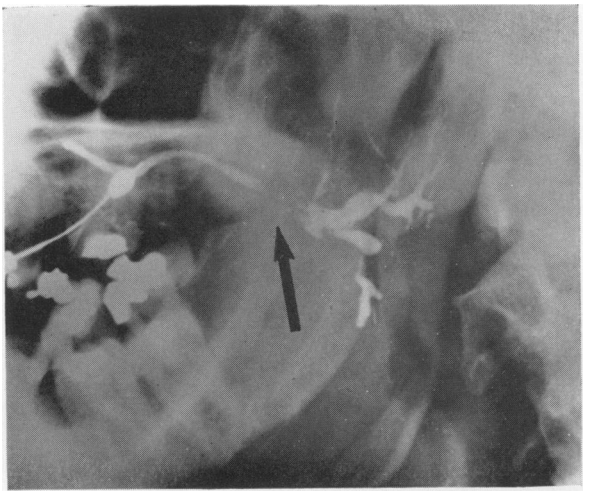

Fig. 2. Sialogram of patient with small parotid calculus impacted at posterior end of main duct showing as negative shadow and dilatation of duct system behind.

narrower than the sub-mandibular duct (Fig. 1), parotid calculi tend to cause obstruction when they are much smaller than sub-mandibular calculi, and a small calculus may be difficult or impossible to see on a plain X-ray against the bones and soft tissues of the face. A calculus may show up as a negative shadow on sialography (Fig. 2). But again, if there is a very dilated duct system, a small calculus may not give a negative shadow against the dense mass of radio-opaque medium, a phenomenon similar to that sometimes met with in operative cholangiography. One of the two common sites of impaction is where the duct turns inwards towards the mouth at the anterior border of the masseter. At this point, superposition may prevent a negative shadow being seen, a similar difficulty to that well known in interpreting X-ray pictures of the pelvi-rectal junction (Fig. 3). But perhaps the principal source of error is that the diag-

nosis is often not suspected because the symptoms of parotid calculi usually suggest inflammation rather than obstruction, being quite different in this respect from obstruction in the sub-mandibular gland. In the latter, however long the obstruction has been present, salivary function usually persists, and there is the typical story of swelling of the gland after meals. In the parotid, secretion is usually diminished or suppressed, and as a result infection supervenes. While similar symptoms to those of stone in the submandibular gland may occur, the typical story is of intermittent attacks of pain and swelling of the parotid gland, often lasting for many days, and without any particular exacerbation in relation to food.

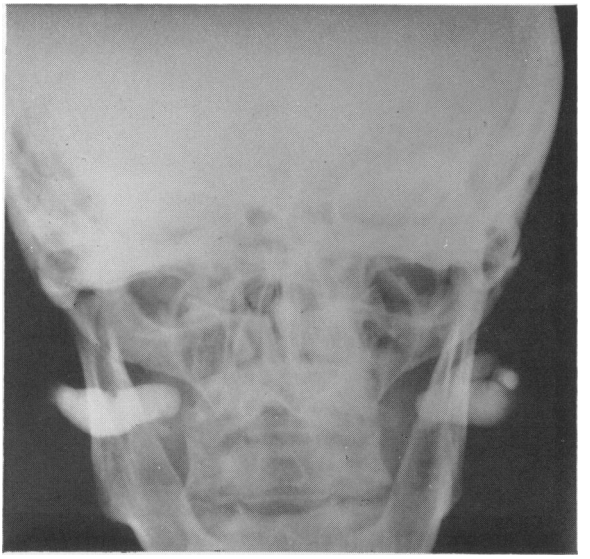

Fig. 3. Bilateral parotid sialograms to show how ducts curve inwards towards the mouth so that negative shadows may be obscured by superposition.

Parotid calculi may be single or multiple, or there may be a history of recurrent passage of calculi, similar to that not infrequently met with in cases of renal calculi. Apart from cases of multiple, usually symptomless, microcalculi occasionally found on routine X-ray examination, I have not seen a case of bilateral parotid calculi, but in one case there was a previous history of removal of a sub-mandibular gland for calculi. The calculus may pass almost as soon as it causes symptoms, or it may remain impacted indefinitely, the two common sites being at the sigmoid bend of the duct into the mouth, as already mentioned, and at the hilum of the gland, which corresponds to a point a centimetre or so back from the anterior border of the masseter. At this point, it may very occasionally be palpable externally. The calculus may ulcerate through the duct and lie

in a small abscess cavity outside but communicating with the duct. Figure 4 illustrates this finding in the sub-mandibular gland, and we have met with this phenomenon at least three times in the parotid. A larger abscess cavity may burst spontaneously either internally or externally depending on the position of the calculus, and in the latter case may cause an external salivary fistula. The discharge of saliva from the fistula is usually small in amount owing to the previous depression of salivary function. Finally, following the passage of the calculus either spon-

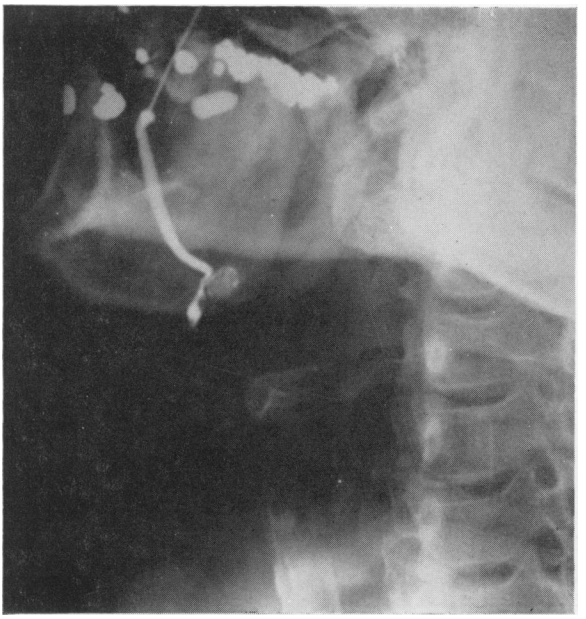

Fig. 4. Sub-mandibular sialogram with calculus in small abscess cavity outside but communicating with duct.

taneously or with the aid of surgery, a stricture may develop. We have had examples of all these happenings in this series.

Strictures may be single or multiple, unilateral or bilateral. Single strictures are usually located at or a little way behind the parotid duct orifice. Apart from those due to calculi which have in the tables been classified under calculi, or following surgery to enlarge the parotid duct orifice, a not uncommon occurrence, the original cause of the stricture is often a matter for speculation only. The triad of factors—obstruction, depression of secretion, and infection—interact in a vicious circle, so that in a late case, unless there is some obvious primary factor such as a calculus, the original cause of the trouble may be impossible to determine.

Under the heading of " obstruction—various " in Table I fall three categories of case. The first definite but probably rare, the other two speculative but possibly common. The first category is constituted by the case of a houseman who a few years ago came to me and said rather shamefacedly that one of his own parotid glands had started to swell after meals. There was nothing to see at the time and I was inclined to diagnose that he needed a holiday. A few days later, however, he came to see me again to say that the trouble had suddenly cleared up, after he had dislodged with his tongue something that was projecting from the parotid duct orifice, and

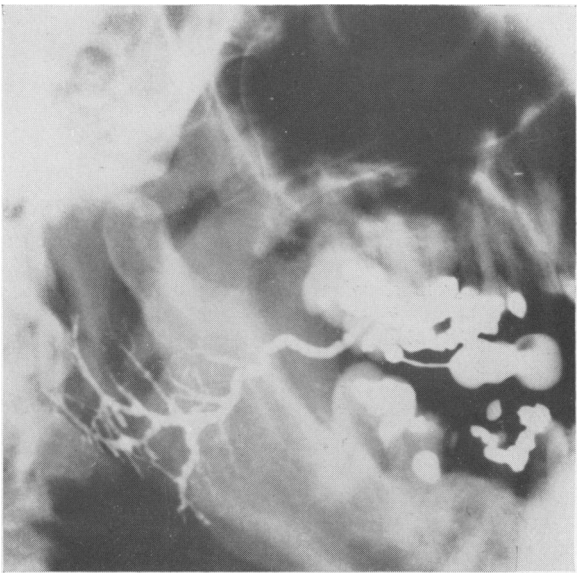

Fig. 5. Parotid sialogram showing slightly dilated duct system with beaded appearance due to mucus.

which resembled an oat husk; presumably it had entered the duct while he was eating porridge. The second category is constituted by a small number of cases that we think may have been due to papillitis, i.e. oedematous obstruction of the parotid duct orifice as a result of trauma or infection as postulated by Rose (1954). The symptoms cleared following a few days' treatment by mouth washes. And thirdly, there were a few patients in whom the obstructive attacks seemed to be caused by an accumulation of mucus, since there was always a gush of mucus as the attacks suddenly cleared. There are frequently mucus-secreting goblet cells in the parotid ducts and, as Patey and Thackray (1956) have shown, mucous glands in the substance of the parotid gland, and it seems reasonable to suppose that, if mucus is present in excess, blockage

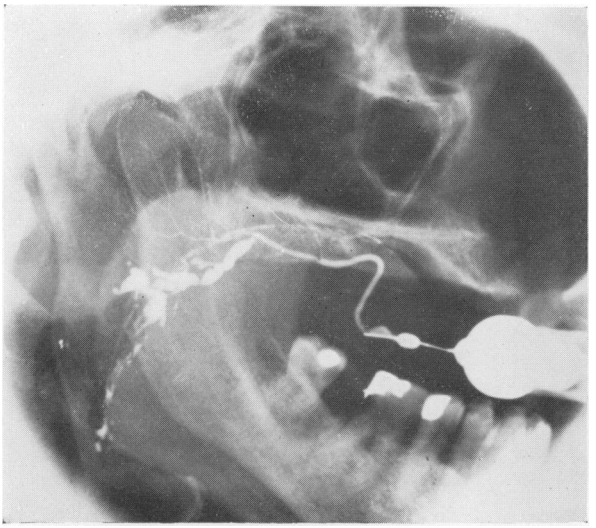

Fig. 6. Parotid sialogram showing obstruction confined to secondary duct and due to a stricture.

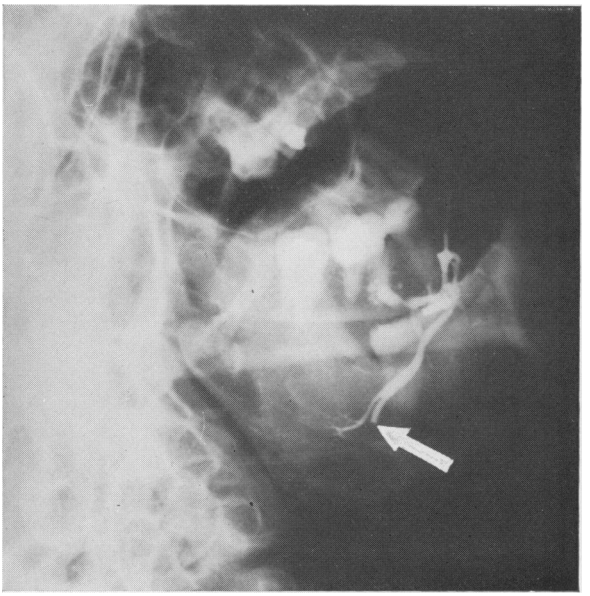

Fig. 7. Sub-mandibular sialogram showing obstruction confined to secondary duct and due to a small calculus which did not show up on a plain X-ray.

might occur in a narrow duct not primarily designed to convey mucus (Fig. 5). For the rest, I have seen as causes of obstruction of the sub-mandibular duct, but not of the parotid duct, an aphthous ulcer at the duct orifice, and a dental plate ulcer, and we are all familiar with the obstruction of the sub-mandibular duct which may follow irradiation treatment of carcinoma of the floor of the mouth.

The last type of obstructive sialo-adenitis that I want to refer to is one that we have only gradually come to recognize during the past few years and that is obstruction limited to a secondary excretory duct. Secretion

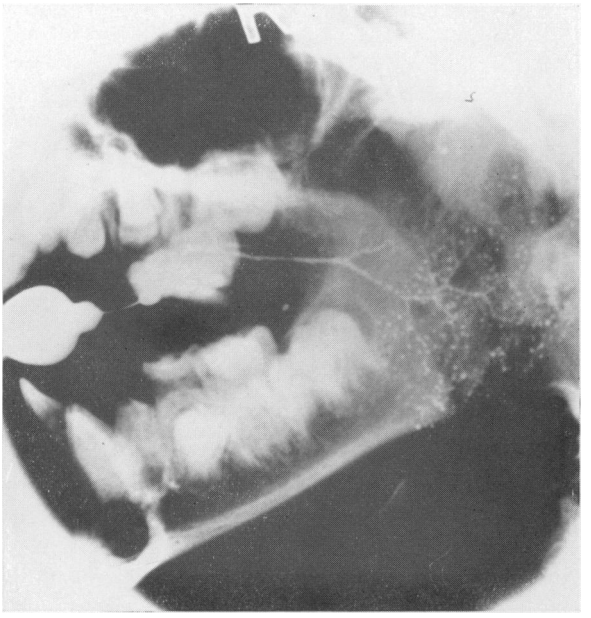

Fig. 8. Typical sialogram of punctate sialectasis.

is held up behind the obstruction, and in the limited obstructed area a focus of infection develops comparable to that behind an obstruction of a bronchus. From time to time there are attacks of pain and swelling of the whole gland due to a flare up and spread of the infection. Difficulty in diagnosis arises if the duct obstructed is a small one, since the condition may then be easily missed on sialography. Figure 6 is an obvious example of the condition in the parotid, and Figure 7 of a rather less obvious example in the sub-mandibular gland in which for a long time the diagnosis was missed.

In the conditions we have considered so far, the typical sialographic picture is some variant of dilatation of the large ducts of the gland—a

condition we may term " large duct sialectasis ". The typical sialographic picture of the next group of conditions we shall consider is quite different (Fig. 8) and the best descriptive term I know is " punctate sialectasis ", though if the opacities are very large " globular sialectasis " is an appropriate term. The term " sialectasis " was used for this condition originally under the impression that the picture was due to dilatation of the small ducts, though as we shall see later this is not the main factor. The chief functional change associated with the sialographic picture is deficiency of salivary secretion, so that an alternative term is the " sicca syndrome ". There are three main ways of presentation clinically—recurrent attacks of pain and swelling of one or both parotid glands, painless swelling of one or both parotid glands, which if unilateral and localized may simulate a tumour, and dryness of the mouth. Swellings of the sub-mandibular glands with or without pain are rare, and we have only had one gross example in our series. We have done sub-mandibular sialograms on a small number of patients with the typical picture in the parotid, but we have not seen the punctate sialectatic picture in the sub-mandibular gland, nor as far as we are aware has anyone else. But the sub-mandibular gland presumably shares with the parotid and the minor salivary glands in the general depression of secretory function which leads to the symptom of dry mouth. Though the symptoms of the parotid condition may be unilateral, the punctate sialectasis and secretory depression are usually bilateral; we have, however, seen three definite examples of unilateral punctate sialectasis.

The condition may develop at all ages, our oldest patient presenting at 70 years and our youngest at 6, but in several children it was said to have started at the age of 2 or 3. Twenty-six patients were female and 14 males. There is an interesting change in the sex incidence with age. Of those aged 10 or under, eight were males and two females, while, of those 45 or over, 10 were females and only one a male, the patient aged 70 already referred to.

Thackray and I (1955, 1956) have described the histological findings in earlier papers, and I will only briefly summarize them here. The earliest change is an accumulation of lymphocytes in and around the walls of the intralobular ducts. The accumulation of lymphocytes fragments the reticulum of the duct wall which is grossly weakened. When radio-opaque medium is introduced into the duct system in the test of sialography, it escapes through the fragmented intra-lobular ducts into the substance of the gland, and it is this extravasated radio-opaque medium that is chiefly responsible for the punctate sialectatic picture. If the disease develops without the addition of gross infection, the picture is one of gradual replacement of secreting salivary lobules by lymphoid tissue. Ultimately the epithelium elements of the gland may be represented only by a series of solid epithelial cords in a lymphomatous stroma.

In cases in which infection has been a marked feature, the main ducts become dilated and fibrosed, and the gland substance shows a mixture of acute and chronic inflammatory changes. The histological changes in an advanced sicca syndrome with associated marked infection approximate to those of the advanced cases with an obstructive infective lesion and histological differentiation may be difficult or impossible.

The secretion of saliva is, as the term sicca implies, typically depressed. This usually occurs on both sides even though the symptoms on one side may be minimal. In most cases the amounts are well below Curry's lower limits of normal, and in severe cases secretion is usually negligible or absent. Exceptionally, secretion may be within the wide limits of normal, but it is impossible to know in a bilateral condition what the normal for a particular individual is, and it seems probable that in these cases too the secretion is depressed below the individual's normal.

You will recognize that the condition I have been describing is part of a spectrum of disease processes which include the condition described by Mikulicz in 1892 and also the syndrome that Sjögren has written many papers on since 1932 and which usually bears his name, though Goujerot (1926) had described the condition previously in 1926. We have no long follow-up of Mikulicz's original case since the patient died shortly afterwards in an obscure East Prussian village of " perityphlitis ", nor of course have we sialograms or secretory studies. We do not even know the histology of the parotid, as Mikulicz never explored this gland surgically. However, he has left us in his article a small drawing of the histological findings in the lachrymal gland, and they resemble closely the findings I have already described in the parotid, i.e. replacement of glandular elements by lymphoid tissue with the epithelial elements converted into solid cords. The extra-salivary features of Sjögren's syndrome were present in many of our patients, particularly the dry eyes with a tendency to attacks of conjunctivitis, and several are under the care of our ophthalmological colleagues for this. In addition, many showed the tendency to connective tissue disorders in the form of fibrositis and rheumatoid arthritis, in one case the rheumatoid condition preceding the parotid symptoms by 14 years: another patient had dermatomyositis and another developed Raynaud's symptoms in the hands coincident with the parotid condition.

In view of the frequent general distribution of the lesions, explanations of the condition based purely on local factors in the salivary glands are clearly no longer tenable, and a more general factor would seem to be operating. The present trend is in the direction of an auto-immune explanation and there is a large amount of evidence in support of this. The histological picture is comparable to that of Hashimoto's disease of the thyroid, the gamma globulin levels in the blood are usually raised, and various antibodies may be found in the blood, particularly anti-nuclear

factor. In addition, there is the frequent co-existence with rheumatoid conditions in which auto-immune processes have been invoked, and the occasional occurrence of Hashimoto's disease and the sicca syndrome in the same patient. We have recently seen a case of this type referred to us by Dr. Deborah Doniach. The patient was a young woman of 22 with proven Hashimoto's disease, including typical thyroid antibodies in the blood, with in addition a story of very slight almost negligible symptoms of pain and swelling of the parotid glands. On investigation, she showed the complex picture of the sicca syndrome including deficient secretion, punctate sialectasis, and anti-nuclear factors in the blood. On the other hand, no one has yet demonstrated organ specific antibodies in the sicca syndrome, nor, in spite of much effort by many people including our own group, has anyone yet reproduced the disease experimentally. If the sicca syndrome is an auto-immune phenomenon, it occupies an interesting intermediate position with the local features of an organ specific condition but with the serological features of a non-organ specific condition. In view of the established relation with rheumatoid conditions and Thackray's demonstration of fragmentation of the periduct reticulum as the primary histological lesion, one is tempted to speculate that the primary fault may be in the periductal connective tissues rather than in the epithelium.

The other general cause that has been suggested is a virus infection, but the evidence for this is scanty and largely coincidental. Many patients say that their trouble started after an attack of mumps, but since the clinical features of the two conditions during an individual attack may be identical, this story must be regarded with suspicion. In a few of our cases, however, the history of other cases of mumps at the same time in the family or school makes the evidence stronger. In one of our cases, the first swelling of the parotids developed during a proven attack of glandular fever, and in another an attack of glandular fever seemed to cause an exacerbation of a previously existing parotid condition. We have also had another patient with what was thought to be a viral infection associated with facial paralysis, who had complete suppression of parotid secretion on the pilocarpine test. Finally, the incidental finding during the course of our animal experiments that the histological picture in the sub-mandibular glands in cytomegalic virus disease of guinea pigs is similar to the early picture of the sicca syndrome with its collections of lymphocytes around the intralobular ducts may prove to be of interest (Fig. 9). All one can say at present, however, is that the possibility of a virus aetiology in the sicca syndrome is worth pursuing further.

We can give only a fragmentary and incomplete picture at present of the natural history of the sicca syndrome, and long-term follow-up of a large, closely observed group of cases would be valuable. It would be particularly interesting to know what determines the change from a predominantly male disorder of childhood to a predominantly female disorder of middle

life. In some cases, the condition gradually seems to " burn itself out ". We have, for example, one boy whom we saw at the age of 7 who was getting attacks of pain and swelling once a month, and who now, 10 years later, is hardly troubled. Again, in only two of the 19 cases noted in Table I, in which we have done parotidectomy for the sicca syndrome, have the symptoms on the other side been severe enough to warrant a second parotidectomy, though since this Table was drawn up there has been one further case. On the other hand, we have had patients who have had attacks for many years, in one case for 23 years, before coming to paro-

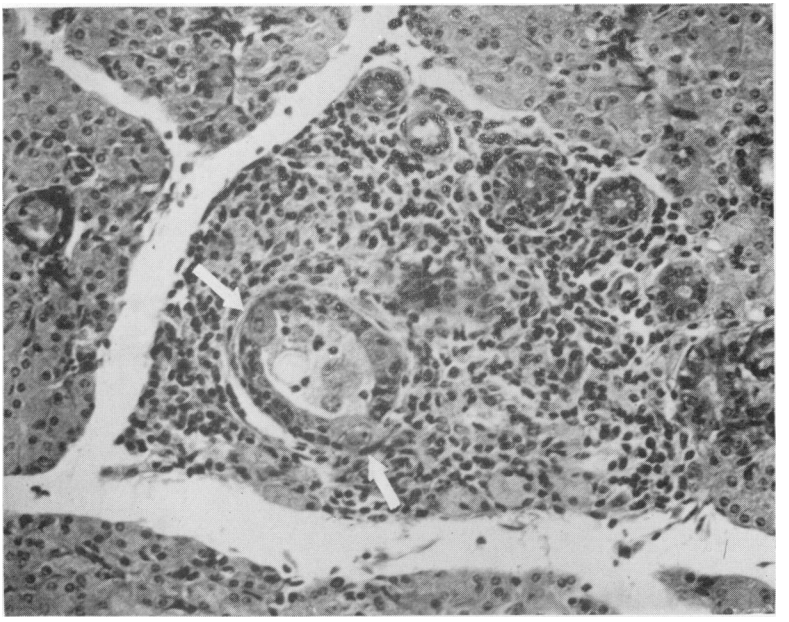

Fig. 9. Histology of sub-mandibular gland of guinea-pig infected with salivary cytomegalic inclusion virus (indicated by arrows) to show accumulation of lymphocytes around intra-lobular duct.

tidectomy, and in one of the cases in which bilateral parotidectomy was performed the second side was operated on nine years after the first. In children, it is often the interference of the attacks with schooling that determines the pressure for definitive treatment.

I will only refer quite briefly to the heterogeneous group of cases classified in Table I as miscellaneous. Several were cases in which we did not determine the cause because they were not fully investigated. There was only one case of tuberculosis of the parotid (Patey and Thackray, 1954) but this is now a rarity in this country though not necessarily in other countries. There were two cases of sarcoid, both presenting as lumps

without inflammatory symptoms. In one, the diagnosis was suggested by the X-ray of the chest, and in the other the condition was an incidental histological finding in a cyst of the parotid. There seems to be an unjustifiable tendency to diagnose sarcoidosis in obscure inflammatory conditions of the parotid, an interesting contrast to the hesitancy in suspecting the much more common parotid calculus. There was also one case of a cylindroma in which the mode of presentation was recurrent attacks of pain and swelling of the parotid, which we thought were probably due to obstruction of small ducts by nodules of tumour. Finally in this connection, there were three cases of patients with lumps in the parotid gland which were thought to be tumours, and which are presumed to have been inflammatory pseudo-tumours since the lumps disappeared spontaneously while the patients were awaiting operation.

I have deliberately left the question of treatment of all conditions till the end, since the same principles apply whatever the cause of the condition. In the case of the sub-mandibular gland the problem is usually simple. Either one can remove the cause, which is usually a calculus, by a small operation in the floor of the mouth, or one can remove the gland, and, provided one takes care to make a skin crease incision and to preserve the sub-mandibular branch of the facial nerve, the results are excellent. In the parotid, the problem is a more difficult one. The decision is between one of three courses—palliation, operations on the ducts, and removal of the gland. The main palliative measures are antibiotics to control infection during attacks, analgesics to control pain, and sometimes the passage of bougies. The last procedure may be useful in helping the discharge of pus pent up behind the oedematous duct orifice, and in the treatment of strictures at or close to the duct orifice. It is clearly useless if the cause of the condition lies far back in the duct or in the gland.

I should here say a word about radiotherapy, which in the days before parotidectomy was frequently used. The results were variable, and it was often difficult to assess any benefit that was achieved owing to the normal variability of the course of the disease. The rationale of radiotherapy is not clear since in severe cases of parotitis, whatever the initial cause, secretion is almost always markedly depressed and often absent, and in the sicca syndrome is deficient from the beginning, such deficiency probably being the principal factor predisposing to infection. Most people would probably agree that radiotherapy is contra-indicated if infection is a prominent feature, or in the presence of a foreign body in the form of a calculus. Owing to recent evidence that small dose irradiation of the thyroid gland in early childhood, incidental to radiotherapy of other neighbouring structures such as the thymus or lymph nodes of the neck, may cause carcinoma of the thyroid (Lindsay and Chaikoff, 1964), irradiation of the salivary glands in childhood should be avoided if possible. Radiotherapy may have a place as an alternative to surgery in cases in which

swelling of the parotid from lymphoid replacement is the most prominent feature with minimal infective changes, but the difficulty is that heavy doses are required for permanent effects, which lead to further diminution of the secretion of the minor salivary glands and a consequent worsening of the dryness of the mouth. More recently, radiotherapy has been used to depress parotid secretion as a preliminary to the still experimental procedure, to which I shall be referring later, of ligature of the parotid duct.

Turning to operations on the ducts, there are two traditional procedures and two new procedures. The two traditional procedures are cutting down through the mucous membrane of the mouth on to a calculus im-

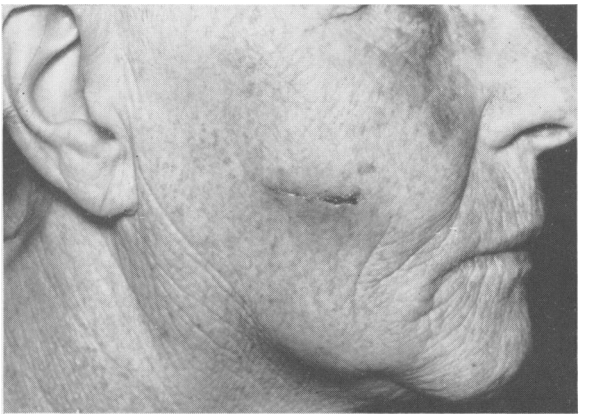

Fig. 10. Photograph of patient referred to in text one week after being treated by external incision of parotid duct distended with pus, and retrograde dilatation of stricture of intra-oral portion of duct.

pacted at the parotid duct orifice or in the intra-oral portion of the duct, and enlargement of the parotid duct orifice. Both are theoretically simple procedures, but in practice they often present difficulties and complications and should not be lightly undertaken. It may be difficult to find the stone in an area of diffuse induration, and following both procedures stricture is a common complication. The two new procedures have developed out of a bolder attitude to the parotid duct rendered possible by the knowledge that, if they fail, the situation can be retrieved by parotidectomy. The first is exploration of the parotid duct through an incision on the cheek. We have used this approach to remove a calculus impacted in the facial portion of the parotid duct and also in the situation in the patient illustrated in Figure 10. This was a patient with chronic painful swelling of the parotid gland associated with acute exacerbations. X-rays showed a calculus at the level of the anterior border of the masseter. Various intra-

oral manipulations had been carried out and an impassable stricture had resulted. A short incision was made at the anterior border of the masseter, a dilated parotid duct identified and opened, allowing the escape of much pus from a dilated duct system, and a calculus removed from a small abscess cavity adjacent to the duct. Probes were passed along the parotid duct into the mouth, the stricture fully dilated, and the duct closed round a polythene tube which was brought out through the mouth. The wound rapidly healed, the patient's symptoms disappeared and she now has a patent parotid duct, though secretion is limited to a small amount of mucus.

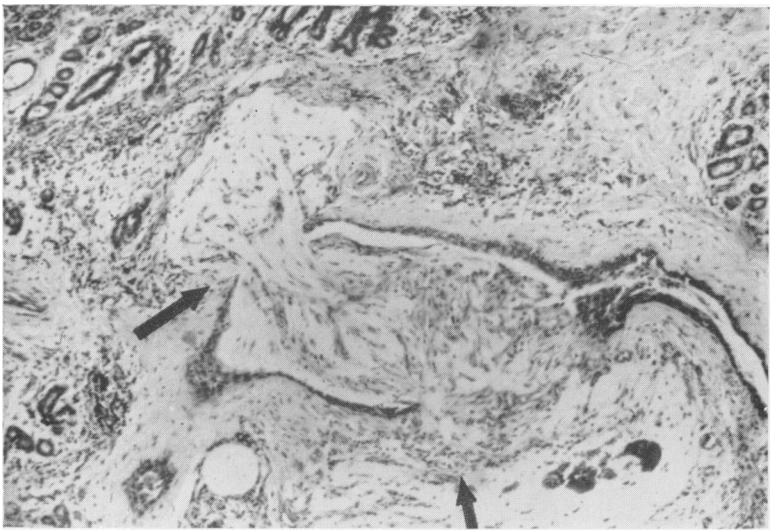

Fig. 11. Histological section of parotid gland removed after failure of the operation of ligature of the duct. A duct distended with mucus can be seen, and in two places the duct has ruptured and the mucus escaped into the surrounding tissues.

The other new procedure is ligature of the parotid duct intra-orally, as advocated by Diamant (1958), after preliminary irradiation of the parotid gland, if necessary, to depress parotid secretion. The rationale of the operation is to convert the gland into a non-functioning organ cut off from the potential infection of the oral cavity. We have performed the operation in four patients, once bilaterally. Three of the patients had substantially no salivary secretion so that preliminary irradiation was not necessary. The fourth had secretion which was depressed by preliminary irradiation. The operation appears to have been successful in two patients, though the follow-up is still short. In the two others, the operation was unsuccessful, symptoms were made worse, and parotidectomy had to be carried out. In one, the cause of the failure appeared

to be a gross accumulation of mucus in the obstructed duct system, which in places had ruptured through the distended ducts (Fig. 11). We have some other evidence bearing on this question in the form of the cases already mentioned, in which stenosis developed either spontaneously or as a result of attempts to enlarge the duct orifice. We had one patient in whom, following stenosis arising in this way, the trouble cleared up for six years until she died from an unrelated cause. On the other hand, we have had several patients whose trouble became so much worse as a result of the stenosis that parotidectomy had to be carried out. The position of this small operation remains to be worked out, but at present it would seem wise to adopt it only if the patient and the surgeon are agreed on parotidectomy in the event of failure. If the operation fails, parotidectomy should not be unduly delayed as fibrosis sufficiently severe to make the operation difficult seems particularly likely to occur.

Finally, if symptoms are severe and other measures have failed, one can offer the patient removal of the parotid gland with conservation of the facial nerve. In most cases the operation is not appreciably more difficult than the similar operation for tumours and may in fact be easier, but occasionally, and especially following duct stenosis or if abscess formation has occurred, fibrosis around the facial nerve may make its separation difficult and bleeding may be more troublesome. With patience, however, the nerve can be preserved, and we have so far had no case of permanent facial paralysis. Functional facial paralysis is commoner after operations for parotitis (Patey, 1963) than for tumours, but this always clears up. In performing parotidectomy for inflammatory conditions, it is always wise to remove the duct well forward into the fatty tissues of the cheek, as, if a long stump of dilated infected duct is left, a residual abscess may develop in relation to it.

The work I have presented has depended in large degree on the collaboration of colleagues at Middlesex too numerous for me to mention all by name, of all degrees of seniority, clinical and non-clinical, and I would like to take the opportunity of acknowledging my indebtedness to them. In conclusion, I hope I have shown that the study of a small back-water of surgery may not only enable us to fulfil better our primary duty of treating the patients concerned, but may also perhaps contribute in some small measure to increasing our understanding of the mechanisms of disease.

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GRANT OF FELLOWSHIP DIPLOMAS

AT THE RECENT Final Examination for the Fellowship, 18 candidates out of 50 were successful in Ophthalmology, one candidate out of 42 in Otolaryngology, and 135 out of 562 in General Surgery.

At an extraordinary meeting of the Council on Wednesday, 9th December, Diplomas of Fellowship were granted to the following:

KIRBY, Norman George (*Birmingham*).
CARTER, Anthony Peter (*Middlesex*).
HOLDEN, Christopher Edward Aldridge (*Guy's*).
‡SHEPHERD, Mary Patricia (*Royal Free*).
TAYLOR, Alfred Roy (*St. Mary's*).
DUNSTAN, Michael Kingsley (*Guy's*).
WRIGHT, John Edward Charles (*King's College*).
GEDDES, John Ronald (*Birmingham*).
KANE, James Francis (*Liverpool*).
McCOY, David Richard (*The London*).
BAILEY, John Stuart (*St. Mary's*).
EARLAM, Richard John (*Liverpool*).
BALL, John Robert (*St. Mary's*).
DALRYMPLE, James Oxenham (*Charing Cross*).
*‡KNOWLES, Shirley Heather (*Royal Free*).
YEN, William (*Royal Free*).
BROOKS, Peter Lewis (*King's College*).
DICKSON, George Haines (*St. Mary's*).
ROWE-JONES, David Colin (*Westminster*).
ONYEASO, Onyemara Nduche (*Westminster*).
HOMEWOOD, Michael John Harper (*The London*).
SINGLETON, John McLellan (*University College*).
HOBBS, Kenneth Edward Frederick (*Guy's*).
HUNTER, Gordon Andrew (*University College*).
KESTLE, Michael John (*Birmingham*).
WALROND, Errol Ricardo (*Guy's*).
AUBREY, David Alan (*King's College*).
HARTFALL, William Guy (*Guy's*).
HOPKINSON, Brian Ridley (*Birmingham*).
EL GHATIT, Ahmed Zaki (*Cairo*).
SAAD, Magdy Naguib (*Alexandria*).

* In Ophthalmology. ‡ Woman.