

RECURRENT PAROTITIS IN CHILDREN

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Recurrent parotitis is a well-documented condition and is usually described as occurring in both adults and children.¹⁻³ Only a few authors have specifically described the syndrome as it occurs in childhood,⁴⁻⁸ and the following review of 44 children suffering from recurrent parotitis is presented in an attempt to elucidate the aetiology and pathogenesis, natural history, and management of the condition. The series comprises 23 patients under the care of the surgical unit at the Transvaal Memorial Hospital for Children, Johannesburg, over the past 12 years and 21 personal patients of one of the authors (D. J. du P.).

REVIEW OF 44 CASES

Race incidence. Of the 44 children, 40 were White and 4 Cape Coloured. This racial incidence is partly due to the fact that there are only White children at the Transvaal Memorial Hospital for Children. However, no case in a Bantu child has been encountered by us, and this is probably significant. The condition is known to be very rare in Bantu children,^{9,10} and Royce⁶ has reported that the condition is seldom seen in American Negro children.

Sex incidence. Twenty-six of the patients were boys and 18 girls. This confirms the findings of others^{9,7} that recurrent parotitis in childhood is slightly more common in boys than in girls.

Symptoms and signs. The attacks of parotitis consisted of pain and swelling in the region of one or both parotid glands, of sudden onset and lasting on the average 3-7 days.

On examination during an acute attack, the parotid gland was found to be swollen and tender, but with no

erythema of the overlying skin. Flakes of pus (the 'snow-storm' appearance¹¹) could usually be expressed from Stensen's duct, and the orifice of the duct was frequently red and oedematous. The systemic reaction to the infection varied from trivial to severe, with a high temperature necessitating, in some cases, confinement to bed and consequent loss of schooling.

When examined between attacks, several children exhibited a slight residual firm swelling of the parotid gland, but in most cases the gland was impalpable. The orifice of Stensen's duct was oedematous in a few cases, though usually of normal appearance. In only one patient was residual swelling between attacks really marked, but this patient also complained of pain and swelling in the gland while eating, and was one of those found to have a secondary obstruction of the duct system.

The inflammatory process sometimes also affected the accessory parotid gland which lies close to the main duct anterior to the parotid gland.¹² On two occasions this presented clinically with a palpable mass in the region of the main parotid duct in the cheek. These swellings were first thought to be parotid calculi in the duct, but both, on exploration, proved to be enlarged accessory parotid glands.

Age at onset and frequency of attacks. The age at onset of attacks varied from 8 months to 12 years, though in most cases the attacks started between the ages of 3 and 6 years. With the exception of 2 patients who first presented to us as adults (aged 21 and 34 years), the children had been suffering from the condition from 1 month to 8 years when first seen. The acute attacks occurred

with varying frequency, most commonly every 3-4 months.

Side involved. In 26 patients the attacks of parotitis occurred on one side only (right side—14, left side—12) while in 18 patients both sides were involved, though most of these patients had started with unilateral attacks, the opposite side becoming involved months or years later. In patients with bilateral involvement both sides rarely suffered attacks simultaneously, and the frequency and severity of attacks usually varied considerably from side to side.

Complications. The only complication due to parotitis was the development of a parotid abscess in 1 patient. This was drained at another hospital, and it healed without fistula formation.

Associated conditions. In 2 children there was a definite history of allergy, e.g. asthma or hay fever.

Two children suffered simultaneous attacks of parotitis and acute tonsillitis, while in 11 others there was a history of recurrent tonsillitis not coinciding with the attacks of parotitis.

In 4 patients the parents associated the first attacks of parotitis with the cutting of teeth, and in 5 it was noted that several carious teeth were present.

Family history. Two mothers and one father reported having suffered from a similar condition during childhood with spontaneous resolution. There were no patients whose siblings suffered from a similar condition.

INVESTIGATIONS

Serum proteins and electrophoretic pattern. These tests were found to be normal in 4 children on whom they were carried out.

Agar-gel precipitin tests against extracts of normal parotid tissue. No parotid antibodies were detected in 5 patients tested.

Mucoviscidosis. No evidence of this was found in 2 children tested.

Culture of saliva. This was done on 13 patients and revealed *Staphylococcus aureus* in 2 instances, pneumococci in 3 instances, and *Streptococcus viridans* in 8 instances. There appeared to be no relationship between the severity of attacks and the type of organism cultured as reported by Jones.⁷

Sialography. Sialograms were obtained from 29 patients, 10 bilaterally. In the earlier cases in the series lipiodol was used as contrast medium, but this was later changed to neohydriol fluid. A standard technique was not used in all cases since the earlier sialograms in the series were performed by various radiologists; and one technique used, for instance, was to inject dye until the patient complained of pain in the gland, usually after 1-1½ ml. of dye had been injected. This method tended to produce an over-filled gland exhibiting a general mottling owing to acinar filling which obscured the clear outline of the duct pattern¹³ (Fig. 1). Our method is to inject only ¼-½ ml. of dye slowly to avoid excessive extravasation of dye within the gland substance and the foreign-body reaction which it produces.¹⁴

All the sialograms demonstrated the abnormality of 'sialectasis'¹⁴ consisting of small globules of contrast medium associated with the intralobular ducts. The distribution was usually diffuse (Fig. 2), but occasionally patchy.

A common explanation for this radiological appearance is that the contrast medium has entered dilated ducts (similar to bronchiectasis), and in view of the young age of these patients this was formerly thought to be a congenital abnormality—hence the term 'congenital sialectasis' used for this condition. There is, however, much evidence to show that the sialectatic appearance is produced by rupture of the intralobular ducts with extravasation of the contrast medium into the interstitial tissue of the gland.

Patey and Thackray¹⁴ showed this to be the case by means of serial histological sections after sialography, and Ranger¹⁵ has demonstrated that sialectasis may be produced by injecting a normal gland under very great pressure. We have also noticed that 'sialectasis' appears before the acini are completely filled, proving the abnormal fragility of the intralobular ducts in this disease, since good acinar filling can usually be produced without the appearance of sialectasis.¹⁵ In addition, if these globules of contrast medium were in dilated ducts, the flow of saliva would be expected to flush out the ducts; but we have often seen contrast medium still in the gland on X-rays taken days or even weeks, and on 1 occasion as long as 5 years, after sialography.

The degree of sialectasis may vary considerably from very mild to moderate or severe. We have not noticed any relationship between the size of the globules of contrast medium and the severity of the disease or the causative infecting organism, and it is probable that the degree of sialectasis is related to the volume and pressure of the material injected during sialography. This view is supported by the fact that the globules of dye are so very similar in size in each individual case.

Sometimes sialography may demonstrate the appearance of a space-occupying lesion within the parotid gland owing to an area of lymphoid hyperplasia.¹⁴

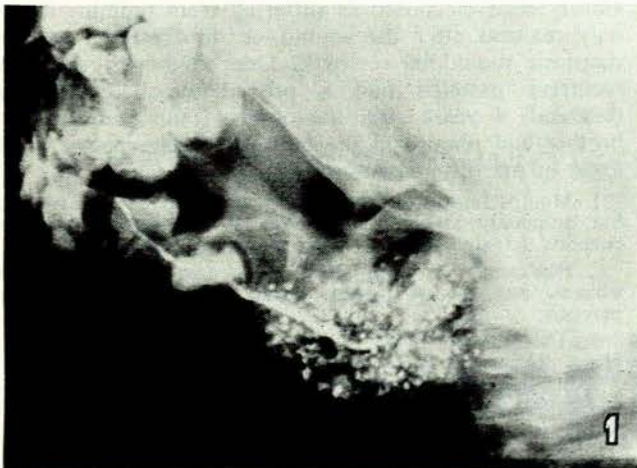


Fig. 1. Overfilling of the gland, which causes acinar filling and so obscures the duct outlines.



Fig. 2. Diffuse moderate sialectasis.

Usually the main duct is unaffected by this disease and appears perfectly normal on the sialogram, but in some instances the duct outline is irregular (Fig. 3) presumably from inflammation involving the lining of the duct. The end

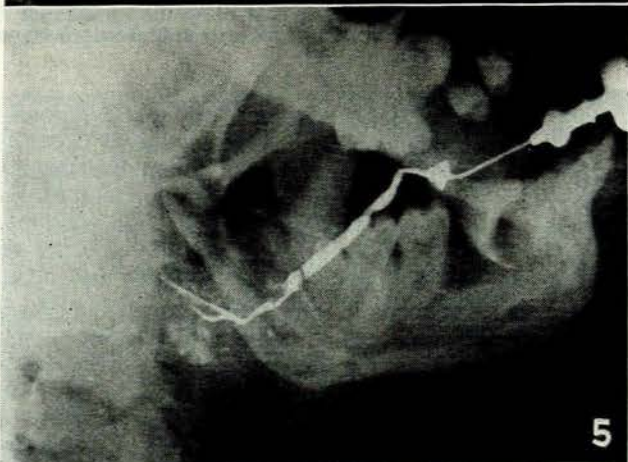
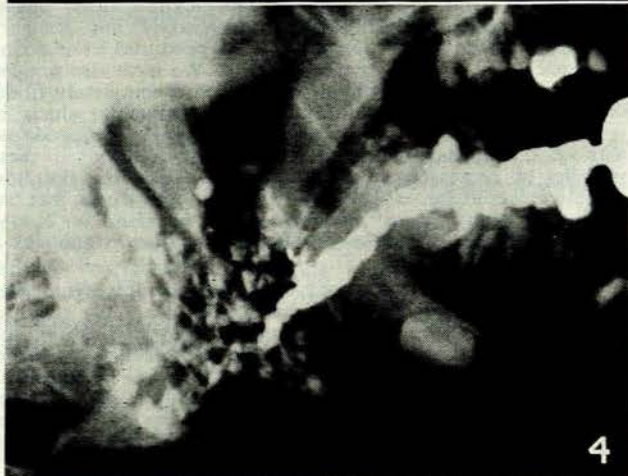
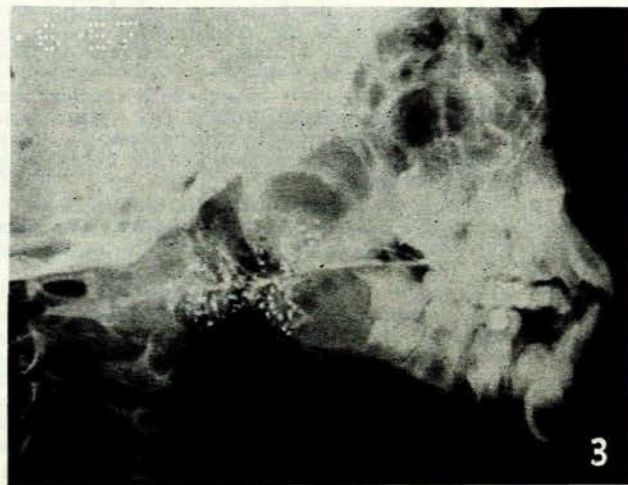


Fig. 3. Sialectasis of the accessory parotid gland lying above the main duct which is irregular.

Fig. 4. Sialectasis plus a dilated and tortuous main duct owing to a secondary inflammatory stricture near the duct orifice.

Fig. 5. A filling defect in the main duct produced by enlargement of the accessory parotid gland.

result of such a process would be an inflammatory stricture of the duct, and in one of our patients such a stricture formed with a distended tortuous duct beyond it (Fig. 4). This was the child in whom the gland was enlarged between attacks of acute parotitis and became further swollen on eating.

In one of the patients with a palpably enlarged accessory parotid gland there was a filling defect in the duct which, on surgical exploration, proved to be due to pressure on the duct from outside by the inflamed accessory gland (Fig. 5).

In no patient did the symptoms improve after sialography, nor was the investigation responsible for any serious complications, although it often resulted in some swelling and discomfort which only lasted for a day or two.

Histology

The histological appearance has been fully described before, particularly by Patey and Thackray,¹⁴ who have drawn attention to the classical picture of duct epithelial hyperplasia, periductal lymphocytic infiltration, and acinar atrophy and fibrosis—leading eventually to the disappearance of the acini with the ducts represented by solid columns of cells among the lymphocytes and fibrous tissue. In this series all these features have again been seen.

It has been pointed out on a previous occasion¹⁶ that these features are not pathognomonic of any one disease, but probably represent a non-specific reaction on the part of the parotid salivary gland to chronic irritation, whatever the nature of this irritant. The diagnosis cannot therefore be made on the histology alone, but on the composite picture including clinical features, sialography as well as histology.

These histological changes are not seen in all the lobules of the gland in every case, and in some instances some normal lobules are found. This explains the patchy distribution of 'sialectasis' in some cases. Whether this represents only an early stage of the disease, which will later spread to involve all lobules of the gland, is uncertain.

If secondary obstruction occurs owing to blockage of the duct by epithelial hyperplasia or an inflammatory stricture, the distal ductules will distend (Fig. 6). If a biopsy is taken during an acute inflammatory episode pus cells will be found in the distended ducts.

An interesting finding is the abnormality of the basement membrane of the intralobular ducts which can be seen with special reticulin stains. In the normal gland it is regular and well formed (Fig. 7), but in recurrent parotitis it appears to be irregular, fragmented and even absent in some areas (Fig. 8). This will explain the increased fragility of these ducts which is responsible for the extravasation of dye and the appearance of 'sialectasis'.

Differential Diagnosis

The condition most frequently confused with recurrent parotitis is mumps. Three children in the series had previously been diagnosed as suffering from mumps; it was only realized after the second or third attack that the diagnosis should be reviewed. One child with unilateral recurrent parotitis had a proved attack of mumps (bilateral) 4 years after the onset of the parotitis. Her brother had mumps at the same time. The points which assist in the differentiation are:

1. Mumps frequently begins as a unilateral parotid swelling but ultimately involves both sides. It is rare for recurrent parotitis to involve both sides simultaneously.
2. Pus cannot be expressed from Stensen's duct in mumps, whereas it is always present during an attack of recurrent parotitis.
3. The serum amylase is usually raised during the early phase of mumps. It is normal in recurrent parotitis.
4. A neutropenia is usually present in mumps, while the white blood count is usually raised during an acute attack of parotitis.

Other conditions which may be confused with recurrent parotitis are adenitis of the intraparotid lymph nodes,¹⁷

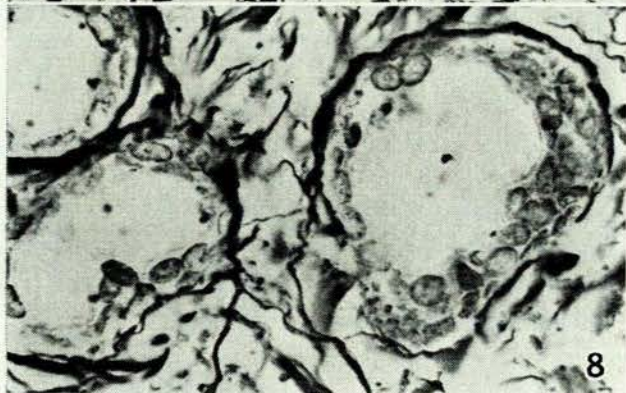
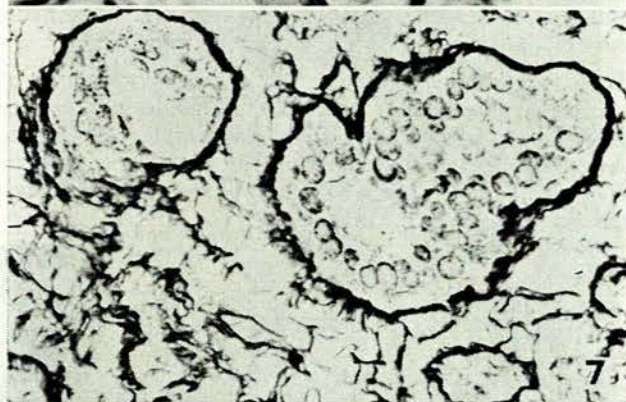
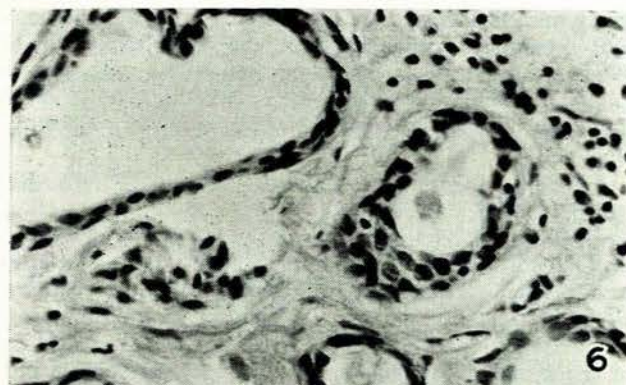


Fig. 6. Duct dilatation owing to a secondary obstruction.

Fig. 7. Reticulin stain to show intact basement membrane in a normal gland.

Fig. 8. Reticulin stain in recurrent parotitis in a child, showing fragmentation of the basement membrane.

neoplasms of the parotid gland, sarcoidosis, and leukaemia; but these can usually be excluded by careful examination and investigation.

TREATMENT

Acute Attacks

Patients seen during an attack of acute parotitis were treated with a variety of antibiotics, usually penicillin, and responded satisfactorily within 3-7 days. From the histories obtained it was apparent that a doctor was not consulted for every acute attack, and those not treated with antibiotics appeared to respond just as well.

Prevention of Attacks

Mothers were instructed to massage the parotid glands daily, and the use of acid sweets and chewing gum was advised to encourage a free flow of saliva. It cannot be assessed to what extent these measures were of benefit. Ten children underwent tonsillectomy with improvement of symptoms in 3. One patient was treated with deep X-ray therapy to both parotid glands, after the age of puberty, with no relief of symptoms. Because of the potential danger of radiotherapy in the region of the head and neck in children, it was not used on any other patient. Irrigation of Stensen's duct with antibiotic solutions,²⁸ long-term antibiotic therapy and steroid therapy were not employed.

Surgical Measures

In the 2 patients in whom an accessory parotid gland was palpated this was explored because in one instance it was thought to be a calculus and in the other it was obstructing the duct. In both instances the enlarged accessory parotid gland was excised. In one patient this was followed by formal dilatation of Stensen's duct with complete relief of symptoms; the other was lost to follow-up.

One patient underwent formal duct dilatation and, though there was no evidence of duct obstruction, attacks of parotitis ceased thereafter.

Meatotomy of the duct orifice was performed in 5 patients in whom it was thought that the orifice was somewhat constricted. No benefit accrued from this procedure.

Auriculo-temporal nerve avulsion was performed on 17 patients; bilaterally in 2. In 3 instances section of the 'nerve' avulsed showed 'no evidence of nerve tissue'. Three patients have been lost to follow-up, and we have regarded the 3 patients in whom no nerve tissue was noted as having had inadequate operations. Of the remaining 13 procedures parotitis recurred in 9 glands, although in 2 instances attacks ceased within 6 months of operation. Of the 4 patients in whom attacks ceased postoperatively it was reported that in 2 with bilateral involvement the opposite sides, which had not undergone the procedure, had ceased to suffer attacks as well, and we have regarded these 2 results as probably fortuitous. The other 2 patients, apparently cured by the procedure, were aged 8 and 12 years respectively. In view of the natural history of this condition as noted below, the cure of these 2 patients may not have been due to the operation.

Follow-up

Follow-up has been possible on 31 of the 44 patients in the series, either by means of personal examination or reply to a questionnaire. Of the 31 patients 21 have ceased having attacks of parotitis, and the ages at which symptoms disappeared are shown in Fig. 9. It will be noted that in most of the children attacks ceased between the ages of 11 and 13, and all had stopped by the age of 15 years. Of the 10 patients still suffering attacks, all except 3 are still under the age of 14. These 3 all have some special feature which can explain the persistence of symptoms. One is a girl of 17 with typical 'sialectasis'. Her attacks are becoming increasingly infrequent, and her parents state that they can feel a 'lump' in the cheek. It is possible that there is a calculus present or an acces-

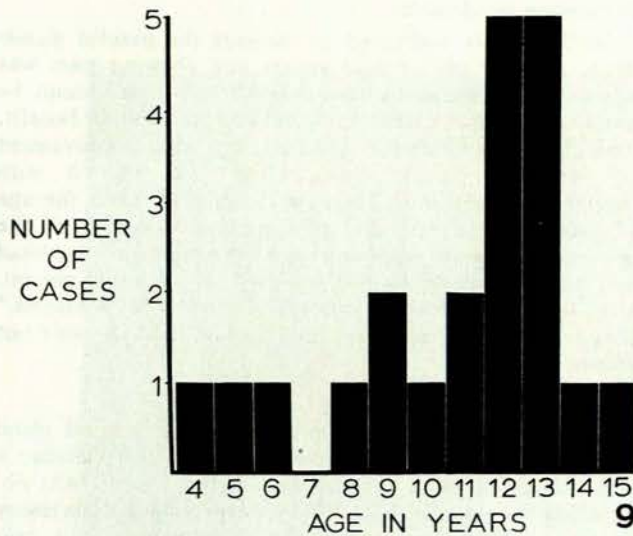


Fig. 9. The ages at which attacks of parotitis ceased in 21 patients.

sory parotid gland causing duct obstruction. The second patient is a man who presented to us for the first time at the age of 21. He had main-duct dilatation and an enlarged accessory parotid gland obstructing the duct from outside. This was excised, but he has been lost to further follow-up. The third patient is a man of 34 who began having attacks at the age of 6. He has had bilateral meotomy of the duct orifices performed and has had deep X-ray therapy to both glands. He has a bilateral abnormality of his bite and is thought to suffer from Costen's syndrome, which may be the cause of his recurrent parotitis at this age.

With 2 exceptions, in all patients with bilateral involvement attacks ceased on both sides within a few months of one another. In several children the parotid gland was still slightly enlarged on palpation even though attacks of parotitis had ceased. This was particularly apparent in the one patient with main-duct dilatation in whom attacks ceased at the age of 13½ after excision of the accessory parotid gland and dilatation of the duct. However, the gland has since gradually subsided over the past 3 years and is no longer palpable.

DISCUSSION

Aetiology and Pathogenesis

Although the clinical syndrome of recurrent parotitis has long been recognized, until recently little has been known of its cause and pathogenesis because of the paucity of histological material available for examination. In 1956 Patey and Thackray¹⁴ published their histological findings in excised parotid glands, the seat of recurrent parotitis, which had previously been investigated by sialography. Their cases fell into 2 main groups:

- (1) Those which showed dilatation of the main duct as the primary feature on sialography.
- (2) Those with 'sialectasis' involving the intralobular ducts as the main feature, with a normal main duct.

In the late stages of the disease the microscopic findings of these 2 groups are very similar, but we agree with

Patey and Thackray¹⁴ that these are 2 separate conditions. Our experience coincides with the view expressed by Pearson¹ that the 'sialectatic' variety is the type seen in children and that the type with main-duct dilatation is common in adults in whom the disease is due to obstruction of the main duct.^{1,19}

In the present series only 2 patients showed main-duct dilatation and both had secondary main-duct obstruction. The one had an enlarged accessory parotid gland obstructing the duct from outside while the other had a stricture near the duct orifice, presumably secondary to the inflammation. This latter patient also had an enlarged accessory parotid gland and was cured after this was excised, but at the same time the duct was dilated and it seems probable that the stricture was the main cause of the main-duct dilatation.

The Cause of 'Sialectasis'

(a) *Congenital.* Since it is now known that the essential lesion in the gland is not a true 'sialectasis', comparable, say, to bronchiectasis, the theory of Bailey,²⁰ that the individual is born with a honeycomb-like gland, is no longer tenable.

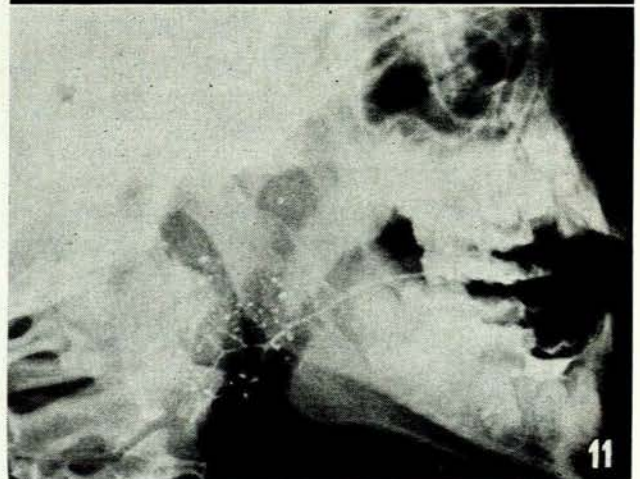
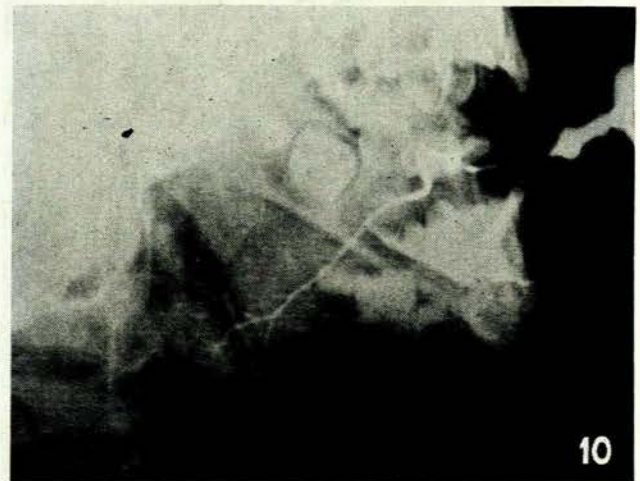


Fig. 10. Sialogram of an unaffected gland on 18 December 1957 showing acinar filling but no sialectasis. This child had recurrent parotitis on the opposite side.

Fig. 11. Sialogram on same gland as Fig. 10 on 9 December 1960 showing sialectasis.

In this series one child with unilateral symptoms had bilateral sialography performed at the age of 6 years, and the asymptomatic side showed no sialectasis, but 3 years later bilateral sialography was repeated and the asymptomatic side now demonstrated typical sialectasis. This demonstrates that the sialectasis is not present at birth (Figs. 10 and 11).

(b) *Obstruction.* This appears to be an unlikely cause of the condition unless it is at the level of the intralobular ducts. It might conceivably be caused by an increase in the viscosity of the saliva, but this has not been demonstrated in recurrent parotitis.² Two patients in the present series were investigated for mucoviscidosis, but no evidence of this was found. Microcalculi are known to form in the ducts in some cases of recurrent parotitis, but they almost certainly do not constitute the primary lesion.

(c) *Stagnation of secretion.* Jones⁷ has postulated that there is stagnation of secretion in serous glands as compared with mucous glands because of the longer and narrower connecting channels in the former. It is noteworthy that no condition similar to 'sialectasis' occurs in any salivary gland other than the parotid. Ranger¹³ in sialographic studies has shown that it is possible to produce the picture of 'sialectasis' in normal parotid glands by the injection of excessive amounts of dye; whereas it is impossible to produce a similar effect in the submandibular glands by the same method. He postulated that the intralobular ducts of the submandibular gland were shorter and wider than those of the parotid, allowing distension of the potentially large acinar space with less likelihood of duct rupture. However, these 2 postulates do not explain why 'sialectasis' should occur in some parotid glands and not in others.

(d) *Increased duct fragility.* It is certain that increased fragility of the intralobular duct walls exists in 'sialectasis'. This increased fragility might result from various disease processes involving the duct wall:

(1) *Allergy.*^{3,4} An allergic history was obtained from 2 children in the present series. Jones⁷ reported a history of allergy in 3 of his 17 patients, but reasoned: 'In view of the frequency of allergy in one form or another in the general population there seems insufficient evidence that it played a causative role in these 17 patients.'

(2) *Auto-immune disease.* In the late stages of the disease, according to Patey and Thackray,² the histological appearances superficially resemble those of chronic lymphadenoid thyroiditis. Mosbech and Kristensen²¹ have reported a case of 'chronic sialoadenitis' thought to be caused by auto-immunization. However, the agar-gel precipitin test for parotid antibodies was performed in 5 patients in the present series and proved to be negative in all.

(3) *Collagen disease.* No evidence has come forward to implicate collagen disease in the aetiology of recurrent parotitis. No trial of long-term steroid therapy has been carried out.

(4) *Infection.* Infection has been thought to be a primary factor in the causation of 'sialectasis'.^{3,7,10} However, in the present series we have encountered 2 children suffering from unilateral parotitis, in whom bilateral sialograms revealed sialectasis not only on the affected side but also in the opposite asymptomatic gland.

Infection is almost certainly a secondary phenomenon caused by mouth organisms. Recurrent tonsillitis was present in 11 patients in the present series, and it represents a fruitful source of secondary infection. The teeth also provide a

source of secondary infection, and the association of the onset of parotitis with the eruption of teeth is probably due to secondary infection from the gums. Carious teeth were noted in 5 patients in the present series.

Florman⁸ believed that mumps might be the primary cause of the condition. That this is unlikely is shown by the fact that 1 patient in this series suffered a proved mumps infection 4 years after the onset of unilateral recurrent parotitis.

Three factors have been noted in the present series which may have some bearing on the aetiology of recurrent parotitis in children:

(1) *Heredity.* In the present series it has been noted that in the cases of 3 children, 2 mothers and 1 father had suffered from a similar condition in childhood. This association has also been noted by Pearson.⁴

(2) *Racial and environmental factors.* It would appear that recurrent parotitis is rare in Bantu^{9,10} and American Negro⁹ children. This might be due to racial factors as well as dietary factors, since Bantu children exist on an almost exclusively carbohydrate diet.

(3) *Hormonal factors.* Since most children with recurrent parotitis appear to be cured spontaneously before or at puberty, it seems possible that some hormonal aetiological factor might be implicated.

NATURAL HISTORY

It has been shown in the present series that recurrent parotitis will subside spontaneously at puberty, usually before the age of 15, unless there is some additional complicating factor, e.g. main-duct obstruction. This fact has been noted by other observers.⁴⁻⁶

It is not known why the attacks of parotitis cease. Possibly there is complete resolution of the pathological process in the gland or else the disease destroys the gland completely; it has however been observed that the parotid glands continue to secrete saliva in 'cured' cases.

It is probable that this phenomenon of spontaneous remission of the disease accounts for the occasional 'cure' obtained from various surgical procedures, e.g. dilatation of the duct and auriculo-temporal nerve avulsion.

MANAGEMENT

In the light of the known facts concerning the aetiology and natural history of recurrent parotitis in children, the following plan of management is suggested:

1. Investigation.

(a) *Sialography.* This should be performed in every case to confirm the diagnosis and exclude main duct obstruction.

(b) *Culture of saliva or pus from Stensen's duct.* This should be done to determine the appropriate antibiotic to be used during acute attacks.

2. *Prevention of stagnation.* The affected parotid gland should be massaged regularly by the child's mother, and the use of acid sweets and chewing gum should be encouraged.

3. *Removal of sources of secondary infection.* If there is a history of recurrent tonsillitis or if the tonsils are obviously septic they should be removed. Carious teeth must be attended to.

4. *Treatment of allergy.* Any allergic manifestations, e.g. asthma, hay fever, should be controlled.

5. Surgical treatment.

(a) *Removal of obstruction.* If obstruction to the main duct exists it should be removed by the appropriate method, e.g. meatotomy, dilatation of the duct, or excision of an extrinsic obstruction.

(b) *Auriculo-temporal nerve avulsion.* In our experience this operation has not been effective in preventing further attacks and it is not recommended.

(c) *Parotidectomy.* This may be partial or complete. It has been reported favourably by various authors,^{14,22,23} though they all stress the hazards involved. They report that no patients in their series suffered permanent facial palsy, though a number suffered temporary paresis or paralysis. Because of the presence of periglandular fibrosis the facial nerve is exposed to great danger, and in our view parotidectomy should not be advised unless recurrent parotitis is causing severe debility, excessive loss of schooling, or gross psychological upset.

SUMMARY

1. The clinical features of a series of 44 children with recurrent parotitis are reviewed.
2. The radiological and histological appearances are presented.
3. The pathogenesis of this condition is discussed.
4. The natural history is presented on the basis of a follow-up study of 31 of the patients.

5. The management of this disease is outlined.

REFERENCES

1. Payne, R. T. (1933): *Lancet*, **1**, 348.
2. Patey, D. H. and Thackray, A. C. (1956): *British Surgical Practice—Surgical Progress*. London: Butterworth.
3. Pearson, R. S. B. (1961): *Gut*, **2**, 210.
4. *Idem* (1935): *Arch. Dis. Childh.*, **10**, 363.
5. Bigler, J. A. (1946): *Med. Clin. N. Amer.*, **30**, 97.
6. Royce, S. W. (1952): *Amer. J. Dis. Childh.*, **84**, 468.
7. Jones, H. E. (1953): *Arch. Dis. Childh.*, **28**, 182.
8. Florman, A. L. (1955): *J. Pediat.*, **46**, 682.
9. Dinner, M. (1963): Personal communication.
10. Schneier, M. (1963): Personal communication.
11. Payne, R. T. (1938): *Proc. Roy. Soc. Med.*, **31**, 398.
12. Du Plessis, D. J. (1957): *S. Afr. Med. J.*, **31**, 773.
13. Ranger, I. (1957): *Brit. J. Surg.*, **44**, 415.
14. Patey, D. H. and Thackray, A. C. (1955): *Ibid.*, **43**, 43.
15. Du Plessis, D. J. (1961): *S. Afr. Med. J.*, **35**, 189.
16. *Idem* (1958): *Ibid.*, **32**, 264.
17. *Idem* (1957): *Medicine in South Africa*, p. 8. Cape Town: Medical Association of South Africa.
18. Ollerenshaw, R. G. W. and Rose, S. S. (1951): *Brit. J. Radiol.*, **24**, 538.
19. Rose, S. S. (1954): *Ann. Roy. Coll. Surg.*, **15**, 374.
20. Bailey, H. (1945): *J. Int. Coll. Surg.*, **8**, 109.
21. Mosbech, S. and Kristensen, H. P. O. (1960): *Acta med. scand.*, **168**, 147.
22. Beahrs, O. H., Devine, K. D. and Woolner, L. B. (1961): *Amer. J. Surg.*, **102**, 760.
23. Perzik, S. L. (1961): *Ibid.*, **102**, 769.