Disorders of the Digestive System

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General preface to series

Student textbooks of medicine seek to present the subject of human diseases and their treatment in a manner that is not only informative, but interesting and readily assimilable. It is also important, in a field where knowledge advances rapidly, that the principles are emphasized rather than details, so that information remains valid for as long as possible.

These factors all favour an approach which concentrates on each disease as a disturbance of normal structure and function. Therapy, in principle, follows logically from a knowledge of the disturbance, though it is in this field that the most rapid changes in information occur.

A disturbance of normal structure without any disturbance of function is not important to the patient except for cosmetic or psychological considerations. Therefore, it is the disturbance in function which should be stressed. Preclinical students must get a firm grasp of physiology in a way that shows them how it is related to disease, while clinical students must be presented with descriptions of disease which stress the basic disturbance of function that is responsible for symptoms and signs. This approach should increase interest, reduce the burden on the student's memory and remain valid despite alterations in the details of treatment, so long as the fundamental physiological concepts remain unchallenged.

In the present Series, the major physiological systems are each covered by a pair of books, one preclinical and one clinical, in which the authors have attempted to meet the requirements discussed above. A particular feature is the provision of cross-references between the two members of a pair of books to facilitate the blending of basic science and clinical expertise that is the goal of this Series.

RNH
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Preface

The approach adopted in this book to disorders of the digestive system is in terms of clinical situation rather than of individual diseases. Common clinical situations such as jaundice or disturbances of defaecation are presented and the student is shown how the experienced clinician analyses the problem and decides on management. Nevertheless, information on any disease can easily be looked up by reference to the index.

Terminology is always a problem for students embarking on a new field, and the clinical scene is no exception. In some curricula students have already studied pathology before coming to the wards, and they should have little difficulty. For the rest, the problem is greater. I have tried to mitigate the difficulty by explaining terms the first time they are used in the text, but a good medical dictionary will be very helpful.

A specific area where terminology can be particularly confusing is drugs: generic names and commonly used trade names may both change from one area of the English-speaking world to another. In general, the generic name in the UK is given in the text, sometimes with a commonly used UK trade name in parentheses; a list of all preparations mentioned is given in the Appendix, with US equivalents.

The general philosophy of the Series, that clinical matters will be described in the light of our knowledge of the underlying disturbances of physiology, has been closely followed in this book. In a few areas, such as gastric secretion in Chapter 4 and gastric emptying in Chapter 5, more details of tests of function are given than is usual in a text meant for clinical medical students, but it is hoped that these will help to illustrate the importance of physiological concepts and measurements in treating some patients. There has been the closest co-operation between Dr Paul Sanford, the author of the companion volume on the physiology of the digestive tract in this Series, and myself during the writing of the pair of books, and there are frequent cross-references in both texts to the companion volume.

Acknowledgements

It is a pleasure to thank my colleagues at The Middlesex Hospital for their help in criticizing the text and providing radiological, clinical and pathological illustrations, and in particular my medical gastroenterological colleague

Dr Peter Cotton. Specific sources are acknowledged in the captions, but I specially wish to thank Dr Brian Thomas of University College Hospital, London, for Fig. 2.2 and Dr John Olney of the Royal Free Hospital, London, for Fig. 6.4.

Miss Alison Thomson nobly bore the brunt of the burden of typing and retyping, and I am grateful to her for her efficiency and endurance!

I greatly appreciate the help given me by the publishers at all stages of the writing and production of this book.

Finally, to my wife and children for whom this book was yet another task that reduced my time with them, I express my gratitude for their forbearance.

1981 MH

Contents

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Salivary glands

Although the secretion of the salivary glands initiates the process of digestion, disorders of these glands are not always considered to be the province of the gastroenterologist. Many other specialists, including general surgeons, otorhinolaryngologists, immunologists and plastic surgeons as well as general physicians, may find themselves treating diseases of the glands; numerically, the majority of patients require surgical attention. In terms of physiological systems, the digestive seems the best one under which to consider disorders of the salivary glands.

Symptoms of dysfunction

Patients whose major presenting complaint falls under this heading include those with a dry mouth and those with excessive salivation. The overwhelming majority of patients with either complaint show no evidence on examination of the mouth to substantiate their symptom; the impartial observer reckons that the dryness, or the obvious salivation, is within the rather wide spectrum of the normal range.

It is useful to have an objective test of the rate of salivary secretion. The best standardized appears to be Curry's, in which secretion from each parotid gland is collected (Sanford: *Digestive System Physiology*, Chapter 2) for 5 minutes after an intravenous injection of 5 mg pilocarpine nitrate, a dose that evokes maximum secretion. The normal range is 3–13 ml of secretion from each gland; patients with a secretion within this range and with no other evidence of disease of the salivary glands can be reassured that their subjective sensation of an abnormality in either direction has no organic basis and must be due to increased awareness of sensation in the oral mucosa. After such reassurance the symptoms usually diminish. The rare patients with a true reduction in secretion are likely to have some form of Sjögren's syndrome (p. 3), while hypersecretion with parotid glands visibly larger than normal has been described but is extremely rare.

Lump in parotid region

The boundaries of the parotid region are shown in Fig. 1.1. A swelling, not arising in skin, and which lies in or overlaps the boundary may be an intrinsic lesion of the salivary gland or may arise in some other structure such as a lymph node or the subcutaneous tissue. However, the chances are 3 : 1 that it is a neoplasm of the parotid itself. Three-quarters of parotid neoplasms are a benign lesion called *pleomorphic salivary adenoma* (the old name, which should be abandoned, was *mixed tumour*); however, this tumour and most other benign salivary tumours show an unpleasant tendency to recur locally as multiple seedling deposits if cells of the growth are shed into the tissues by a preliminary biopsy or during the process of removal. Because these benign lesions show also a distinct tendency to undergo malignant change as the years pass, the logical management of a subcutaneous lump in the parotid region is *complete excision-biopsy*; that is, removal of the lump with a wide margin of normal tissue and without any preliminary biopsy. This policy can be technically difficult because of the facial nerve trunk and its branches that lie within the gland and may be in very close proximity to the tumour, and in an ideal world the surgeon should have special training, experience and interest in this field. The operation is called *conservative parotidectomy* (because it

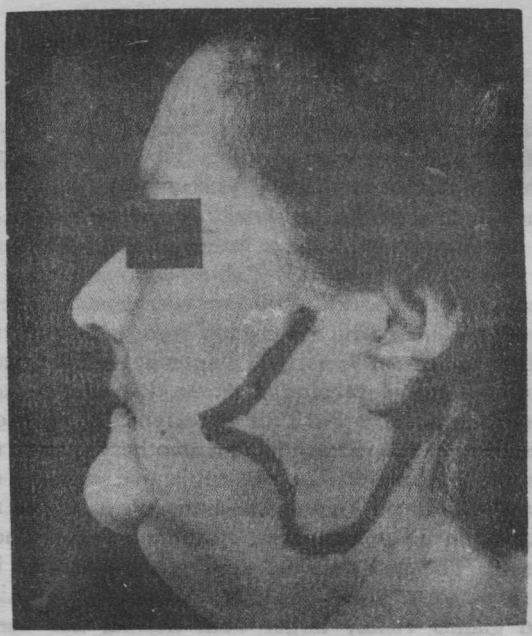


Fig. 1.1 This patient's left parotid salivary gland was indurated (because of Sjögren's disease, p. 3); its margins were easily identified and have been mapped with a skin pencil. Note how far downwards into the neck, and backwards over the mastoid process, the lower pole extends. The extension forwards along the parotid duct is called the accessory lobe.

conserves the facial nerve), and may be *superficial* if the lump is in that part of the gland superficial to the nerve, or *total* if the lump is found to be in the part deep to the nerve so that this part must be removed as well. Section of the facial nerve results in permanent paralysis of one-half of the face—an unpleasant deformity.

Malignant tumours of the parotid gland constitute about 10 per cent of all parotid tumours. They may give rise to a clinical suspicion of malignancy because of aggressively rapid growth or because of fixity to bone; such patients have a poor prognosis, fewer than 10 per cent being alive 5 years after diagnosis, and their management is an unsolved problem but usually involves some combination of surgery and radiotherapy. Sometimes a lump presenting without features of malignancy, and removed with a margin according to the policy of the previous paragraph, is reported by the histopathologist as being more malignant than expected, but the prognosis is substantially better in those cases. Occasionally, during what the surgeon embarked on as a conservative parotidectomy, he finds that the lump appears to be infiltrating one or more branches or even the trunk of the facial nerve rather than pushing them aside. In order to maintain the margin of normal tissue around the tumour, it is then necessary to sacrifice the whole nerve (*radical*) or one or more branches (*semi-conservative parotidectomy*).

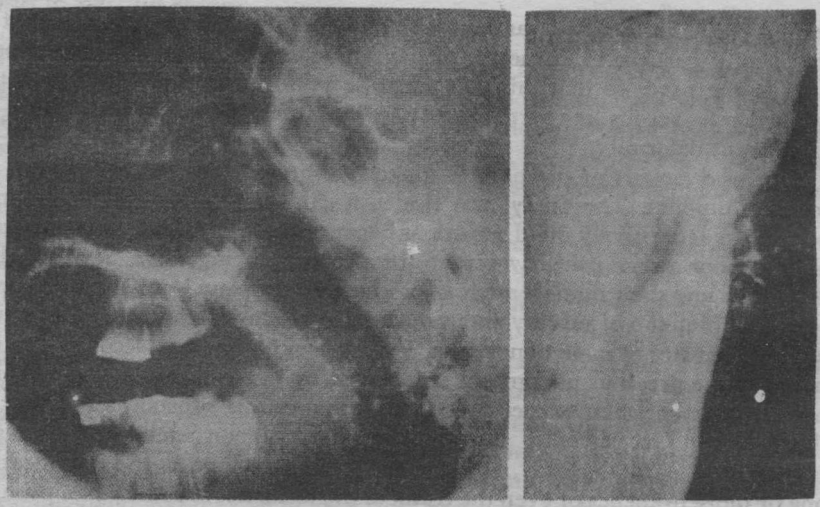
Chronic or recurrent bilateral parotid swelling

The characteristic shape (Fig. 1.1) of the parotid salivary gland is maintained if the gland as a whole enlarges and becomes indurated because of chronic or recurrent disease. Recurrent attacks of acute pain and swelling are usually diagnosed as mumps in the first attack, but this diagnosis has to be reviewed when the attacks recur or if the glands remain persistently enlarged and hard.

This group of patients has not yet been well studied, but it would appear that many show in biopsy specimens the characteristic histological features of *Sjögren's disease* (syn. *benign lymphoepithelial disease*): infiltration with small round cells and proliferation of the ordinary epithelial cells and also the myoepithelial cells (Sanford: *Digestive System Physiology*, Chapter 2) of the parotid ducts, which may proceed to a degree that obliterates the lumen of the ducts. A few of these patients have other clinical features of the group constituting *Sjögren's syndrome*: dryness of the mouth (hyposecretion may be confirmed by Curry's test), dryness of the eyes (due to interstitial keratitis) and various rheumatic manifestations. Two-thirds of the patients have abnormal autoantibodies in their blood, including antibodies to salivary glands. The characteristic radiological finding in Sjögren's disease is punctate sialectasis (Fig. 1.2).

Apart from the suggestion that this is a disease of autoimmunity, little is known about its aetiology. It occurs at any age, and in infants and young adults is usually self-limiting after running a course of several years. Very rarely, some form of non-Hodgkin's lymphoma may develop.

Treatment is restricted to reassurance that in most patients the disease will 'burn itself out', although a few cases respond to large doses of steroids. Occasionally the symptoms are severe enough to warrant parotidectomy.



(a) (b)

Fig. 1.2 Sialogram showing punctate sialectasis: (a) lateral view, and (b) anteroposterior view. The large ducts are normal (note how narrow these normal ducts are), but the walls of the finer ducts at their terminations are weakened by the disease so that the contrast material readily bursts through them to extravasate as globules.

A rare cause of chronic bilateral parotid swelling is *sarcoidosis*. Drugs such as *iodides* and anti-Parkinsonism agents, and malnutrition are other possible causes, although the mechanisms involved are unknown.

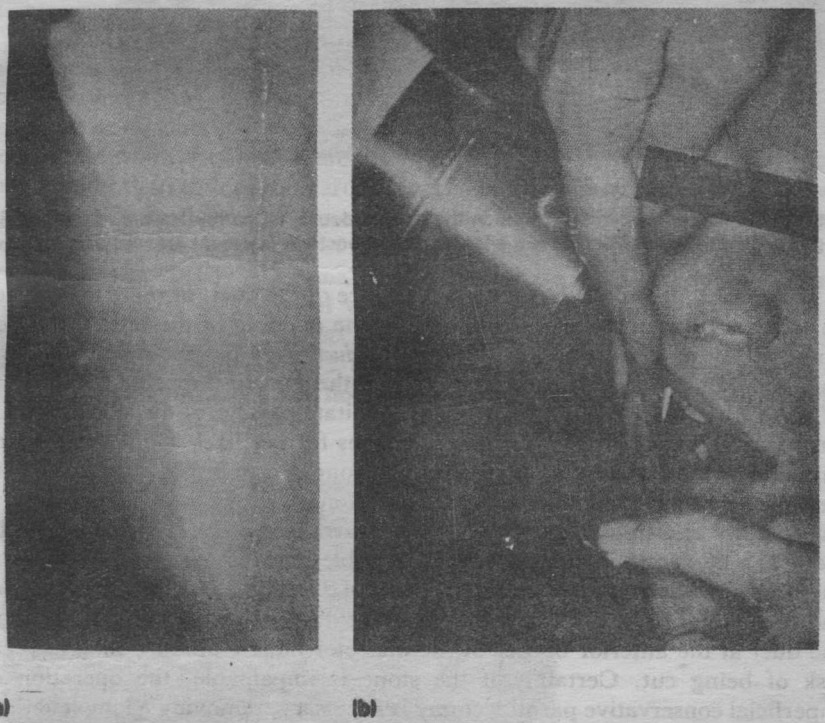
Chronic or recurrent unilateral parotid swelling

One-fifth of patients presenting in this category prove to be cases of Sjögren's disease in which the clinical symptoms are limited to one side, although punctate sialectasis can usually be demonstrated in the opposite parotid as well and in many patients the symptoms ultimately become bilateral. Most of the remaining four-fifths of patients probably have their bouts of painful parotid swelling due to the presence of a stone in the parotid duct, producing periodic impaction and obstruction with proximal sequestration of parotid secretion.

A careful history often distinguishes between Sjögren's disease and parotid duct calculus. In Sjögren's disease the attacks of swelling start gradually, last several days and fade gradually away, while dryness of the mouth is a chronic feature not particularly related to the attack in time or lateralized to the side of the attack. By contrast, the patient with a calculus says his attacks are sudden in onset and in ending, he is sometimes aware that the nalf of his mouth on the side of the affected gland is dry during the attack, the relief of symptoms is often ushered in by a gush of saliva into the mouth on the affected side, and the duration of the whole episode ranges from a few hours to a day or two at

the most. Pain tends to be more intense in the shorter attacks with a stone than in the longer episodes of Sjögren's disease.

Inspection and palpation of Stensen's duct, particularly at its termination in the mouth opposite the second molar tooth, may confirm the presence of a stone or localized inflammation or deformation due to impaction of the stone. Radiology is always necessary to confirm the site and size of the stone because these factors determine treatment. Plain x-rays, particularly the anteroposterior and intrabuccal views (Fig. 1.3), may show the stone;



(a) (b)

Fig. 1.3 (a) Plain x-ray, anteroposterior view, of the parotid region, showing a radio-opaque calculus. (b) Technique for obtaining an intrabuccal view of the termination of the parotid duct. This procedure eliminates the jaws and teeth from the background.

however, it may be very small (the calibre of the normal parotid duct is less than 1 mm) and radiolucent, so it is important to use sialography—the injection of a radio-opaque contrast medium into the duct system of a salivary gland via its oral orifice (Fig. 1.4). The calculus usually expresses itself as dilatation of the main duct system proximal to a filling defect or an apparent stricture, but occasionally produces a complete cut-off appearance of the main duct. These appearances are very different from the 'punctate sialectasis' of Sjögren's disease (see Fig. 1.2).

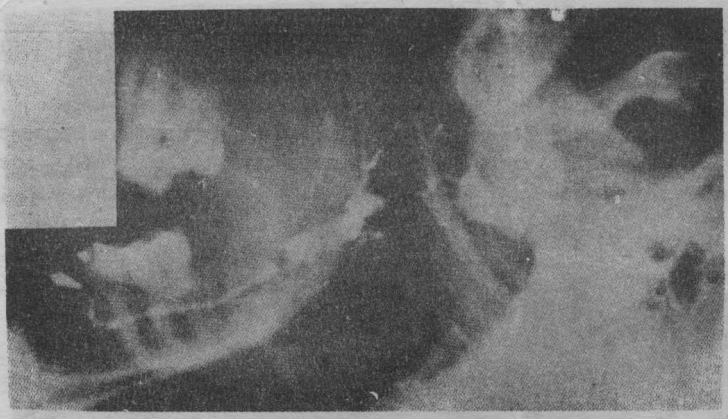


Fig. 1.4 Sialogram showing a stone in the parotid duct. The narrow calibre of the anterior segment, the rounded filling defect and the dilated posterior segment are well seen.

If the calculus is palpable at the oral orifice of the duct, or there is evidence that it has been impacted at that orifice, the opening of the duct should be enlarged by an operation via the mouth (*ductoplasty* or *duct meatotomy*). The same operation may be worth performing if the stone is impacted within 2 cm of the orifice, since this procedure may facilitate passage of the stone the next time it travels forwards and impacts. Stones further back should in the first instance be treated expectantly: certainly stones up to 3 mm in diameter are very likely to pass spontaneously. Larger stones require surgical removal, if the severity and frequency of the attacks warrant this. Even if the site of the stone can be determined because it is palpable in the cheek, cutting down on the stone is a hazardous procedure because of the close proximity to the duct of the two buccal branches of the facial nerve: these branches may wind round the duct at the anterior border of the masseter muscle and are then at great risk of being cut. Certainly if the stone is impalpable, the operation of superficial conservative parotidectomy is necessary, removing a long length of duct with its contained stone in continuity with the salivary gland.

Acute parotid swelling

Whether it is unilateral or bilateral, acute painful parotid swelling is usually diagnosed as mumps. Sometimes there are features that make the diagnosis virtually certain; for example, associated orchitis or pancreatitis, concurrence with an epidemic, or exposure to a contact with mumps 10–14 days earlier. The diagnosis can be confirmed by finding a rising titre of mumps antibody in the patient's serum, but it is not usually considered necessary to perform this investigation. Nearly always the attack subsides spontaneously and there is never any recurrence. This in itself can be taken as confirmation of the diagnosis, although presumably in some unilateral cases the cause was a stone that passed spontaneously at the first attempt!

A mechanical disturbance of drainage from the parotid duct orifice occasionally stems from a local lesion such as inflammation and oedema of a socket following tooth extraction; ascending infection can also be precipitated by poor oral hygiene in a debilitated patient, but this latter cause is rare in centres where nursing standards are high.

Disorders of submandibular salivary gland

The common affection of this salivary gland is a syndrome of recurrent bouts of painful swelling of one gland due to obstruction of its duct by a calculus. The attacks usually come on during a meal, when salivation increases, and subside within a few hours. The shorter duration, compared with symptoms due to a parotid duct calculus, may be due to the fact that impaction is less likely to be firm in the submandibular duct, of which the internal diameter is much greater than that of the parotid duct. The stone may be easily palpable in the duct in the floor of the mouth, or occasionally, if it is large and has impacted at the very beginning of the duct at the hilus of the gland, it may be felt via the neck. Nearly all submandibular calculi are radio-opaque and plain x-rays, particularly an intraoral view of the floor of the mouth (Fig. 1.5), usually confirm the diagnosis; occasionally sialography is necessary. A stone in the duct can be removed via a small incision through the wall of the duct and the overlying mucosa of the floor of the mouth, but a stone in the gland or

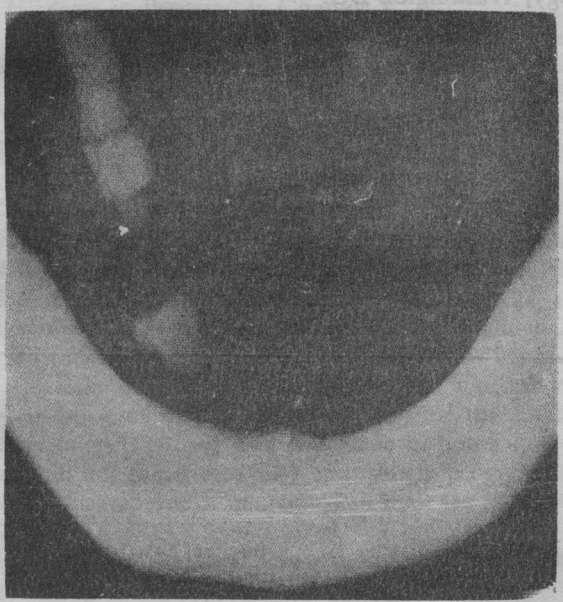


Fig. 1.5 Plain x-ray: intraoral view of floor of mouth, showing several radio-opaque stones in the submandibular duct near its orifice.

a gland that has become chronically infected and remains indurated between acute attacks requires removal of the whole salivary gland via a cervical incision.

All the neoplasms that occur in the parotid can also occur in the submandibular salivary gland. The absolute incidence of all the neoplasms is lower, but the proportion of malignant tumours is higher in the submandibular than in the parotid. In a patient with a palpable swelling in the submandibular region, failure of clinical and radiological examination to reveal a calculus in the gland or its duct suggests that a tumour is present, and an excision via the neck of the whole salivary gland with a wide margin of surrounding normal tissue is indicated.

Further reading

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2

Oesophagus

The predominant symptom associated with oesophageal disease is *dysphagia*, a term which means difficulty in swallowing. Notice particularly that the difficulty is not necessarily pain. Moreover, localization by the subject of the site of his symptom is poor so that a complaint that the food seems to stick behind the lower end of the sternum, for example, is not good evidence that the obstruction is at the lower end of the oesophagus.

Spasm of the muscle of the oesophageal wall produces pain which is characteristically retrosternal and may radiate down the left arm, thus mimicking the pain of myocardial ischaemia. This symptom, independent of dysphagia, is a rare form of presentation of oesophageal disease.

Heartburn is discussed in Chapter 3.

There are no other *direct* symptoms of oesophageal disease, but symptoms due to secondary effects or related causes are considered below.

Dysphagia

History

A patient with dysphagia due to an organic constricting lesion in his gullet classically complains that at first the difficulty occurs only with solid foods which require considerable chewing, but that as time passes the difficulty increases until in the end liquids cannot be swallowed and the patient is in the pitiable state of being unable even to swallow his own saliva.

This relentless progression is typical of a neoplastic lesion. Other organic lesions such as inflammatory stricture or the muscular inco-ordination of achalasia (p. 12) may wax and wane in bouts superimposed on a generally downhill but sometimes only very slowly progressive course. Dysphagia can also be a symptom of neurosis, but then is typically worse with liquids than with solids.

The cause of the dysphagia is sometimes suggested by particular features of the history. The typical association with heartburn related to posture in patients with gastro-oesophageal reflux is discussed in Chapter 3; a history of swallowing corrosive liquids points to the dysphagia being due to a long fibrous cicatricial stricture. Acute inflammatory conditions of the mouth or