Although this is the fully developed background of these attacks they may nevertheless constitute the first warning of serious disease. It is soon after getting into bed that the patient is conscious of dyspnoea. Often it wakes him out of sleep and keeps him from dropping off again, for as soon as he does so he is seized by the throat, so to speak, and apparently threatened with imminent suffocation, often in the form of terrifying dreams. He cannot lie down in bed; frequently it is impossible even to rest against a bank of pillows, and he has to sit up in an armchair. Sometimes even this does not suffice, and he has tried to find what rest he can by leaning forward with his head pillowed on his arms, on the back of a chair, or on a bed table. Indeed, one of the essentials in treating these attacks is to make provision, by means of one or other of these supports, for such small measure of comfort as is left to the unfortunate victim. I doubt whether there is any more distressing experience that falls to the lot of mankind than this, and it is most tantalizing to be able to offer so little help. Fortunately it is one of the many forms of agony in which the use of morphine brings relief. On the whole I am inclined to think that the value of the morphine is enhanced by combining atropine with it, but about the value of the morphine there can be no doubt whatever. By way of preventing recurrence of the attacks the best remedies to use are, I believe, the xanthine derivatives, particularly caffeine and diuretin. In some instances, when the supervention of fibrillation has precipitated the onset of these nocturnal paroxysms, the use of digitalis may, for a time at all events, decrease the overwhelming load borne by the ventricle and restore the patient to comparative comfort. Otherwise, the chief indication is to reduce the blood pressure by rest, dieting, and purgation, if this has not already been accomplished by the failure of ventricular output.

Acute suffocative oedema of the lungs is rare in my experience. A brief note of a case recently seen with Dr. Paul Bodman, to whom I owe the account of the attack itself, will serve by way of description.

The patient, a high-strung woman aged 60, had for some months experienced pain behind the sternum on exertion, relieved by stopping still. Recently she had also had attacks of dyspnoea at night. In one of these Dr. Bodman found her cyanosed, her face appearing bloated and hot, while the extremities were cold; she was intensely dyspnoeic, with bubbling respiration, and was coughing up blood-stained fluid. Her head felt full, but the veins in the neck were not distended. The blood pressure in the attack was 180/110, whereas at other times it was not so high; yet nitrates gave no relief. One curious fact about the attacks was that they were particularly apt to occur on Saturdays; another, that before the onset of an attack she often felt remarkably well; and also that in the attack there was often an imperative call to relieve the bowels. The attacks seemed to come on when she lay down. She found that in an hour or two the attack passed off, without much help from treatment. We decided to give her a course of diuretin, and this was followed by a relatively long period of freedom from the attacks, which relapsed as soon as the drug was stopped.

Dr. Anthony Birrell, who has lately published an account⁹ of some examples of this syndrome, has found that venesection brings relief from the attack. He notes that the blood flows centripetally—that is, from the opening in the vein on the distal side of the incision. This observation may ultimately prove significant in determining the nature of the attacks, the clinical background of which appears to be the same as that of cardiac asthma.

One or two lessons emerge from a study of cardiac attacks. The first is the immense importance of listening to what the patient has to say; after all, he has been through the mill, and may therefore be presumed to know something about it. Even if the attack is obviously functional it is probably the source of much anxiety and distress, for there is something inherent in cardiac sensations of all kinds that appeals vividly to the instinct of fear. Much harm may be done by an unsympathetic attitude towards patients of this class, and it is especially important to avoid even the use of the word "nerves." On the other hand, I believe it is an even worse mistake to frighten the patient whose attacks are really serious by talking to him about sudden death. Usually he is only too well aware of the dangers that threaten him, and it will do more harm than good if the apprehensions that not unnaturally beset him are reinforced rather than allayed.

The patient's age is significant. Attacks due to organic

heart disease are rare in young subjects, while those that are symptomatic of functional disorder are common. Conversely, cardiac attacks arising after 40 are rarely functional in origin. In a majority there is a basis of organic disease, even though this may be overlaid by a superstructure of functional neurosis. It is in cases of this kind that one has to be most careful, especially as the patient's relatives, to say nothing of one's own inclinations, are apt to urge a diagnosis of "nerves." I must confess that I know of no golden rule by which errors of this kind may be avoided, except that which applies to every branch of diagnosis—namely, that of taking trouble over it.

In conclusion, it must be admitted that mistakes are bound to occur, and we must learn to take risks, always bearing in mind that it is the duty of the doctor to consider the safety and well-being of his patient at the expense of his own reputation.

REFERENCES.

- ¹ Galli: *Gazz. degli Osped. e delle Clin.*, 1927, xlviii, No. 29.
- ² Mackenzie: *Angina Pectoris*, Oxford Medical Publications, 1923, p. 92.
- ³ Parkinson and Bedford: *Quart. Journ. Med.*, 1927, xxi, 21.
- ⁴ Ghrist and Brown: *Amer. Journ. Med. Sci.*, 1928, i, 336.
- ⁵ Cotton and Lewis: *Heart*, vii, 23.
- ⁶ Ryle: *British Medical Journal*, 1928, i, 537.
- ⁷ Parkinson and Bedford: *Lancet*, 1928, i, 4.
- ⁸ Horder: *Medical Notes*, Oxford Medical Publications, 1921, p. 67.
- ⁹ Birrell: *Bristol Med.-Chir. Journ.*, 1928, xlv, 239.

Observations

ON THE

VALUE OF RADIOGRAPHY IN THE DIAGNOSIS OF OBSCURE DENTAL SEPSIS.

(With Special Plate.)

BY

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THE value of radiography in the diagnosis of obscure dental sepsis is now universally recognized. Physicians who, previous to the discovery of x rays, had urged that many of the "rheumatic" group of diseases (arthritis, fibrositis, myositis, neuritis, and certain forms of heart disease) were due to dental sepsis, were faced with the argument that those patients with the most obvious dental sepsis were the patients who suffered least from these diseases. When, however, it became possible to demonstrate on a radiograph deep-seated and unsuspected lesions, the physicians were able to show that these "rheumatic" conditions, which had not shown the slightest response to other forms of treatment, were cured or their progress checked by the removal of the teeth shown to be involved.

Most medical men are familiar with the obstacles which these early workers had to surmount, not only from the members of our own profession, but also from the dental surgeons, who were not prepared to sacrifice useful and "comfortable" teeth without more evidence of their dangerous character.

Patients suffering from dental sepsis may be roughly divided into two groups: (a) those in whom the sepsis is associated with local pain; (b) those in whom the sepsis is unassociated with local pain. The patient belonging to Group (a) usually makes the earliest appointment with the dentist, and in the majority of cases he is able by his clinical examination to detect the seat of the pain. In a few cases, even after a thorough examination, the cause of pain may not be detected, particularly as the patient's localization is so poor, and frequently the pain mysteriously ceases before or while the clinical tests are being applied and cannot be elicited by further testing. This may happen on several occasions. A careful radiographic examination of these cases may reveal the cause in the form of hidden or deep-seated caries, and save the dental surgeon much time and annoyance and the patient much pain. The changes revealed by the radiograph may be obvious or very slight, but even so it may afford the only clue to the site of the pain. It is, however, chiefly with patients in Group (b) that the radiograph is of the greatest value, because the lesion is often unsuspected by the patient, and frequently is not, and apparently cannot be, detected by the most carefully applied clinical tests; and it is not until

the physician or surgeon finds signs or symptoms of a diseased condition which he knows to be generally due to absorption of toxic products from septic teeth that the teeth are radiographed and the lesion demonstrated.

There would appear to be a very great difference of opinion amongst dental surgeons as to the significance of the radiographic findings. It is therefore advisable that the radiographs be interpreted by an unbiased observer. Some still attach no importance to any radiographic appearance, however gross, if the teeth are comfortable and apparently sound; others are prepared to treat lesions which show on the radiographs as dark areas (light areas on the prints made from radiographs as in the accompanying illustrations) of absorption of the periapical tissues, but do not feel justified in "sacrificing" useful and comfortable teeth solely on "slight" radiographic evidence. Experience has now proved that the extraction of such teeth often relieves the symptoms of the patient, and Price states that when such teeth, or cultures therefrom, are inserted into experimental animals the latter die of toxic absorption or infection. On the other hand, some dental surgeons go to the other extreme and extract teeth the best radiographs of which show not the slightest change. Yet Price says:

"I wish to stress that we have come to the time when involved teeth can be so definitely differentiated from those that are not involved or with a sufficient limit of error that we are not justified in condemning all the teeth for fear they may be involved. I am seeing continually patients who are suffering more from the inconvenience and difficulties of mastication and nourishment than they did from the lesion from which their physician or dentist had sought to give them relief."

The important clinical manifestations of hidden dental sepsis were described in 1922 by Dr. Leonard Mackey, whose valuable paper is supported by all the researches which have since been made.

THE RADIOGRAPHIC APPEARANCE OF SEPTIC TEETH.

It must be clearly understood that the evidence which the radiograph gives is only a "skeleton" picture of the pathological lesion. The actual lesion, certainly in bone pathology, is always of much greater extent than the radiograph suggests to an untrained observer. Therefore in dental conditions, where the important changes are relatively small in size, it is important to have radiographs which will show these details. Fortunately the close proximity of the radiographic film to the tooth enables a sharp and detailed picture to be obtained which will permit of magnification, and as the danger of the lesion is not to be judged by its size, such magnification will not mislead by showing the slight changes to be definite irregularities.

The outline on the radiograph of a normal tooth enlarged to the size of the radiograph of the femur head is seen to be quite as regular as the latter, whereas the outlines of teeth which show "slight" changes are markedly irregular—a femur head so irregular would be judged to be gravely involved.

Weston A. Price, who with a team of dental surgeons, bacteriologists, and laboratory workers has carried out a very extensive investigation on dental sepsis, considers that 10 per cent. of the teeth with septic roots give no indication of this on the radiograph; but even so, most of the process illustrations which he used in describing the "failure" of the radiographs show definite though "slight" changes, yet process illustrations cannot give the detail of the original radiographs.

There is no doubt in my mind that the figure of 10 per cent. can be considerably reduced by the skill in the radiographic technique and the care exercised in examining and interpreting the radiograph. Further, I am of the opinion that, *every tooth which is producing systemic disturbance shows definite changes on the radiograph.* The importance of this is apparent when we remember that the clinical tests, even when skilfully employed, may fail to reveal quite extensive lesions.

Burchard and Inglis state that "In cases of granuloma or blind chronic apical abscess there may be no subjective or local symptoms of disturbance cognizable to patient or operator."

A patient suffering from a toxæmia was sent for an x-ray examination of her teeth. She was annoyed at the suggestion that her illness was due to her teeth, and informed me that she paid regular visits to her dental surgeon, who had recently carefully examined the teeth and reported them to be quite sound. (See Fig. 1.)

Price, who has made an exhaustive study of the individual and family clinical histories, clinical, radiographic, and bacteriological examination of the teeth of 681 selected cases in a series of 1,400, has classified patients into three groups. He says that dental infections tend to produce the same type of tissue reaction around the teeth of different members of the same family.

Group A.—The radiographs of the teeth of this group of patients show very extensive rarefaction around all the involved teeth, and often extensive pyorrhoea (as in Fig. 2). He regards this local bone absorption as a sign of a good reaction and resistance to the infection on the part of the patients. Such patients, he says, rarely show symptoms of the rheumatic or degenerative disorders, and he accordingly classes them as having "absent susceptibility." Nevertheless, such patients come to hospital complaining of gastro-intestinal or other disorders probably brought about by the septic teeth. Clinically the gums show discharging sinuses, and the teeth are often loose, readily anaesthetized, and easily extracted; the sockets heal with great rapidity and without discomfort or secondary infection.

Group B.—In this group of patients the radiographs of the teeth show similar changes to those in Group A, but the periapical areas of rarefaction are bounded by a zone of sclerosed and therefore denser bone (as in Fig. 3). He regards this sclerosed bone as a sign of breaking down of the local resistance to the dental sepsis with the entrance of the septic material into the blood stream. Clinically there are signs of old fistulae and a history of former tenderness. Anaesthesia is less easily produced in patients in this group than in those in Group A. The teeth frequently are difficult to extract, and the sockets do not heal so readily. He says that these patients had acquired a susceptibility to the dental infection, and that, while no rheumatic symptoms were seen when the condition was acute, such symptoms had begun to appear, and they cleared with the removal of the septic teeth.

Group C.—The radiographs of the teeth of this group show condensing osteitis around the infected root with little rarefaction (as in Fig. 4). This appearance he regards as a sign of lack of local resistance to the dental sepsis. Clinically there is no evidence of fistulae; the teeth are seldom tender. They appear to be more liable to caries, but are usually free from pyorrhoea. Anaesthesia of such teeth is far from easy, and they are often very difficult to extract. There is great tardiness in healing, and the sockets tend to become infected and painful. Price says that these patients have an inherited susceptibility to the rheumatic group of disease.

This classification tends to explain why some patients with extreme and obvious dental sepsis have often few of the symptoms usually associated with septic absorption, while others with marked rheumatic symptoms have often no obvious dental sepsis, and the radiographs of their teeth show relatively slight changes—so slight that when indicated by the physician or radiologist the dental surgeon is loath to extract the teeth; yet it is often in patients with these "slight" dental lesions that extraction produces marked beneficial effects. As these important indications are relatively slight it is all the more essential that the radiographic technique should be good.

Most physicians will agree that these dental groupings are correct with regard to the susceptibility to rheumatic infections, but few, I think, will agree with the ideas on local resistance.

An examination of the radiographs showing bone sepsis of different types permits of the classification into three groups which resemble those indicated by Price.

Group (a).—Patients with bone sepsis due to pyogenic bacteria. The radiographs in these cases show rapid absorption or erosion of the infected bone, and frequently a large amount of pus is found. As the condition of the patient improves and his resistance is increased the rarefied bone increases in density and the patient shows little or no sign of rheumatic disorders, even though the degree of septic absorption, as judged from the patient's condition, was very severe.

Group (b).—Patients who formerly showed radiographic changes similar to those in Group (a), but, owing to the separation of sequestra or the inclusion of some septic foreign body, healing is very slow, and the radiograph shows that the

FIG. 1.—Radiograph showing large "cyst" (probably arising from the root-filled bicuspid) which has eroded the apices of the first bicuspid, canine, and central and lateral incisors. Patient had no local physical signs or symptoms, but a toxæmia which cleared on extracting teeth.

FIG. 2.—Radiograph showing extensive alveolar and periapical absorption.

FIG. 3.—Radiograph showing a large apical abscess with sclerosis of its bony wall and erosion of the tooth apex. The lateral incisor is root-filled and there is a little erosion of its apex.

FIG. 4.—Radiograph showing small areas of rarefaction at the apices of the bicuspid and molars with sclerosis of the surrounding bone. Patient suffered from pains in the back and along the course of the sciatic nerve as well as in the shoulder-joints.

FIG. 5.—Radiograph of molar with one root filled with opaque material, the other showing no sign of a nerve canal. There is sclerosis of the pericemental bone and secondary caries beneath the amalgam filling. Patient suffered from neuritis, which improved on removal of the tooth.

FIG. 6.—Radiograph showing retained stump with large abscess.

FIG. 7.—Radiograph of incisor teeth showing destruction of the pericementum around the apices of the central and lateral incisors and extensive destruction of the cancellous bone around. There is no definite boundary to the destructive process. Note the pulp stones in the central incisors.

FIG. 8.—Radiograph showing a large cyst with erosion of the apices of the central and lateral incisors which have had the pulps extracted and the root canals partly filled.

FIG. 9.—Radiograph showing "bulbous" roots to root-filled molar with periapical absorption.

FIG. 10.—Radiograph showing large area of periapical absorption with erosion of the apices of the first bicuspid, which shows secondary caries beneath the filling. There are similar but less marked changes in the second bicuspid.

FIG. 11.—Radiograph showing sclerosis of the pericementum due to the large filling and a normal antrum.

FIG. 12.—Radiograph showing erosion of the wall of the antrum and of the approximated root-filled bicuspid pericementum with dimming of the antrum.

FIG. 13.—Radiograph showing apical absorption around central incisor, not shown on "flat" radiograph.

FIG. 14.—Radiograph showing root-filled lateral incisor and canine. On the bicuspid side of the canine the pericementum will be seen pushed away from the side of the middle of the root. There is marked erosion of the apices of both teeth and very little root-filling, which shows signs of shrinking and retraction.

FIG. 15.—Radiograph of root-filled lateral incisor and canine, showing retraction and shrinkage of the root-filling preparation. The nerve canals are shown to be much wider in calibre. There is also an area of rarefaction of the periapical bone of both teeth and erosion of the apex of the lateral incisor.

FIG. 16.—Radiograph showing marked shrinking of the root-filling preparation and septic erosion of the apex of the tooth.

FIG. 17.—Radiograph of root-filled upper molar teeth, showing retraction and shrinking of preparation in anterior root and erosion and absorption around the distal root, which shows calcareous deposits. The floor of the pulp chamber had been perforated, and a deposit of calcareous material is shown on the projecting root-filling.

FIG. 18.—Radiograph showing root-filling in bicuspid roots; the filling projects beyond the apex in one, and an area of rarefaction has been produced in the surrounding bone.

FIG. 19.—Radiograph showing root-filling in bicuspid roots with areas of apical erosion and absorption. A small fragment of an instrument had been broken at the apex of the root and is lying in the rarefied area.

surrounding bone is sclerosed and very dense. This sclerosis most of us look upon as of the nature of a protective reaction against a chronic infection, and not as a breakdown in the resistance of the patient, though such patients often do gradually develop rheumatic symptoms, due probably to the long period of slow septic absorption.

Group (c).—Patients suffering from non-specific infective arthritis. The radiographs of the infected joints do not suggest ordinary bone sepsis; rarefaction, erosion, and sclerosis may be seen, but the changes are slow to form. They resemble the changes in the teeth of Group C. Numerous bacteriological researches have been made to attempt to discover the causal organism; various organisms have been suggested, but the lack of uniformity in the findings suggests that the condition is produced either by different organisms or by some bacteria or toxin which has not been discovered.

Price has shown that septic teeth from patients in this group, even when boiled, retain some toxin which is fatal to experimental animals. He found, too, that extracts from such teeth passed through a Berkefeld filter are also toxic to animals.

One of the most important points that Price has brought out is that the septic teeth from which absorption is taking place in patients with an inherited susceptibility to the rheumatic and degenerative disorders produce very little in the way of local clinical signs, which are small in magnitude. The fact that the patient has edentulous gums does not exclude the possibility of hidden dental sepsis. It is a fairly common thing to find that such patients have a retained stump which shows marked periapical erosion indicative of sepsis (as in Fig. 6). An x-ray examination may show definite evidence of apical sepsis of teeth which have not been filled and which show no evidence of caries. In some cases infection of the periapical area leads to the development of large cysts, which in the upper jaw may be mistaken for extension of the antrum (as in Fig. 8), but they can be readily distinguished from the latter by the fact that in the dental cyst the apices are denuded of the pericementum, whereas with the antrum extension the wall of the latter appears to be folded around the apices. Concomitant with the periapical erosion hypercementosis may occur, and the roots may assume a bulbous appearance on the radiograph. Such teeth may be very difficult to extract. In one case recently, where the radiograph showed similar changes to Fig. 9, the dental surgeon had great difficulty in extracting the teeth, and he told the patient that there had been a mistake in the radiographic report as the tooth was too firmly held to have been diseased. Brubaker reports that neuralgia, functional blindness and deafness, chorea, epileptiform fits, paralysis, cardiac neuralgia, insanity, and other related conditions have been cured by the extraction of hypercementosed teeth. In contradistinction to hypercementosis we have erosion of the apices as a result of sepsis (as in Fig. 10). When the infected apex is near the antrum the infection may pass into the antrum (as in Fig. 12).

Radiographs with less marked changes are frequently seen, as in Fig. 13, which shows a large area of rarefaction, due to acute infection chiefly to the lateral side of the root-filled lateral incisor. The root-filling is not very opaque. It will be noted that the sclerosed pericemental bone is "wiped" out about half-way to the apex on this aspect, and to a lesser extent on the medial aspect of the lateral incisor. The area of rarefaction may not always be at the apex of the tooth, since many teeth have lateral canals through which the pericementum may be infected.

THE PULPLESS TOOTH.

A tooth which has been deprived of its pulp has lost its vital power, and inasmuch as it contains within its unfilled nerve canals and dentinal tubules (estimated as measuring three miles in length) dead organic matter which is brought into association with the circulating blood through the dentino-cemental foramina, it is liable, even if sterile when the pulp was extirpated, to be infected by any organisms circulating in the blood. It has been shown that, even when caries has only extended a quarter of the way through the dentine towards the pulp, approximately 50 per cent. of the pulps of such teeth are infected, and further, that it is impossible, with the most scrupulous care and the use of the best known bactericidal medicaments, to

be certain that the infection present has been eliminated. It is probably very rare for the nerve canals to be completely filled, even though the canal be simple and straight, as most of the substances used cannot be made to fill the canal accurately, and owing to absorption of the solvents used in the preparation the filling contracts and leaves a space which, with the dentinal tubules, is unprotected by any vital agents and is liable to become the storage place for bacteria and toxins. Price says:

"It is to be remembered that a small portion of a gangrenous pulp beneath a root-filling is equivalent to an entire gangrenous pulp as a cause of pericementitis. The vast majority of cases occur as a sequel to putrefaction of the pulp, either before or after instrumentation or as a result of infection of the apical tissue by instruments either unsterilized or reinfected by contact with oral fluids, septic fingers, etc."

When the nerve canal is delta-like, or has lateral openings, the pulp remnants cannot be removed, nor can the canals be suitably filled even with impregnation methods. To get out of this difficulty some authorities respect the apex of the tooth.

Bacteriological examination has shown that, no matter what medicament is used in the treatment of infected apices, dressings left in for forty-eight hours are found to be infected, and that even when all care has been used root-filled teeth may be a source of great danger to the patient. Experimentally it has been proved that such root-filled teeth may contain bacteria or toxins which, together or separately, produce toxæmia and death when inserted into animals.

Mayo says:

"Although the possessor of a tooth with dead pulp without local reaction may be enabled to eat better for a time, he is conducting his health on borrowed capital, as he may not have a physician or dentist who will appreciate that the disease or broken health that may develop is due to such an apparently trivial cause that persons comparatively well may later develop a variety of diseases that destroy health and happiness, if not life."

While Inglis says:

"At present it looks as though no root-filled tooth can be considered other than as a life risk, while sterile filled roots can be infected from other sources of infection, of which there are many."

RADIOGRAPHIC PITFALLS.

The errors in dental radiographic diagnosis may be due to (a) faulty photographic and radiographic technique; (b) misinterpretation of the radiograph.

With regard to (a) it can be said that the manufacturers have done their best to produce an x-ray unit which is as simple in working as any snapshot camera. It is common knowledge that with either kind of apparatus the proportion of films of good quality is dependent upon the care, skill, and experience of the operator. The simplicity of the apparatus will enable the most ignorant beginner to obtain chance successes, and these chance successes are stored and exhibited to give a sense of self-satisfaction which tends to prevent further progress. It is not to be wondered at, therefore, that marks on radiographic films due to faulty technique may (1) be interpreted as pathological changes, which are not found at subsequent operation, (2) mask a pathological lesion which is present. I have known of a number of cases in which the pathological lesion was missed because the films were flat. Fig. 13 is an example of such a case. No amount of clinical or pathological knowledge will enable anyone to give a diagnosis from a faulty film.

Misinterpretation of the Radiograph.

This is due chiefly to: (1) lack of experience in interpreting radiographic shadows; (2) lack of knowledge of general and dental pathology.

With regard to (1) every radiologist who has correlated the radiographic and clinical, operative or *post-mortem*, findings knows that the radiograph may show only slight changes even when a massive lesion is present. Thus with acute inflammatory lesions of the skeletal tissues the radiograph may give no sign; even with the enormous development which one sees in some cases of periosteal sarcoma the radiograph may show nothing abnormal or perhaps a little localized thickening of the periosteum. It can almost be said, particularly in acute conditions, that the more attractive the physical signs, the less the radiographic signs.

In the case of dental radiography, as the lesions are often small, it is all the more important to pay attention to the finer details.

As regards (2) there is no question that for the best interpretation of radiographs the observer must have a good knowledge of the normal and pathological anatomy of the part. Most books dealing with the radiography of the teeth instance the interpretation of the mental and palatine foramina and loculi of the maxillary antrum as areas of periapical absorption, so that these mistakes are hardly likely to be repeated except by the beginner; even if they are, the worst that can happen is the sacrifice of one or perhaps two sound teeth.

The result of the wide knowledge of the faulty interpretation of these foramina has no doubt been responsible for the much more grave error which is brought to one's notice from time to time—that is, the interpretation of a definite periapical absorption as a normal foramen or sinus. It would almost seem that the larger the cyst the greater the possibility of it being interpreted as a shadow of the antrum. (See Fig. 8.) This is of great importance, because the systemic disease from which the patient is suffering continues or increases in severity as the suspected cause has been overlooked and allowed to remain—there being no local signs apparent to the patient or the clinical observer.

BIBLIOGRAPHY.

- Burchard and Inglis: *Dental Pathology and Therapeutics*. Henry Kington, London, 1927.
 Colver, J. F.: *Dental Surgery and Pathology*. Longmans, Green and Co., 1923.
 Goadby, Sir Kenneth: *Diseases of the Gums and Oral Mucous Membrane*. Oxford Medical Press, 1928.
 Hess, Walter: *The Anatomy of the Root Canals of the Teeth of the Permanent Dentition*. John Bale, Sons and Danielsson, 1925.
 Hopewell-Smith, A.: *The Normal and Pathological Histology of the Mouth*. J. and A. Churchill, 1919.
 Howe, Percy R.: The Focal Theory of Infection in its Application to Teeth. *National Dental Assoc. Journ.*, vii, 1920, 635-641.
 Mackey, Leonard: Dental Sepsis and Apical Infection. *British Dental Journ.*, September 1st, 1922.
 Mummery, J. Howard: *The Microscopic and General Anatomy of the Teeth*. Oxford Medical Publications, 1924.
 Price, Weston A.: *Dental Infections—Oral and Systemic*. The Penton Publishing Company, Cleveland, Ohio.
 Rickert, W. G.: The Status of Pulpless Teeth. *Dental Cosmos*, lxxviii, 1170-1178.

SOME PROBLEMS OF GLYCOSURIA.*

BY

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THE introduction of insulin in the treatment of glycosuria has enabled us to overcome the urgency of diabetic coma in a way never before possible, but many problems remain.

Can the administration of insulin be regarded as a replacement therapy? There is much evidence that such is the case. It is usually recognized that the respiratory quotient rises under its influence from 0.7, indicating metabolism of proteins and fats, to unity, indicating the metabolism of carbohydrates. The glycogen store has been shown to increase very materially under its use. The blood sugar curve—that is, the amount of sugar in the circulating blood—is very materially altered; whereas the administration of glucose alone led in one case to a rise from 0.2 to 0.4, the combined administration of glucose and 20 units of insulin resulted in a fall to 0.05.

Regarding insulin, then, as an efficient replacement therapy, we are still left with very definite limitations.

1. The amount of insulin required depends upon the amount of carbohydrate to be dealt with.
2. The insulin must be given at the time of carbohydrate absorption.
3. The duration of maximum insulin efficiency would appear to be only four hours.
4. The preparation must be given hypodermically.

A consideration of these factors at once shows the very serious limitations of this line of treatment. The daily life of most persons includes four meals, which vary in themselves, and from day to day, as to their carbohydrate content. A complete replacement therapy would therefore demand four doses of insulin, given hypodermically, in

quantities calculated to deal accurately with the carbohydrate content of each individual meal. This is quite impossible, and it is found that patients are very reluctant to tolerate more than two hypodermic injections daily. The diabetic patient under insulin treatment, therefore, finds himself still limited as to time, quantity, and quality of food; he must be on a diet as rigid as heretofore, although containing material previously forbidden; and, in addition, must tolerate two hypodermic punctures every day. Two of the meals will be relatively generous, while the other two are designed to contain the minimum amount of carbohydrate.

There are two schools of thought in the composition of diabetic diets. One believes that the best results are obtained by keeping the patient on the lowest diet which will maintain weight and strength, and, in consequence, the minimum possible daily dose of insulin. The other school holds that a generous diet allowing for plenty of available metabolic material, with a relatively large dose of insulin, is the better line of treatment. A careful paper by Rabinovitch in the *Quarterly Journal of Medicine*, in which the results of about 1,200 cases have been analysed, has led him to the conclusion that a minimal diet yields the best results. This is the type of conclusion usually arrived at by a statistical method, but it is liable to omit the human factor. It is a matter of common experience that diabetics generally not only have larger appetites in consequence of the disease, but that they have always liked or required quantities of food beyond the average. If this be so, it is possible that treatment including a generous diet will be more satisfactory to the patient and be more likely to be maintained; and it will prove much less irksome than treatment along rigidly abstemious lines. It is possible that medical practitioners generally underestimate the limitations from which these patients suffer; the continued dieting, the regularity of their meals, and the ever-recurring hypodermic punctures, with careful consideration of any food provided outside their own homes, make for a monotony which usually proves hard to bear, and the more rigid its limitations the greater the mental load. If insulin can legitimately be regarded as a replacement therapy, then, in my opinion, it should be used to replace so far as it is practicable; the introduction of double strength insulin has enabled this to be achieved more easily than was the case when only one strength was available.

The relation of proteins, fats, and carbohydrates in the total diet offers a further problem to which no generally accepted answer has yet been found. Anything approaching the usual proportions of the average meal in this country would involve the use of impossibly large doses of insulin. Even the proportion found in milk (protein 3, fat 4, carbohydrate 5) requires relatively large injections, but is probably a basis which yields good results.

Certain difficulties present themselves in relation to groups of cases. The man who has to earn his own living by hard labour obviously needs a more generous diet than the well-to-do woman with no particular occupation. The dieting of diabetic children offers great difficulty, for they must be given suitable food in sufficient quantity to permit of adequate growth, and they are nearly always severe cases. It has been my experience that no satisfactory result can be obtained among the younger patients unless three insulin doses a day are given.

Changes in dieting when the insulin dose has been established are from time to time necessary, the commonest cause, perhaps, being loss of appetite which results from some pyrexial illness. There is no doubt that this constitutes one of the greatest dangers to the diabetic to-day. If the patient does not take the carbohydrate part of his diet the insulin would appear to become unnecessary. It frequently happens that a diabetic patient contracts influenza, or some other disease associated with a rise in temperature, and is put on a low diet without insulin. The metabolic processes are increased with the rise in temperature, the carbohydrates being the chief source of heat production; in this respect the diabetic fails completely, with the result that the proteins and fats are inadequately split, a rapidly rising ketosis occurs, and coma becomes imminent. The occurrence of pyrexia in a

* A paper read before the Lancashire and Cheshire Branch of the British Medical Association.