other parts of the book, notably in the chapter on hydro-nephrosis, which is the best of the chapters that deal with the kidney.

There is little that is original, and the author has drawn largely on the wealth of German and French urological literature. He appears to have little acquaintance with modern English practice. For instance, if he were to use the aëro-urethroscope invented by Hurry Fenwick and perfected by Wyndham Powell, he would not describe ulcerations and erosions of the urethra. These ulcerations are never seen with the aëro-urethroscope, which, being passed down under air distension, under control of the eye, does no damage to the urethral mucosa.

The chapters on gonorrhoea are a little disappointing, as they are very prolix on theory and not sufficiently clear on details of treatment, and it is not of practical assistance to discuss prostatitis in a separate chapter under the heading of diseases of the prostate, when it is part and parcel of most cases of gonorrhoea. We are glad to read a book which is an earnest advocate of local treatment from the very onset of acute gonorrhoea, but we are surprised to read that the author has obtained better results with injections than with Janet's irrigations.

The chapter on serum and vaccine therapy is excellent in theory, but here again it is disappointing in practice, as none of the author's own detailed results in this line of treatment are given, and we should be surprised to learn that they are as roseate as the remarks on theory would lead us to believe.

A good feature of the book is the excellence of the illustrations; those illustrating operative surgery are very clear and useful, and Plate III, a coloured plate of conditions seen by the cystoscope, is a masterpiece.

NOTES ON BOOKS.

THE first volume of the *British Journal of Surgery*¹⁰ presents a very handsome appearance owing to its ample page, well-spaced type, and numerous illustrations. It comprises the first four numbers of the periodical and contains some 750 pages. The papers are for the most part of a technical sort, and will appeal mainly to operating surgeons, but each number in the volume has as a frontispiece a portrait followed by a brief biography of an eminent surgeon of the past. Those in this volume are Lord Lister, John Hunter, Sir Astley Cooper, and John Abernethy. Moreover, there are accounts of visits to certain surgical clinics at home and abroad, and some notes—anonymous, as we observe—on “instructive mistakes,” which will be read with interest by every practitioner.

A useful little work on *Health Preservation in West Africa*¹¹ has been composed by Dr. J. C. RYAN, and Sir Ronald Ross has written an introduction to it, in which he describes the remarkable improvement in the health of Europeans which has taken place on the West Coast in the last ten years. He believes that Dr. Ryan's book is full of wise hints and of information useful to everyone, and in this we heartily agree. All the tropical diseases usually met with in Africa are dealt with. Due importance is given to sanitation, and in addition the subjects of dietary, exercises and treatment find a place. If the laity will only peruse its pages they will gain information which will save them from much trouble and sickness.

¹⁰ *The British Journal of Surgery*. Bristol: John Wright and Sons, Ltd.; London: Simpkin, Marshall, Hamilton, Kent, and Co., Ltd. 1914. (30s. net.)

¹¹ *Health Preservation in West Africa*. By J. C. Ryan, L.R.C.P.I., L.M., L.R.C.S.I., L.M., with Introduction by Sir R. Ross, K.C.B., F.R.S. London: John Bale, Sons, and Danielsson, Ltd. 1914. (Cr. 8vo, pp. 108; 1 illustration. 5s. net.)

THE tenth Italian Congress of Midwives will be held at Venice on June 14th and two following days. The Sanitary Chamber of Venice has consented to form the Executive Committee of the Congress, with Professor Guicciardi as chairman.

THE Italian Medico-Parliamentary Committee at a recent meeting discussed the conditions of service of health officers, the hygienic duties of naval and military medical officers, and the modifications that should be made in the law regarding hospital appointments. A draft scheme was submitted for a conference of the directors and medical staffs of hospitals to study various questions relating to hospital administration which it is proposed to hold in Rome.

DENTAL SEPSIS.

By J. G. TURNER, F.R.C.S.

STRICTLY speaking, dental sepsis means foulness of the teeth (Gr. *σήπο* = make putrid); but the meaning of sepsis has been enlarged to indicate, in a loose, indeterminate fashion, the presence in noxious amounts of any germs and their products other than a few well-recognized varieties—the so-called specific germs—such as *B. tuberculosis*, *B. diphtheriae*, *Treponema pallidum*, etc. It may be premised, however, that the word is not ill-chosen, since a foul smell is present in most cases of dental sepsis.

The term “dental sepsis” is here proposed because it indicates the dependence of the septic condition on the presence of teeth. Just as well as the term “oral sepsis,” it can be made inclusive both of results and causes—of pyorrhoea alveolaris, dental caries and abscess, and tartar, which are found in varying proportions in a septic mouth, as well as of the germs, with or without decomposing food and debris, which constitute the original state of sepsis.

Nowadays no one will be found to dispute the existence of dental sepsis; but while on the one hand many regard its recognition as almost revolutionizing the practice of medicine, and as having a large influence on surgery, others are inclined to think that too much is made of it. If we put the former view in its extreme form, we may say “every mouth is septic, and wherever there is sepsis there is infection, actual or potential.” The latter view we may similarly epitomize thus: “There are germs in every mouth, but only seldom in such amount as to be injurious; moreover, the individual may be ‘salted’ by his mouth-germs.”

DEFINITION OF TERMS.

Consideration of the following facts may help to a reasonable attitude towards dental sepsis. To begin with, the conditions of dental sepsis and the terms used in describing them need consideration.

Septic Organisms in the Normal Mouth.

The simplest condition of dental sepsis is that in which the oral tissues are healthy and the teeth sound and free from deposits of tartar, but in which germs of various sorts are to be found harboured among the teeth, and, to a lesser extent, free in the oral fluids.

This is the normal mouth; it is healthy, but it is a potential source of danger.

Pathological Consequences of Sepsis.

The deviations from this normal condition are to be traced on the one hand to the deposition and stagnation of germs, food, and other foreign matter, including lime salts of the saliva forming tartar, on and among the teeth, and, on the other, to the many forms of tissue reaction exhibited by soft tissues and bone to injury (used in its widest physiological sense).

Starting from this condition of normal sepsis—a condition which is universal among animals living in a germ-laden environment—a point is soon reached at which the microscope is no longer needed to demonstrate the presence of bacteria; they are present in tangible masses as a greasy white deposit sticking to the necks of the teeth. The mouth is now definitely septic, and the inflamed state of the edges of the gums shows that they, at least, resent the presence of this deposit.

Solid Stagnation.

Leaving dental caries aside for the moment, the earliest abnormal state to which a distinct name may be given is that in which, although the gums are clinically or to the naked eye healthy, yet from under the gum-edge ~~there~~ may be squeezed up a pultaceous mass shown by the microscope to be almost entirely composed of germs. This may be called the stage of solid stagnation.

Often this pultaceous mass has a fetid smell, and we have the earliest stage of putrid stagnation, depending in this case probably on decomposition of mucus, dead epithelium and leucocytes, and comparable to the mucus plugs found in the tonsil.

Putrid Stagnation.

Putrid stagnation may next be noted, since it is a condition which may occur without visible tissue changes,

though generally it is associated with the tissue changes included under the term "pyorrhoea alveolaris."

Apart from the condition just mentioned, it is due to the decomposition of food debris among and around the teeth, and in the cavities of carious teeth.

MARGINAL GINGIVITIS.

The earliest clinical sign shown by the tissues (other than teeth) is a chronic or subacute gingivitis. The edge of the gum is blue or red, and swollen; the interdental pad of gum protrudes instead of receding slightly between the teeth. The condition may be general or localized to the gum around one or two teeth.

In a short time another clinical sign is to be found. The sharp line of demarcation which normally exists at the junction between the mucous membrane of the cheek or lip, which has so copious a blood supply, and the tightly bound down, pink muco-periosteum (or gum) disappears on account of inflammatory engorgement of the latter. This line is well called by American dentists the health line. The gingivitis may develop into a persistent purulent form, the pus coming from the toothward surface of the gum. A generalized purulent marginal gingivitis of this type is not common, but in all cases of persistent marginal gingivitis small amounts of pus can be demonstrated by the use of a fine steel probe. In this condition there is no deepening of the periodontal sulcus except by swelling of the gum edge.

Oedematous and true inflammatory overgrowth may next lead to local or general hypertrophy of the gum margin.

"PYORRHOEA ALVEOLARIS."

From the stage of marginal gingivitis we pass to destruction of gum and formation of pockets. This is the stage which is spoken of as "pyorrhoea alveolaris." The term means "flow of pus from the alveolus," and is applicable strictly only to those cases in which pus can be seen, clinically, to exude from the sockets. It is but one stage in a generally chronic destructive disease of the alveolus—a stage, moreover, which is not necessarily present at any period of the disease. In so far as it tends to rivet the mind on one sign of disease and to make that sign the one index of danger it is a term which should be discarded, since it is abundantly evident that even in its absence danger may and does exist. If, however, it be kept to its original use, or if it be agreed to include under the name "pyorrhoea alveolaris" the whole range of inflammatory tissue reaction to septic infection gaining access through the periodontal sulcus, then in one of these senses the term may be useful.

The tissue reaction is generally subacute or chronic, but sometimes acute or even fulminant.

The acute reactions take the form of abscess or even rapid spreading necrosis, when the disease is generally reported as acute septic necrosis of the jaw. The acute abscess often appears in connexion with a tooth which has a living pulp and in a mouth showing little obvious pocketing, destruction, or even swelling of the gum; indeed, the mouth may look surprisingly healthy. Sometimes it is impossible to trace the breach of tissue by which infection has reached the part of the tooth socket at which the abscess has formed, and tissue infection by a living organism seems the only explanation.

It is important to have a clear conception of the normal gum, pale pink in colour, thin edged, closely applied to the neck of the tooth, with slightly receding interdental pads filling in the interdental spaces. The subacute and chronic reactions are the more common. They show themselves in chronic hyperaemia of the gum; in local and general oedematous swelling of the gum; in local or general hypertrophy of the gum; in local or general destruction of the gum—disappearance of the interdental gum pads and denudation of the teeth; in local bony overgrowth forming bosses over the roots of the teeth, which later may fuse together into shelves or ledges, running almost round the mouth, mostly on the outer side; in sclerosing osteitis; in general bone hypertrophy leading to great thickening of the alveolus; or to general osteosclerosis with, in the mandible, enlargement of the body of the bone; in chronic osteoporosis and destruction of bone, leading to the formation of pockets and eventually to loss of the teeth; in pus formation, which may be plentiful and clinically demonstrable or at no time more

than microscopic. The caries may be purulent or dry. The dry form may change to, in fact generally ends in, the purulent; the purulent may be arrested so far as to become dry.

Pain is not a frequent symptom—or at least not such as to bring the patient to the dentist—but its existence may often be elicited on inquiry, especially in the subacute cases, where the gums are congested, and in these cases, as happens when other mucous membranes of the head are engorged, there is often pain on stooping or running. There may be pain due to irritation of the exposed roots of the teeth, though these are generally protected from outside influences by a layer of tartar or putrifying debris.

The engorged gums bleed readily, leaving no trace of breach of surface. There is often excessive secretion of saliva and mucus and accumulation of mucus in the mouth or pharynx during the night. The accumulation of decomposing food and debris among the teeth and in the pockets leads to a foul taste in those who do not become habituated to their condition, and to foul breath.

The teeth may become loose, a condition which may be due to two factors—(1) loss of attachment, and (2) replacement of surrounding bone by granulation tissue. Or they may be firm, though but little attachment is left, as in the slow dry form of disease, or more firmly implanted than normal owing to sclerosing osteitis.

The whole process culminates in a general septic stomatitis, chronic or subacute, with swelling of lips and tongue, sore, oedematous mucous membranes, puckering and fissuring of the tongue and cheeks, thickening and heaping up of epithelium, or atrophy of the mucous membranes and thinning of the epithelium.

Caries of teeth results from the stagnation of germs and carbohydrate food. It is itself dependent on dental sepsis, and in turn adds to it. The cavities in decayed teeth form reservoirs for germs and decomposing food. They are nearly always foul smelling. Later, decomposing tooth pulps and the discharges of chronic dental abscesses add to the general sepsis. Dead roots are seldom other than septic. Once the dental tubules are infected they are extremely difficult to sterilize. Clinically, on extraction granulation masses will be found adherent to most dead roots.

The normal anatomy of roots renders sterilization difficult. They are often curved, and a sharp bend of the apical portion is common. The root canal is seldom a simple round canal running from pulp chamber to apex. It may be dumb-bell-shaped, or it may take the form of a mere ribbon running down the root. Near the apex it may split into several minute canals, or turn off sharply to end on the side of the root.

In effect it is difficult, often impossible, to track out and sterilize a root canal once septic. It is equally difficult, if not impossible, to sterilize the surrounding dentine, owing to the difficulty of penetrating the dentinal tubules. It is not, then, surprising to find that most dead roots on extraction show signs, in the form of adherent granulations and absorption, of their presence having been resented by the surrounding tissues.

Nor is it surprising to find that the cementum, the only living part of the so-called dead root, has undergone exostosis, that is, inflammatory hypertrophy. These chronic changes are to be found frequently in roots implanted in apparently healthy alveolar tissues.

TARTAR.

Tartar occurs in two forms—a bulky light-coloured deposit found on the exposed parts of the teeth, and a thin dark deposit found under the gum edge. It invariably contains large numbers of bacteria, and the more bulky deposits entangle organic debris under their edges, and smell correspondingly putrid. Tartar is thus always septic, and the tissues in contact with it are always in a state of chronic inflammation or ulceration.

ARTEFACT STAGNATION.

The last factor in dental sepsis is the artefact factor. Rough fillings, fillings with overlapping edges, ill-fitting gold caps, with edges thrust up under the gum, ill-fitting crowns and bridges, all offer artefact stagnation areas, and are of necessity septic. Tooth-plates, as generally worn, are a fertile source of artefact sepsis. They have a faint sour smell, sometimes an actually putrid smell, and to

their surface adheres a thin layer of white greasy material consisting of germs and food debris. That the tissues resent their presence is shown by the state of the mucous membranes. Often the shape of a plate is accurately outlined by an area of inflamed mucous membrane, and the edges of the gum round the teeth against which the plate lies are practically always in a state of chronic inflammation. Where, also, these unclean plates lie against the natural teeth, caries is a frequent consequence, since the deposit on the plates contains the elements—germs and carbohydrates—needed for its onset. Thus is started a further round of dental sepsis.

DENTAL SEPSIS.

Of the existence of some degree of sepsis in every human mouth there can be no doubt. That it is a possible source of danger is equally indisputable, since various germs of well-known pathogenic powers are always present. The questions which the practical man wishes to see answered are:

- I. At what point does dental sepsis become a danger?
- II. How is its presence to be recognized?
- III. How is it to be prevented, and at what stage does it require treatment?

I. THE DANGER POINT IN DENTAL SEPSIS.

To find an answer to Question I, attention may first be directed to the teeth. Dental caries is a product of the stagnation of carbohydrates and germs of known and unknown pathogenic powers. In the strictest sense it is a result of dental sepsis. Moreover, it is so insidious in its onset that many people cavil at the statement "clean teeth cannot decay." The minutest stagnation area is sufficient for its commencement. Viewed in this light, the smallest amount of dental sepsis is a danger.

The alveolar tissues may next be considered. What amount of dental sepsis can they tolerate without showing resentment—that is, without showing clinical signs of inflammation? Bearing in mind the description of healthy gums given above, it would seem that they early resent the presence of even small amounts of dental sepsis. An absolutely healthy gum margin, even in early childhood, is a rare thing. Marginal gingivitis is to be found in almost every child around temporary and permanent teeth alike, and it is the first stage in the chain of pathological events leading to "pyorrhoea alveolaris," and all the varied manifestations of inflammatory reaction to injury enumerated above. From this point of view, also, a very small amount of dental sepsis becomes a danger.

There is another and extremely significant way in which the local tissues show their resentment of dental sepsis. The roots of septic teeth, both living and dead (that is, with live pulps or with dead pulps), become surrounded and absorbed by a bed of granulations, while at the same time they undergo inflammatory exostosis. The tissues, in fact, regard them as injurious foreign bodies. Absorption of septic permanent teeth is a very common event, and teeth of young adults are frequently in a state of well-advanced absorption.

Of 492 permanent teeth extracted at the Royal Dental Hospital of London, 301 showed to the naked eye either absorption or exostosis of the roots; and of 595 roots, 507 showed similar conditions. Absorption and exostosis were not limited to the root apices, but frequently affected the whole root or only a part of the root near the neck, pointing to the periodontal sulcus as the source of infection.

So far as local results are concerned, even small amounts of dental sepsis must be regarded as dangerous. But what of general effects? Can these small amounts by any possibility produce serious general effects? The answer is to be found in the law of attack and resistance. Among the constant or occasional inhabitants of the mouth, forming part of the general sepsis, are to be found *Streptococcus longus*, *Diplococcus pneumoniae*, *Staphylococcus albus* and *aureus*, and so long as resistance is good no harm ensues. But if resistance be lowered, as by prolonged exposure to cold, fatigue, undue fasting, it may not require more than a few pathogenic germs to bring about serious infection. Here, then, a small amount of dental sepsis becomes a dangerous condition.

Passing to the more pronounced conditions, what special

dangers do they possess? Think first of the decomposing food hidden in carious teeth and in pockets among and around the teeth. With every act of deglutition some of the products of decomposition are swallowed. The injury caused by this factor of dental sepsis is sometimes diagnosed as ptomaine poisoning. It may not, indeed, be true ptomaine poisoning, and *B. botulinus* may be absent, but the practical fact remains that putrid decomposition of food held up among the teeth is an extremely common condition. Its very frequency may breed contempt, but it cannot be more dangerous to swallow decomposing food from a plate than from among the teeth.

Think next of the condition of the tissues surrounding the septic teeth. Under the edges of tartar the tissues are in a state of acute catarrh or of actual ulceration; the track of a chronic dental abscess is practically an open ulcer of the bone; at the bottom of each "pyorrhoea" pocket is a patch, small it may be, of carious bone; the toothward surface of the gum flap of each pocket is in a state of chronic ulceration, demonstrable to the naked eye when these gum flaps are excised. I have examined a large number of these gum flaps after excision, and have always found them in a state of ulceration; the so-called normal pockets have not yet come under my observation.

The catarrhal, and much more the ulcerated, surfaces must afford a means of entry for injurious substances; the very condition of the tissues shows that they are fighting something they resent: germs and toxins—a term which may be used in a sense looser than the strictly bacteriological.

The possible extent of this absorbent surface may be roughly estimated by measuring the necks of the teeth and the depth of the periodontal sulcus. Normally this should be rather a potential than an actual sulcus; but when, as a result of persistent sepsis, the gum edge has become swollen, it may be allowed an average depth of $\frac{1}{8}$ in. This is before any deepening has occurred by destruction of tissue. Twelve molars may be allowed an average circumference of $1\frac{1}{2}$ in., twenty other teeth an average circumference of 1 in. We thus get a possible secreting and absorbent surface of $5\frac{1}{2}$ sq. in.—or, to be on the safe side, let us say 4 sq. in.—before the advent of pockets. With the advent of pockets any single tooth may account for an open ulcer of $\frac{1}{4}$ to 1 sq. in., and, as before stated, septic teeth are often held in place by a mere bed of granulation tissue, telling its own tale of infection. On the one hand, this enormous ulcer drains into the mouth, and, on the other, any failure of drainage means absorption into the general circulation. Can there be any doubt of danger? The condition is common, very common, but to deny its danger or its possibilities is to deny the validity of all modern pathology. The injuriousness of swallowing pus may be doubted, but it is unnecessary to lay stress on that factor alone. There are germs and putrid decomposing food to be swallowed and there are germs and toxins to be absorbed.

A reference to comparative pathology shows that the same train of events occurs in the lower animals. Dr. Plimmer, working independently in the prosectorium of the London Zoological Society, incriminates the "solid stagnation," described above, occurring under the edges of apparently healthy gums, as the source of infection in monkeys and allied animals dying of enteritis, and observations I have made, and specimens I have collected, by the kindness of the authorities, of animals dying in the Gardens show that dental sepsis in captive lower animals is productive of the same evils as in man.

When does Dental Sepsis become a Danger?

The answer to this question may be summarized as follows:

Every mouth may be a carrier of, for example, the *Diplococcus pneumoniae*, and may thus be a potential danger to the possessor or an actual danger to others.

Solid stagnation, cervical deposits of greasy material, putrid stagnation, tartar, dental caries, are tangible signs of danger. Clinical inflammatory signs, from the most acute to the most chronic, show that local resistance is breaking down and that general infection may follow.

Ulceration, pockets, loosening of the teeth, beads of pus, are gross signs of danger. Waiting for them is like waiting for enlarged glands or fixation to make the diagnosis

of epithelioma of the tongue—the diagnosis then is but a fool's diagnosis.

The dry form of alveolar destruction, the form which gives rise to an appearance of premature old age of the jaws, in which destruction of bone and shrinking of gum go on at an equal rate, is far less dangerous than the purulent form, in which the gum remains to form a pocket round the tooth. In this dry form drainage may be good till the bone between the roots of the molars is attacked, when drainage fails, and the condition becomes purulent. In other cases the dry variety gives rise to fibrous thickening of the gums and chronic enlargement of the alveolar bone, neither tissue at first undergoing absorption. This form also degenerates into the purulent, but earlier than that just mentioned. Putrid stagnation and pocketing among the teeth are likely to be discovered early in its course.

II. THE RECOGNITION OF DENTAL SEPSIS.

The second question, How is the presence of dental sepsis to be recognized? is best answered by describing a routine examination of the mouth. This examination must be very minute. The impression made at first sight may be, and often is, utterly wrong. Clinically, the gums may look healthy, yet pockets may be found—ulcerated and foul. The periodontal sulcus may be of normal depth, but its contents putrid. The point of discharge of a chronic dental abscess may at the moment be invisible.

Half an hour is often well expended in looking through an apparently normal mouth.

Note, first, whether the breath be foul; with the mouth open most people breathe through the mouth, and, willy-nilly, the observation is soon made. Sometimes it may be noted that there is no fetor when the patient breathes through the nose. Tonsils may also be a cause of foul breath, but in most instances the mouth is at fault. To incriminate the stomach the patient must be supposed capable of continuous and unconscious eructation. Note whether the oral secretions are in excess, viscid and ropy, or frothy. Take out any tooth-plates present, and note their condition. They should be absolutely free of smell or deposit.

As before noted, most tooth-plates have a sour or even foul smell, and are coated with a greasy adherent deposit of germs and food. Look especially in any receding part and on the insides of bands for this adherent greasy deposit. Note the condition of the mucous membrane underlying the plates and of the teeth touched by them. The mucous membrane, especially the gum edges, are often in a state of chronic or acute catarrh and the teeth decayed. Note the condition of the gums and the presence or absence of the health line mentioned above. Any inflammatory deviation from the normal is an indication of local reaction to injury. The gums may be firm and pale, but thickened by chronic fibrosis, and the alveolar bone enlarged or even bossy. The gums may be receding in one part of the mouth, hypertrophic or simply swollen in another, normal in yet another part. In young people marginal gingivitis is the condition generally met with.

Note the presence of obvious tartar.

As far as possible, press the gum over each tooth, both on the vestibular side (outside), and on the lingual side, in such a way as to squeeze any underlying deposit or pus out at the gum edge. Except along the vestibular surfaces of the front teeth, this is not an easy thing to do.

With a fine steel probe, probe gently round the whole circumference of each tooth for the presence of pockets, and note their depth and width, and whether bare bone is present at the bottom (this is rare). Note especially whether the pockets extend among the roots of the molars or upper bicuspid; both upper bicuspid may have two roots, but this is more frequent with the first. Note also the presence of subgingival tartar—it is to be found before pocketing, and as pocketing occurs it extends along the denuded root. Generally it is hard, dark, and very adherent, and slowly formed. Sometimes it is softer and light coloured, and is rapidly deposited under the edge of a slightly everted gum. Its presence is an indication of stagnation, and so of possible or actual sepsis. Pockets will often be found in unsuspected places—so-called normal pockets.

Wrap a wisp of cotton-wool round the end of a fine steel probe, and with it mop out the whole of every peri-

odontal sulcus and every pocket. In doing this the presence of visible pus is often revealed where none was shown on squeezing; for this reason the gums should be closely watched while the examination is carried on.

As each wisp of cotton-wool is brought out, note what is on it and smell it. The shallowest periodontal sulcus may contain pus and putrid stagnant material. If there seems to be no smell, rub the wisp between the dry finger and thumb and smell again; the broad warm surface often makes a putrid smell evident. This appeal to the nose sounds repugnant, but it is the only way available of recognizing putrid stagnation. In making this examination have several probes and antiseptic solution at hand.

Note whether the roots of the teeth are denuded and whether the denudation passes between the roots of the molars or upper bicuspid.

Note whether any teeth are tender on pressure by pressing or tapping each one in turn.

Inquire for a history of occasional pain and tenderness of all the teeth, for, like any other chronic condition due to infection, the periodontal inflammation of dental sepsis is subject to exacerbations and remissions.

Note how loose or firm each tooth is; a tooth, apparently well held by the gum, may yet be implanted in a bed of granulation tissue. Pressure on such a tooth sometimes suggests that it is seated in an air cushion. Loosening of the teeth is a sure sign that the tissues regard them as undesirable.

Examine the teeth for caries, and in probing remember that the denuded necks of teeth are often very tender.

Examine carefully all repair work—fillings, crowns, caps, bridges of all sorts. The surface of every filling should be smooth, and the cervical edge (that nearest the gum) demonstrably flush with the rest of the tooth. Towards the neck the contour of the surfaces of the tooth becomes concave, and to give a good finish to a filling becomes a matter of extreme difficulty. On the vestibular and lingual sides the edges of fillings may be examined with a fine steel probe; if it catches under the edge the filling overlaps, and must be condemned as affording an artefact stagnation area. Between the teeth the fillings may be examined by passing a strand of silk between the teeth lightly in contact with the surface to be examined, and, when as far down or up as it can be taken, pressing it firmly against the surface and drawing it vertically out again. If the silk have passed below the edge of the filling it will catch against any overlapping edge, but it must be remembered that fillings may reach so far beneath the gum that neither probe nor silk can reach the edge. In this case they are *ipso facto* condemned, since the chances of making a good edge in such a place are practically none. The same system of examination applies to crowns, caps, and bridges. They must be examined for overlapping joints and edges, and again any cap or bridge support thrust up under the gum is *ipso facto* condemned; it is impossible that it should be otherwise than septic. The cleansability of the "body" of bridges must be examined. Bridges that rest on the intervening gum and bridges with concave surfaces are from their shape uncleanable. Unfortunately, the prettiest bridges—those with porcelain face teeth—are most often fitted to the gum, and so most probably septic.

As already mentioned, dead roots—crowned roots are always dead roots, but capped teeth may or may not be "dead" before capping—are always open to the suspicion of internal sepsis. Examine "dead" teeth by giving them a tap sideways; tenderness is often elicited in this way when there is none on delivery of a vertical tap. If a "dead" tooth have been filled immediately after a living pulp has been killed and taken out, the reasonable probability is that it is aseptic; if, however, before filling a course of treatment has been necessary to cure a septic state of the root canals, it must always remain suspect.

Note the presence or absence of "roots"—they cannot be other than septic, a persistent blood congestion of the overlying gum may proclaim it, but apparently healthy gum may cover roots in a lesser state of sepsis. Examine for the openings of dental abscesses. They may be obvious in the form of discharging nipples, almost invisible as a minute pin-point opening surrounded by a small reddened area of mucous membrane, or a bead of white pus may be just on the point of breaking the surface. A white,

fibrous nodule or a puckered scar may mark the site of an old abscess.

Inquire for the history of absent teeth, and if there be uncertainty as to extraction in its entirety or as to its retention, a good x-ray picture may be of great help. The sinus leading to a septic buried root, or even to a retained wisdom tooth, may be extremely difficult of discovery.

Remember that children, even as young as 3 years, may suffer from pronounced dental sepsis. Dental sepsis is as prevalent among children as among adults.

(To be continued.)

ROYAL COMMISSION ON VENEREAL DISEASES.

THE following is the official report issued to the press by the Secretary of the Commission:

Mortality, Notification, Medical Education, Lectures to the Public.

At the thirty-eighth meeting of the Royal Commission on Venereal Diseases evidence was given by Sir William Osler, Regius Professor of Medicine in the University of Oxford.

Sir William stated that the official statistics of deaths published by the Registrar-General were totally inadequate to represent the actual incidence of venereal diseases; for instance, in 1910 the deaths from syphilis in England and Wales were given at 1,649, but this was a very unsatisfactory and incomplete estimate. It was necessary to take into consideration and add a very large number of deaths appearing under other descriptions. If regard were had to these, he thought that it would be safe to say that of the killing diseases syphilis came third or fourth. Gonorrhoea he regarded as one of the great disabling diseases, causing an enormous amount of ill health and playing a very large part in the production of blindness. Sir William laid stress on the importance of early treatment of these diseases, and was of opinion that every general hospital should provide in respect of them out-patients' accommodation and proper accommodation in the wards. It was part of the work of the governors of hospitals to provide for these diseases, and they ought not to be left out; in the past they had been too much neglected by the charitable public.

Sir William was in favour of compulsory notification of venereal diseases; he thought there was a possibility that this would result in some concealment, but that this risk might now be taken.

On the question of the education of medical students Sir William was strongly opposed to venereal diseases being dealt with as a special subject added to the curriculum, with special lectures, and an additional set of separate examination questions. He considered that if this education could be given in out-patient clinics and in the wards, and by general teachers it was as much as could be expected of the student, looking at the short space of time at his disposal, and the great congestion of the curriculum.

Sir William thought that it would have an immense effect if the public were instructed by means of lectures regarding venereal diseases. Lectures of this kind, he thought, should not be given by a layman, but by a well-trained medical man who should be provided with proper diagrams and slides. It would be a very useful thing if the lecture could be widely given to the senior forms of the big public schools, in the universities and at large institutions employing many persons. He was not in favour of teaching sex physiology or hygiene to young children.

Skin Affections: Treatment: Notification.

At the thirty-ninth meeting Dr. J. H. Sequeira, Physician to the Skin Department of the London Hospital, gave evidence. He stated that the statistics of adult cases treated in the London Hospital skin clinic during 1913 showed that 13 per cent., both in the case of men and women, were suffering from obvious syphilis of the skin and mucous membranes. In the case of the women the proportion in the primary and secondary stages was

smaller than in the case of the men, and this he attributed to the fact that a larger number of women were unaware that they were affected with the disease. He gave a number of instances of innocent syphilis, and said that in his hospital experience he found that a larger number of women were infected during their married life. With regard to the use of salvarsan and neo-salvarsan he was confident, as a result of his experience of a large number of cases, that these remedies provided a most powerful means of influencing the disease. He was, however, of the opinion that it was necessary to combine salvarsan and neo-salvarsan treatment with the use of mercury. By prompt treatment the risk of the spread of infection could be enormously diminished.

Dr. Sequeira was not in favour of compulsory notification of syphilis, as he thought it would result in many people seeking advice from quacks. On the other hand, he thought there were conditions in which a medical man should be armed with some power to prevent the spread of infection. It should be held to be the duty of a medical man to do all in his power to prevent the spread of infection, and he should be held to be immune from any penalties in the exercise of his duty.

Dr. Sequeira insisted strongly on the need for increasing the accommodation available in general hospitals. There should, he said, be no hindrance whatever to any patient receiving treatment, and the fact of his or her suffering from the disease should be the sole indication for admission.

THE CULTIVATION OF TISSUES IN VITRO.

A MICROSCOPIC and lantern demonstration on the cultivation of human and other tissues *in vitro* was given by Dr. David Thomson (Grocers' Research Scholar) before the Royal Society of Medicine on May 11th. The research was carried out in the Marcus Beck Laboratory in that institution under the directorship of Sir Ronald Ross, and as the work represented the first-fruits of this new laboratory, the author made a brief reference to the memory of Marcus Beck, and expressed his thanks to the President, Sir Francis Champneys, to the Council, and to Mr. MacAlister, the secretary of the society, for the facilities which they had given him.

The tissues were cultivated according to the technique employed by Carrel at the Rockefeller Institute. The tissue of embryonic chicks obtained from hatching eggs was largely employed, and also human tissue obtained from operations performed by Sir John Bland-Sutton at the Middlesex Hospital.

It was found that when a small portion of embryonic chick tissue was implanted in a medium composed of the blood plasma of a fowl one part and extract of chick embryo one part, the new growth radiated out from the original tissue on every side, without any tendency to produce a definite shape or structure. The original cells were for the most part round cells, but the new cells produced in this medium were as a rule elongated and tended to branch, so that the new tissue differed markedly from the appearance of the original material.

It was noticed, however, that in spite of this common characteristic the types of growth produced from various species of tissue differed from each other. Connective tissue produced a radiating outgrowth of long, thin, branching cells, whereas the new growth from epithelial tissue was more of the nature of a solid extension of flat cells which did not elongate or branch so much as in the case of connective tissue. Brain tissue produced long axis cylinders, and the tissue of spleen and bone marrow produced large numbers of amoeboid leucocytes of various kinds, and also red blood cells. The tendency to elongation and branching of the cells of the new growth appeared to be due to the solid, or jelly-like consistence of the medium. In a liquid medium composed of white of egg or a mixture of serum and plasma, the cells of the new growth remained round like those of the original tissue.

Growth *en masse* of definite shape or somatic growth *in vitro* was also obtained by placing a very young chick embryo—twenty-four hours old—in the plasma medium. It was found that this embryo increased in size while retaining its definite shape. If the toe of a young embryonic chick were placed in this medium it increased