certain internal remedies, I have, without disturbance of the patient's health, succeeded in restraining the cough within harmless limits, in averting secondary evils, in improving the general health, and in guiding the course of the malady to a somewhat speedier issue than otherwise it would have reached.

With respect to regimen I have to observe that in all the cases brought before me the patients were plainly overfed; that, in addition to ordinary meals, there were frequent "interludes" of different kinds of food, mostly preserved, and that alcoholic beverages of some sort or another were freely administered. For my own part, I am unable to believe that the laws of digestion are suspended because an organism is troubled with a malady of this kind, that there is no physiological limit to the administration of food in disease, and that you are not certain to make a bad fire when you keep putting too many coals upon it.

Improvement has almost invariably followed the enforcement of a simple, but liberal, dietary arranged into three, or at most four, meals a day, abstinence from alcohol, cold or tepid sponging, warm, but not too warm, clothing, active outdoor exercise, early hours, and general discipline. Poor platitudes these, it may be said. Yes; plainly so on paper, but not so in practice where they can effect so much and are yet overlooked so often.

Of local applications to the interior of the throat, only two have done good service; the first is glycerine of borax with oxychloride of bismuth and morphine, and the second is the same combination with the substitution of cocaine (10 per cent.) for morphine. These applications should be brushed over the whole interior of the throat after each meal, and also at bedtime. In using cocaine, care must be taken in its application to the larynx, which is sometimes provoked by it into dangerous spasm.

Among the internal remedies which I believe to be of use in this malady there are also two; the one is a syrup of the bromide of quinine and iron, with small doses of arsenic, and the other is a pill of reduced iron, valerianate of zinc, belladonna, and *nux vomica*.

This latter combination, often successful in controlling the cough, must be given in doses, slowly increased until the physiological effects of the belladonna become apparent, and then, slightly diminished; the amount administered should be maintained at that level.

There is still another remedy to which, in laryngeal nervous affections, specialists on throat disease attach great importance, and that is a long sea voyage. Undoubtedly it is sometimes successful; but undoubtedly also in my experience it is oftener the reverse. Furthermore, it is costly, it is difficult to carry out, it interrupts the work of education, and occasionally in boys it begets habits of a kind which permanently interfere with a right conduct of the business of life. For my own part, when I now review the experience of the effects of sea voyages upon young invalids, I am more strongly impressed than ever I was before with the necessity of extreme caution in prescribing them.

But although in all these ways the benefits which we shall be able to confer upon our patients may not appear to be great, there is one way in which, perhaps, we may be enabled to make them great beyond expectation or expression. We have seen that almost all the subjects of this malady are "nervous," and from this we know that they tend to become self-conscious, to dwell upon their disorders, to give way to self-distrust, to take interest only in themselves, and at last to fail in all that is useful as well as in all that is high in life. Coming at this early time of youth under our instruction and influence, we may help them to strive after self-effacement, to aim at some higher ideal than themselves, to find in labour strength, and through suffering tranquillity; and so, transfiguring their lives, to discover therein sacrifice instead of selfishness, and gladness instead of gloom.

SUCCESSFUL VACCINATION.—Dr. Samuel Hamill, B.A., Public Vaccinator of Burnham District Docking Union, Norfolk, has been awarded the grant for successful vaccination under the provisions of Section 5 of the Vaccination Act, 1867.

GUY'S HOSPITAL LONDON UNIVERSITY CLUB.—Dr. Pavy presided at the annual general meeting of this club. The officers for the coming year were elected: Mr. Howse and Dr. F. Taylor being chosen vice-presidents, and Dr. Horrocks, hon. secretary. Subsequently, the annual dinner took place, at which Dr. S. Wilks, F.R.S., was in the chair. In spite of foggy weather there was a good attendance, and a most enjoyable evening was spent. The club consists of nearly one hundred members—Guy's men and graduates or undergraduates of the University of London.

EXTRACTS FROM THREE LECTURES ON TUBERCULAR DISEASES OF BONES AND JOINTS.

Delivered at the Royal College of Surgeons of England.

By W. WATSON CHEYNE, M.B., F.R.C.S.,

Hunterian Professor; Surgeon to King's College Hospital; and to the Paddington Green Children's Hospital, etc.

LECTURE III.—CARIES OF BONE.

FOLLOWING the destruction of the cartilage, and in some cases beginning to a slight extent before the cartilage had disappeared, we have the carious changes in bone. Roughly speaking, we may say that clinically the articular ends of bones are carious when the cartilages are destroyed, but examination of a number of specimens where the articular cartilages have disappeared shows that the resulting state of matters varies much, and if we were to limit the term caries to tubercular disease of the surface of the bone itself, we should exclude many cases which are clinically reckoned as caries. It will be best, therefore, to study in one group the various conditions which we find when the articular cartilage has disappeared.

In some cases we find that the cartilages are absent, and that their place has been taken by fibrous tissue, but it is seldom that the whole surface is covered simply with fibrous tissue; usually there are tubercles or tubercular tissue at some part, either at the free surface, or in patches in the tissue. This is, however, usually an early stage, found only shortly after the disappearance of the cartilage, for it is seldom that the process ceases at this stage, or that the new tissue does not become the seat of development of tubercles, if, indeed, it has not been tubercular from the first.

[A number of specimens were shown to illustrate the various appearances met with where the cartilages have been more or less completely destroyed, but where the tubercular material has not yet penetrated into the superficial cancelli of the bone.]

The following are the most common appearances met with in true tubercular caries:

By far the most common type of caries, in my experience, is that seen in the section of the head of the tibia which I previously exhibited, as showing a soft caseating deposit in the substance of

Fig. 6.

the bone. (Fig. 6.) On the upper part of the surface of the bone we see that the superficial cancelli are infiltrated to a depth of about one-eighth of an inch with dense tissue, which, on examination under a higher power, is seen to be composed of tubercles and tissue infiltrated with epithelioid and giant cells, this tissue undergoing caseation at the edge. Lying among this tubercular tissue we see the trabeculae of the bone, which are con-

siderably thicker than elsewhere, and have in parts become completely detached, and lie loose in the cheesy material. At some little distance from the tubercular layer the bone is in a condition of rarefying osteitis, but immediately beneath it the cancelli contain young fibrous tissue, and it is here that the thickening of the trabeculae is occurring. The process here is condensation of the superficial trabeculae, invasion with tubercular tissue, lacunar absorption and death of the thickened trabeculae and rarefying osteitis beyond. It is curious that in some cases this rarefying osteitis occurs at a considerable distance from the surface, and often in patches, while between these patches and the carious surface there may be an interval of comparatively healthy tissue. This specimen also bears out very well what I said about the destruction of cartilage in the neighbourhood of tubercular deposits. It will be remembered I pointed out that although we might have expected that in the neighbourhood of tubercular deposits in the bone tubercles would readily form in the superficial cancelli of the bone and destroy the cartilage from beneath, this was not as a rule the case. And here we see that the only piece of cartilage left and the only part of the surface of the bone which has not yet undergone the carious change is in the immediate neighbourhood of the primary osseous deposit.

[Specimens were shown illustrating this and other types of tubercular caries. Among these may be mentioned a type in which there is no marked condensation of the trabeculae in the carious part, and where the tubercular tissue is more cellular, presenting more the form of tubercular infiltration, and rapidly undergoing caseation; one in which the condensation of the bone beneath the tubercular layer is very marked—a condition which, however, is rare in joints, and chiefly met with in the form termed caries sicca; and one in which the layer of newly formed bone beneath the carious surface presents a marked reticular arrangement, exactly like the network seen in some parts of a strumous dactylitis.]

The following is, in a few words, what happens in the course of tubercular disease of the surface of bone. The first changes occur in connection with the cartilage, though in some cases, but more rarely, the superficial cancelli of the bone first show signs of osteitis, and still more rarely, the presence of tubercles. The cartilage disappears in one of the modes formerly described, by far most commonly by the spread of tissue over the surface, and then we find the surface of the bone covered with young fibrous tissue containing tubercles, or with very vascular tissue containing tubercles, or with plain fibrous tissue. The tubercular growth soon extends into the superficial cancelli and young fibrous tissue is formed beyond it, and sclerosis of the trabeculae occurs. As the tubercular invasion extends, portions of these osseous trabeculae become detached, and caseation takes place. Beneath the tubercular invasion there is usually a line of condensing osteitis, of varying breadth, while further away there may or may not be rarefying osteitis, often in patches. The amount of condensation underneath the carious part varies much in extent and character, the bone being in rare cases very dense, and in others not noticeably sclerosed; the sclerosis of the trabeculae in the carious part varies also in amount, and may in rare cases be entirely absent. By the continued extension of these processes, the gradual destruction of the surface of the bone goes on, being naturally most marked at points subject to pressure. The presence of this thickening of the trabeculae previous to the tubercular invasion shows that active formative processes have been going on in the part, and that the death of the bone is not primarily a degenerative process, as various authors, such as Cornil and Ranvier, have asserted, but usually follows a previous stage of hyperplasia. The detachment of the fragments occurs by lacunar absorption of portions of the trabeculae; and the reason that these fragments are frequently not absorbed is because, in these cases, caseation occurs too rapidly. Where caseation occurs more slowly, more or less complete absorption may take place, and we may find on the surface a layer of completely caseous material without any fragments of bone.

Of great clinical importance are the following facts, namely, the slight depth to which the actual tubercular disease extends—seldom more than a quarter of an inch—the tendency to sclerosis immediately beneath it, and the occurrence of rarefying osteitis at some distance away, and often in patches without any tubercular growth. It is very curious to note in many cases that a layer of comparatively normal cancelli separates the carious part from that where the rarefying osteitis is most marked, but of this fact I am unable to offer any rational explanation at present.

[Owing to want of time the consideration of the next three forms of tubercular bone disease was omitted. Caries sicca is a term which was brought into prominence by Volkmann, and was applied to a chronic form of tubercular disease of the joints, which goes on, as a rule, without suppuration, in which there is much sclerosis of the bone and obliteration of the joint cavity, and also a concentric atrophy of the bone. In diffuse condensation of bone in connection with tubercular disease the shaft of the bone appears very dense and yellow, as if containing caseous material, but this appearance is due to fatty degeneration of the contents of the cancelli, and not to infiltration with tubercular material. The other diffuse change of bone in connection with tubercular disease is the opposite condition of diffuse softening of the bone, the medullary cavity becoming enlarged and filled with "red marrow." This is a very bad type of disease, as a number of bones are usually involved, and general tuberculosis is very apt to occur.]

8. *Tubercular Periostitis and Tubercular Osteomyelitis.*—Before leaving the subject of the tubercular changes in bone I must shortly describe the course of events when the disease attacks bones at a distance from the articular surfaces, and I shall especially refer to tubercular disease of the short long bones. In these cases the disease may either begin on the surface of the bone in connection with the periosteum or in the medulla of the bone, that is to say, we may either have a tubercular periostitis or a tubercular osteomyelitis.

Tubercular periostitis as a primary affection occurs most frequently in the ribs and the vertebrae. In the case of the ribs tubercular disease may commence either in the interior of the bone or in the periosteum; in the latter most commonly. Where tubercular periostitis affects a rib the bone at the seat of the disease soon becomes thinner than usual and presents a worm-eaten appearance. This superficial erosion of the bone steadily progresses till a more or less marked defect in the bone is formed; indeed, such a bone not uncommonly fractures. This appearance is due to the fact that the tubercular new growth, occurring first in the deeper layers of the periosteum, soon spreads into the bone along the Haversian canals, fills up the superficial cancellous spaces, and causes erosion of the bone on the surface, and also destruction beneath the surface, while sclerosis of the bone occurs in the neighbourhood. The disease spreads along the periosteum, and may involve a considerable extent of the bone, eating into it irregularly at various points. In the case of tubercular periostitis the disease spreads along the affected bone for a considerable distance, and it is almost always associated with chronic abscess, the tubercular disease spreading into the soft tissues as well as into the bone.

Perhaps the most common seat of tubercular periostitis is the vertebrae. Tubercular disease of the vertebrae commences either in the substance of the bone as a circumscribed tubercular deposit, or a more diffuse tubercular osteomyelitis, or on the surface as a tubercular periostitis. The latter is the most common form in adults; it involves a number of bones; curvature is at first absent, and is seldom acute, and chronic abscess very frequently occurs. [Specimens of disease of ribs and vertebra were shown.]

In the case of the short long bones, such as the phalanges, with their metacarpal and metatarsal bones, the tubercular disease generally commences in the interior of the bone in the form of tubercular osteomyelitis, and it may either cause a general enlargement of the bone, with or without necrosis in the interior, or a more marked and more limited expansion of the bone, the condition typical of strumous dactylitis. The former is more often the condition in adults, the latter in children.

The disease in both cases commences in the medulla of the bones, and is very correctly described as a tubercular osteomyelitis. In some cases it is accompanied by sclerosis and new formation of bone, leading to the production of a sequestrum, but the sequestra in these cases are seldom so entirely dense as those in the ends of the long bones; usually they have undergone a good deal of absorption, being thus in parts dense and in parts soft. More often, however, tubercular osteomyelitis results in the formation of an extensive soft mass in the interior of the bone, which, however, is not so well circumscribed as the soft deposits in the ends of the long bones, which I have previously described; and we frequently find in these cases that the tubercular growth spreads for a considerable distance along the medulla of the bone, leading to great sclerosis, and sometimes necrosis of the shaft outside. This infiltrating tubercular growth occurs perhaps most often in the bodies of the vertebrae and in

the smaller spongy bones, especially those of the tarsus, but it does not occur so often as the circumscribed soft deposits which I have previously described, and it is very rare in the epiphyses of the long bones.

Fig. 14.

This specimen (Fig. 14) is an excellent example of tubercular osteomyelitis with sequestrum formation. It is a complete section through the first phalanx of a finger of a patient, aged about 28, who was also suffering from phthisis. There was a sinus on the inner side of the finger leading to the front of the bone. On examining the specimen we see marked enlargement, more especially of the middle of the bone, in the centre of which is a cavity containing cheesy material and a sequestrum. This sequestrum is in parts much thickened, and in other parts partially absorbed, the spaces being filled with caseating material. Surrounding the caseating wall of the cavity is a layer of tissue containing tubercles either isolated or in the form of tubercular infiltration. Beyond this we have a layer of much thickened trabeculae, which are becoming eroded on the side towards the cavity by the tubercular growth. Beyond this layer of thickened and newly-formed trabeculae we have at the proximal end fairly normal tissue. Towards the distal end of the phalanx there are one or two tubercles in the cancelli, and in some of the sections the tubercular growth is beginning to creep over the cartilage at the anterior part. This cavity in the bone communicates freely with the surface anteriorly, the bone being entirely absorbed there, and the tubercular tissue is infiltrating the soft parts. The posterior part of the bone is also destroyed at one or two places, leading to the formation of holes communicating with the soft tissues outside.

[A transverse section of the radius was also shown, in the centre of which there was a soft tubercular deposit which had reached the surface at one part; in other parts there was marked new formation of bone from the periosteum.]

The most common form of tubercular osteomyelitis in young children is seen in strumous dactylitis. We do not usually have

the opportunity of examining these cases thoroughly, because amputation is but rarely necessary, and certainly not in the early stage, where the appearances would be most typical. I have, however, obtained a specimen of the lower end of the ulna which presented this form of the disease, as is not infrequently the case in children.

[Sections from this case were shown. There was a cavity in the bone which was filled with caseous material, and lined with soft tissue of a fibrous character containing a few tubercles. Outside this soft tissue the osseous trabeculae were thickened and more numerous than normal, and in places arranged in a peculiar reticulated manner, the cancelli containing fibrous tissue. Corresponding to the most dilated part of the bone there was new formation of bone from the periosteum.]

The process is essentially the same as that seen in some of the other cases of tubercular disease of bones previously described, namely, the deposit of tubercles in the medulla of the bone, a formative inflammation around, resulting in the production of fibrous tissue, thickening of existing trabeculae and formation of new trabeculae, subsequent invasion of this newly-formed bone by the tubercular growth and consequent absorption of the trabeculae. This process is accompanied by great enlargement of the bone, which again becomes thinned from the interior, and also by new formation of bone from the periosteum. By-and-bye the shell of the bone disappears at one or more points, and a communication forms externally.

A special investigation has been made by Renken as to the tubercular nature of the soft material in the interior of the bone in these cases of strumous dactylitis. In five cases he examined the soft material microscopically for tubercle bacilli, and at the same time inoculated guinea-pigs from each case. In every instance he found bacilli, though usually in small numbers, and all the guinea-pigs became tubercular.

PATHOLOGY OF CHRONIC ABSCESS.

I must in conclusion add a few words as to the pathology of chronic abscess. I have used the terms "chronic abscess" and "suppuration" in these lectures in connection with tubercular disease, although as a matter of fact these terms are incorrect

Fig. 15.

for when we speak of suppuration or chronic abscess in connection with tubercular processes, we do not mean the same pathological process as in ordinary suppuration and acute abscess. Nevertheless, the terms are so incorporated with medical literature that it is hardly possible to give them up, more especially as it is by no means easy to find a suitable short word as a substitute. There need, however, be no confusion if we bear in mind their meaning, and more especially if, in speaking of true suppuration, we add the adjectives "acute" or "septic."

The process of chronic suppuration will be best understood if

we trace the development of a chronic abscess in the soft parts. The earliest commencement of these abscesses is the formation of a small nodule, which steadily though slowly increases in size. After a time—when it has attained the size of, say, a nut or a pigeon's egg, sometimes earlier—the centre softens, the swelling increases more rapidly, and the contents become fluid—in fact, a chronic abscess has been formed.

If we examine such a nodule in the early stage, we find that it consists of a mass of tubercles, which at the oldest part have become confluent. At this point, which is shown in the accompanying photograph (Fig. 15) caseation begins, and a collection of caseous material is formed surrounded by tubercular tissue. This caseous material becomes infiltrated with fluid and also with some leucocytes, and thus we have a cavity containing fluid, fatty material, fragments of cells and leucocytes, and around this cavity tissue showing tubercular infiltration, and further away isolated tubercles. At the spreading margin the tubercular tissue continues to invade the surrounding structures, while not only does the caseation extend around the original cavity, but it also commences in independent parts of the wall. These fresh caseous centres ultimately communicate with the original cavity, and thus we have produced the ragged appearance of the wall so well shown in this photograph (Fig. 16). This is a complete section

Fig. 16.

through the wall of a chronic abscess, and we see the ragged appearance of the part next the abscess cavity, while the darker portions in the wall, which are numerous, are points where caseation is occurring. As the result of the formation of these isolated caseous deposits in the wall, portions which have not yet completely caseated become detached and fall into the cavity, forming the flakes and masses so constantly present in chronic abscesses.

These abscesses extend by fresh growth of the tubercular tissue in the surrounding parts, while caseation goes on at the centre. Hence, whatever part of the wall of a chronic abscess is examined, caseating tubercular tissue and frequently isolated tubercles will be found as is seen in this photograph (Fig. 17), which is taken from the wall of the abscess just shown under a low power. Here we see these tubercles with large giant cells, and the tissue in the neighbourhood is infiltrated with epithelioid and giant cells. When these abscesses burst externally, or are opened, a sinus is left, in the wall of which tubercles and tubercular tissue are present (as shown in a photograph exhibited).

Exactly the same process occurs in the case of chronic abscess in connection with tubercular disease of bones and joints. When suppuration occurs in a joint in connection with tubercular disease the layer of tubercles on the inner surface of the synovial membrane caseates, as I have previously shown in a number of specimens, the caseous material falls into the cavity of the joint, fluid is poured out, and leucocytes also pass out in varying numbers. Where the abscess forms in the substance of the synovial membrane a tract of tubercular tissue caseates and the abscess spreads in the manner formerly described.

Where a tubercular osseous deposit makes its way to the surface it infects the periosteum over it, destroys it, and then the tubercular growth spreads in the cellular tissue, giving rise to the formation of a chronic abscess. In such a case part of the wall of the abscess is formed by the carious surface of the bone. [A section through the upper cervical vertebrae from a child aged $1\frac{1}{2}$ year, where there was a large retropharyngeal abscess, was shown to illustrate these points.]

König ascribes an important rôle to fibrin in the formation of the walls of chronic abscesses and in the thickening of the synovial membrane. He says that fibrin is poured out and coagulates on the free surface of the cavity, that granulation cells spread into this layer, and tubercles appear in the granulation tissue, fresh layers of fibrin are deposited, and so the process goes on. This pathology will be seen to be quite different from the foregoing, but in my opinion it is erroneous. A careful study of complete sections of diseased synovial membrane and of the walls of chronic abscesses will, I think, show that the description which I have given is correct. There can be no mistaking the fact that the

Fig. 17.

granular material on the inner surface of the walls of chronic abscesses and of tubercular synovial membrane is, in the great majority of cases, derived from the degeneration of preformed tissue, and is not due to fresh deposit of fibrin on the surface, for the whole series of changes can be demonstrated, and in the first lecture I showed specimens in which the remains of degenerated tubercles are seen in the layer of this material on the surface of the synovial membrane, and one in which the actual involvement of a giant cell was evident. And further, in specimens which I have just shown, the earliest commencement of the process in the substance of the tissue and the production of material similar to that found on the surface are well seen.

While, as we have seen, caseation of the tubercular growth plays a very important part in the formation of chronic abscess, we must not therefore suppose that chronic suppuration is the same thing as caseation, or that these are convertible terms. In chronic abscess we have, in addition to caseation of the affected tissue, effusion of fluid and formation of pus cells, sometimes in very large numbers—in fact, there is more or less acute inflammation added to the tubercular process. Caseation is a very constant occurrence in tubercular growths, while chronic suppuration is by no means always present. Hence it is evident that some additional factor must come into play in determining the occurrence of chronic abscess. Some investigators have suggested that they owe their first formation to the ordinary pyogenic cocci, which settle in the affected tissue and cause suppuration, and that afterwards these organisms die out. Apart from the fact that the character of the contents of these abscesses differs from that of the contents of acute abscesses, the early symptoms of chronic abscess do not present the acute character which we

should expect if the pyogenic organisms were at work, while chronic abscesses have been examined at a very early stage, and I have done this repeatedly without any other organisms than tubercle bacilli being found. Further, as Garré has pointed out, pyogenic organisms grow luxuriantly in the pus of chronic abscesses, while they do not do so in pus which has previously been the seat of their growth. And, again, these cocci do not die so rapidly as we must assume to be the case in order to account for their absence from the contents of these abscesses; they can retain their vitality for months in the same material. I do not think that we can at the present time give any thoroughly satisfactory explanation of the occurrence or absence of chronic abscesses in connection with tubercular diseases of bones and joints. I believe that it has to do with the constitution of the patient, whatever that term may imply, and the local state of the part where caseation is occurring—that is to say, that the factor or factors at work do not come directly from without. These chronic abscesses are most common in patients who have a hereditary tubercular history, who suffer from multiple tubercular affections, who have fallen into a low state of health, where there has been exacerbation of the local trouble as the result of injury, and so on; the more definite meaning of these facts must be left for future research, but none the less we must bear in mind the tubercular nature of chronic abscesses, and the fact that the whole wall is infected and infective, and therefore in treating them we must pay attention to this wall, and try in some way or other to render it innocuous. For my own part, I believe that the best way of treating these abscesses is to dissect them out as if they were cysts whenever they are accessible and not too large, and if they cannot be dissected out, to open them very freely and peel out the wall as completely as possible, destroying what remains with some strong antiseptic, such as undiluted carbolic acid.

[Some remarks were made, in conclusion, as to points which could not be dealt with for want of time, as to the bearing of the facts shown on treatment, and as to the use of the lantern in illustrating lectures on pathology. The tendency of the facts exhibited was to show that in many cases these diseases are localised to one part of the joint in the first instance, and do not always commence diffusely over the whole structures of the joint, and the bearing of this point, and of the facts as to the exact extent of the tubercular disease in the affected part, is very important as regards treatment, seeing that we can now, by the aid of aseptic treatment, open joints at an early stage of the disease, and, while they deviate but little from the healthy condition, and by removing the tubercular focus in suitable cases, arrest the disease and rescue the functions of the joint.]

AN ADDRESS ON CERTAIN POINTS IN THE PROGNOSIS AND TREATMENT OF DISEASES OF THE HEART.

Delivered before the Hunterian Society.

By P. H. PYE-SMITH, M.D., F.R.S.,
Physician to Guy's Hospital.

(Concluded from page 1347.)

III. VALVULAR LESIONS.

Prognosis of Ulcerative Endocarditis.—It is unnecessary to insist upon the great danger of this condition. It is, in fact, not a mere mechanical obstruction to the circulation, but a form of internal pyæmia, in which micrococci and the chemical poisons which accompany them are continually being deposited in the most favourable possible situation for distribution over every part of the body by the blood stream. But just as the surgeon meets with occasional cases of chronic and articular pyæmia, like those described by Sir James Paget, so we meet with exceptional cases in which some difference in the quality of the infection or some increased power of resistance in the tissues robs this disease of what Dr. Osler calls its malignancy. One such case came under my observation in May, 1883. A young man was admitted into hospital under my care with valvular disease from previous rheu-

matism. To this was added recent acute ulcerative endocarditis, shown not only by the high and variable temperature and by the changing character and locality of the murmurs, but also by the occurrence of embolism, first in one radial artery, and then in the dorsalis pedis. In the former case we had a striking illustration of the effect of a septic embolus in causing acute softening of an arterial wall and expansion under diminished resistance to the blood pressure, for an acute aneurysm formed in the space of a few days in the obstructed artery. This patient lost his fever, gained his appetite, and after a time insisted upon leaving his bed. Finally, he left the hospital with the physical signs of the original valvular disease of course remaining, but with no symptoms of an active ulcerative process. His temperature was normal, he had gained flesh, and was eating and sleeping well.

Chronic Valvular Sclerosis.—Passing now to some points in the prognosis and treatment of what are the more frequent and most important of cardiac diseases—those of the valvular apparatus—the first question is what is the relative prognosis of the several anatomical lesions, circumstances being equal.

Lesions of Tricuspid Valves.—Tricuspid stenosis is almost always a complication of a similar contraction of the mitral valve, and I do not know whether it increases or diminishes the effects of the latter.

Tricuspid regurgitation is always secondary to valvular disease of the left side or to chronic obstruction in the pulmonary capillaries. Its existence is a sign of serious disease, but does not add to the gravity of the case, and may be regarded rather as a conservative effect in distributing increased venous pressure and allowing relief by anasarca.

Lesion of Pulmonary Valves.—Pulmonary, or, as I should prefer to call it, “dextro-sigmoid,” regurgitation is extremely rare, except as the result of ulcerative endocarditis of both sides of the heart, and it can scarcely add to the very grave prognosis of that disease.

Pulmonary, or dextro-sigmoid, stenosis is well known as a congenital lesion—either a true malformation or the result of intrauterine endocarditis; and its presence may be recognised both by symptoms and physical signs during life. The prognosis depends upon the narrowness of the opening and the condition of the ductus arteriosus and foramen ovale. In extreme cases life cannot be maintained long after birth. In the more favourable ones the patient may grow up with more or less of cyanosis, dyspnoea on the least exertion, liability to cold hands and feet, and to attacks of bronchitis if he spends the winter in England. The longest case I have known of well-marked morbus cœruleus from this cause was one of a young man who lived to about the age of 25, but I have heard of longer ones.² Besides the immediate effects of imperfect aëration of the blood, there is no doubt that these patients are particularly liable to death from tubercular disease of the lungs.

Lesions of Aortic Valves.—Of the valvular diseases of the left side it is generally admitted that aortic regurgitation is the most dangerous, the shortest in duration, the least remediable, and the most liable to sudden death; and that aortic obstruction is, other things being equal, the least severe in its symptoms and dangerous in its results. There is, however, considerable difference between an aortic lesion, the result of rheumatic fever in a young and otherwise healthy subject, and the same mechanical alteration when it occurs as the result of chronic atheromatous arteritis with extensive degeneration of the arteries. In the latter case aortic stenosis, though causing slight symptoms, may lead to sudden and fatal syncope.

Lesions of Mitral Valves.—The prognosis of chronic mitral disease is recognised by all authorities as more favourable than incompetence and less favourable than obstruction of the aortic valve. According to Dr. Peacock, Dr. Wilks, and Dr. Bristowe, while aortic obstruction is the least serious of valvular lesions, stenosis of the mitral valve comes next, and regurgitation is far more serious, though less so when affecting the mitral than the aortic orifice. Other writers, however, have regarded mitral stenosis as a graver defect than incompetence.

Perhaps the explanation of the difference of opinion lies in the following considerations: Mitral stenosis is in adults more frequent than simple incompetence; but a large number of obstructive cases first come under notice when regurgitation has also taken place, and the physical signs of the primary lesion are then not always

² In the slightest form this malformation may allow life to be prolonged till over 50 years of age, as in a case recently under the writer's notice.