to mere acidity, to the positive ion H. Stronger solutions drive it into rigor. Dr. Fletcher has shown that by a previous application of oxygen these effects can be prevented. The mammalian heart lying after death in an atmosphere of oxygen does not become acid, or but slightly so; and after perfusion with oxygenized Ringer's solution, even some hours after, no trace of lactic acid appears. Dr. Fletcher has shown me how many occult flaws there may possibly be in the balance of colloidal mechanisms in the cardiac muscle; and more than this—that the heart may have abundant fatty deposit in it, and yet the muscle cell on the motor side be intrinsically intact, and prove no more vulnerable than one histologically normal; and this he illustrated by the figure of an engine clogged with dirt and grease, which notwithstanding may do better work than another engine bright and clean but with tight bearings, or an unseen crack in its cylinder. When beyond unequal metabolisms we pass into the region of toxic influences we recognize again how near the edge our gyroscope may be revolving.

Finally we know that the heart's balance depends on that of many other parts also; the gaseous exchanges are not in the myocardium only, not even in the lungs only; they are burning in every muscular area, in every viscus. Thus, even if the haemoglobin be constant in quality and quantity, if velocity begins to fall we are in a vicious circle; velocity—which means oxygen exchange—slows down in every organ, especially in those which, beside the lungs, have a double capillary machinery, such as the liver and the kidneys; in these areas we very soon find evidences of stasis. And retardation in the lymph system also is not to be forgotten. Such defaults as these may for whiles miss or neutralize each other, but when at some unhappy moment a summation of adverse alternating conditions comes about—capillary retardations in the viscera, tissue calls, lymph pressures, vagus and vasomotor waves, psychic thrills—in such cases as these, cases not without their prior forebodings, we shall not be at a loss to comprehend at last the suddenness of death. But the riddle, which I have done so little to read, is the frequent suddenness of death in one who scarcely having known illness expires under no extraordinary effort; or in the peace of his own bed or elbow chair passes silently away.

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At the annual meeting of the board of governors of the Wolverhampton and Staffordshire General Hospital, held on March 12th, Dr. John Grout was elected a Vice-President in recognition of the valuable and memorable services rendered by him in connexion with the King Edward VII Memorial.

THE late Lord Wandsworth, a member of the firm of Stern Brothers, bankers, has bequeathed £10,000 to be used for medical research "as my old friend and doctor Sir William Bennett shall decide is best"; £5,000 to the Bolingbroke Hospital, Wandsworth Common; £1,000 each to the London Hospital and to St. George's Hospital, and £500 to the Hospital for Cancer, Fulham Road. After the payment of certain sums the residue of the estate, which it is estimated will exceed £1,000,000, has been left for the foundation of an orphanage to bear Lord Wandsworth's name.

THE third International Medical Congress on Accidents will be held at Düsseldorf in August (6th to 10th). Among the subjects proposed for discussion are the present state of the law as to compensation for accidents in different countries and the question of insurance against industrial diseases; the importance of early treatment directed to the restoration of functional disturbance after accidents; vascular disease and accidents; arthritis deformans and accidents; the influence of accidents on the origin and increase of tumours; methods of examination in cases of injury after accident. Communications relative to the Congress should be addressed to Professor Liniger, Düsseldorf.

A Post-Graduate Lecture ON THE ESTIMATION OF HEART POWER:

AND THE TERMS

BLOOD PRESSURE; HYPERTENSION; HYPERPIESIS; HYPERTONUS.

BY

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GENTLEMEN,—A request made by some of you that I should give a clinic on the present position of "blood-pressure" was communicated to me, and I of course at once expressed my willingness to speak to you on the subject.

At the outset permit me to say that, in the consideration of many clinical problems, the accuracy of our first step often determines the accuracy of our further steps, and of the conclusion we ultimately arrive at. As illustrating this proposition I would impress upon you that it is necessary to realize that, when we as clinicians feel the radial pulse, the initial and primary object in doing so is to obtain information as to the vigour or strength of the heart. The second object is to gain some knowledge of the condition of the channels through which the blood is propelled. As directly bearing upon our subject I recall to you various terms used in relation to it.

BLOOD PRESSURE.

This term means the pressure exercised by the blood inside the vessel, and the driving power of the heart is judged of by the estimate we form of this pressure of the blood upon the arterial wall. In much recent writing the term unfortunately is used to indicate, or as a mere synonym for, haemomanometer readings. Whatever the blood pressure be inside the artery it represents in some measure the power with which the blood is driven out of the left ventricle. This is the plain and fundamental fact, and this is the reason why the examination of the pulse occupies such an honoured place in our clinical methods. Other words are, however, in use in the study of this same problem of heart power.

HYPERTENSION.

The term "hypertension" is at present in common use, and has, I think, been popularized by the French school. In past years high and low tension pulses were spoken of. The word "tension" applied in this way was, however, long regarded by critical clinicians as unsatisfactory. The question always was whether it merely meant the amount of pressure exercised by the blood on the arterial wall or the taut state of the wall caused by the pressure, combined with changes in the wall itself. So far as I am able to see, the use of the term "hypertension" in no way answers the old question, although in many quarters it is treated and used as a "blessed word" and placed above the zone of criticism. Other clinicians steadily abstain from using the word "tension," in any of its possible meanings, as applied to the pulse, and with this attitude I am in complete accord. Without clear thinking and the use of suitable words the confusion which surrounds this subject will continue.

HYPERPIESIS.

The term "hyperpiesis" has been introduced by our veteran clinician, Sir T. Clifford Allbutt. Etymologically it means excessive pressure, and throws no light on the problem of how much is blood pressure and how much is arterial wall. If it carries with it the idea of fullness, the question at once arises of the relation of fullness to pressure. In measuring arterial pressure with the haemomanometer, the question remains, Are we measuring the pressure wave only, or are we not also measuring vessel wall?

HYPERTONUS.

The only other word I need refer to is "hypertonus." I am commonly regarded as having definitely applied it to an arterial condition. It means a contraction of the muscular

coat of arteries over and above the normal or average tonus. For many years, before the haemomanometer came into use, I had watched the occurrence of this over-tightening up of the arteries and the relaxation of the over-tightening. It is a clinical phenomenon which is as certainly present and is as easy of recognition as any phenomenon appreciable by the senses.

ARTERIO-SCLEROSIS.

I must next ask your attention to the term "arterio-sclerosis." It was introduced almost as another word for "atheroma." The thickened radial arteries which are so commonly met with clinically are, however, only rarely atheromatous—the thickening is due to hypertrophy of the middle, that is the muscular, coat. This thickening may occur with hardly a trace of atheroma, and most of the arteries with a muscular coat may be so thickened. It is a very common condition.

THE HAEMOMANOMETER.

The modified Riva-Rocci haemomanometers all have an armlet, which is usually applied to the upper arm. The bag encircling the arm has air pumped into it until the pressure has stopped the pulse wave, as indicated by the disappearance of the radial pulse at the wrist. The pressure in the bag is shown by a U-shaped mercurial manometer, or by a spirit manometer as introduced by Dr. George Oliver, or by a neat little mercurial manometer recently introduced by that ingenious physician. It does not really signify what manometer is used, for they are, of course, all carefully standardized, and either of the two latter is easily carried about. The armlet is practically the same in all.

THE CLAIM.

The armlet when applied to the upper arm obliterates by pressure the lumen of the brachial artery. This artery when perfectly normal is so thin-walled that when empty it collapses by its own weight. In this condition of artery the pressure required to close the vessel corresponds with the blood pressure inside the vessel. This physiological fact was taken up by some distinguished physicians who maintained that, no matter what pathological changes might be present in the artery compressed, this instrument recorded blood pressure—that is, recorded the same pressure as would be recorded were a cannula inserted into the artery and attached to a manometer.

THE AUTHOR'S POSITION.

My contention has been and is that when the artery wall is thickened the thickening increases the resistance to compression, the resistance then becoming *blood pressure plus vessel wall*.

The difference between the thickness of the normal brachial artery, which, as I have said, collapses by its own weight when empty, and an artery thickened by sclerosis—that is, by muscular hypertrophy—is great. In addition, however, to the muscular thickening, the thickened muscular coat retains the power to contract and to relax. When it contracts the wall becomes still thicker, and the lumen becomes still smaller. Now I submit to your common judgement that, when you put the armlet over such a thickened vessel, you have not only to overcome the resistance of the blood pressure, but of the hypertonic vessel wall in addition. The phenomena have passed from the physiological to the pathological, and therefore to the clinical.

As it is true that the thick-walled vessel requires greater force to obliterate its lumen than the thin walled vessel requires, it follows that when the thick muscular coat contracts beyond its normal *tonus*, the vessel wall is further thickened, and the lumen is further diminished, so that still greater pressure is required to obliterate the lumen and to stop the pulse wave. The *hyper-tonically* contracted sclerosed artery is more resistant to pressure than the tonically contracted one.

THREE FACTORS IN HAEMOMANOMETER OBSERVATIONS.

It thus follows that in taking haemomanometer observations three factors of clinical importance have to be considered, namely, (1) blood pressure; (2) structural changes in the wall of the artery; (3) hypertonus of the muscular coat. Upon the correctness of your estimate of these separate factors will depend the correctness of your conclusions, and will determine the real value that this

instrument will be in your clinical work. If you take its readings as only giving the first factor, namely, blood pressure, you will make the most appalling and disastrous mistakes, and I have known such to be made.

RECENT EXPERIMENTAL WORK.

I am aware that this subject can appear so clear to you, and my reasoning be so convincing, that further evidence may be regarded by you as unnecessary. But the fact remains that my contentions have not yet met with universal acceptance, and to help the conversion of our doubting friends I may tell you of some recent experimental work on the subject.

In the first place I shall refer to Dr. J. C. Janeway, of New York, who, I think, wrote the first book on blood pressure after the haemomanometer came into common use. He was a pure blood pressurist, and held that there was no evidence that the condition of the arterial wall affected haemomanometer readings, that the readings always meant blood pressure and nothing else.

He has recently taken up the question experimentally, in conjunction with Dr. E. A. Park, using arteries taken from freshly killed cattle for the purpose. It is interesting to find from the record of these experiments¹ that jointly they are satisfied that *hypertonus in normal arteries* may represent a pressure of 51 to 68.5 mm. of Hg. This is a large rise from the previous zero, and it is acknowledged that the effect of hypertonus had been overlooked—not of course by me, either clinically or pathologically! They say, "We must acknowledge that Russell has called attention to a hitherto *insufficiently* recognized influence of arterial contraction on our clinical readings." They would have been more correct had they said *totally unrecognized*. In the sentence immediately preceding this halting acknowledgement the statement is made that "it seems to us reasonably proved that Russell's contention that hypertonic contraction of the arteries and not high blood pressure is the cause of the high readings obtained with clinical instruments has no basis in fact." I am entitled to claim that when my work is criticized it should be presented accurately, and the sentence just quoted misrepresents my contentions, and indeed has "no basis in fact." My critics have not acquainted themselves with my work as they ought to have done, seeing my book was in their hands. They overlook the clinical and pathological fact upon which I based my contentions—namely, that the structural change present in the majority of the thickened arteries, recognized as such during life, was hypertrophy of the tunica media. I suggested that the term "arterio-sclerosis" should be confined to this condition, as it was the commonest form of thickening in radial arteries, and had no resemblance whatever to atheroma. It is clear that my critics have entirely failed to realize the significance of that contribution to clinical and pathological knowledge. They are entitled, perhaps, to confuse atheroma with other arterial thickenings, but they are not entitled to represent me as doing so. Having failed to note the significance of my pathological findings, they also failed to grasp the contention that arteries with such a thick muscular wall presented much greater resistance to compression by the armlet of the haemomanometer than normal arteries present. The further point they have also, as a consequence, missed—namely, that the hypertrophied tunica media retained its power of passing from tonus to hypertonus, and that hypertonus in such an artery leads to further thickening of the wall and to diminution in the size of the lumen, and consequently to still greater resistance to compression by the armlet. My critics have acknowledged that the hypertonus of a normal artery may give an increase of pressure of from 50 to 68 mm., but they will also find that when hypertonus is present in an artery with a hypertrophied muscular wall the resistance is greatly added to. I may mention that arteries with such walls as I describe will not be found in cattle; they must be sought for elsewhere.

My critics, however, have experimented not only with normal but with atheromatous and calcareous vessels, and speak of these changes as arterio-sclerosis. The use of the term "arterio-sclerosis" for all arterial thickenings and degenerations is deplorably inaccurate, and the matter under discussion will not be satisfactorily settled until totally different pathological processes cease to be called by the same name. Clinicians must accept the fact that

the thickened arteries, with which they so frequently meet, are not usually atheromatous, but are thickened as I have indicated and act as I have indicated. The atheromatous vessels used for the experiments were suspended in a glass cylinder, and the compressing medium was water introduced into the cylinder. It is shown that the water picks out the unaffected portions of such arteries, and compresses the artery at these points, and on this observation it is contended that *arterio-sclerosis* (which in the vessels used clearly means *atheroma*) has no effect in making the vessel more resisting to pressure. The observation so far as atheromatous arteries are concerned is correct, and is in harmony with our pathological knowledge of the scattered and patchy character of the lesions in *atheroma*, but a grave mistake is made in not allowing for the difference between the experimental method adopted and the clinical method. The observers might have seen, that, while a water-jacket would necessarily pick out and compress the smallest normal areas in the course of an atheromatous vessel, the armlet assuredly bridges over such areas unless they are extensive. No doubt the pressure exercised by the compressed muscle of the arm is spoken of as "fluid pressure," but to be led into experimental or clinical error by the use of this term, in a matter of this kind, is not reasonable. Muscle is not fluid in the sense in which water is fluid! The same method has been used by other experimenters, but Drs. Janeway and Park are the first to find that normal arteries when hypertonically contracted may add over 60 mm. to the pressure required to obliterate them. From this observation the authors of it must see that the conclusion is unavoidable, that the muscular thickening, to which I hold the term "*arterio-sclerosis*" ought to be confined, must add to the resistance to compression, and that when hypertonus is also added there is a great additional resistance offered to the air-bag.

I may state, what I have stated elsewhere, that in this condition the thickening is uniform and not patchy. It is, perhaps, also necessary to state that the hypertrophied muscle of an artery wall may lose its tonus, as the hypertrophied wall of a left ventricle may, and when it does so it relaxes, and resistance is then, of course, lowered. Thickness and tone of wall and size of lumen must always be considered together, if accurate understanding is to be reached.

In the BRITISH MEDICAL JOURNAL in 1908 I published observations made on a thickened brachial artery, obtained shortly after death, tested by means of an air-bag, and found that it indicated a pressure of something over 100 mm. Hg, and I do not think that this is the limit of resistance of the arterial wall.²

In July, 1911, Dr. Leonard Findlay published an interesting series of observations on brachial pressure, as compared with digital pressure at different ages.² The digital pressure was measured by Gaertner's tonometer. In early life there is little difference between them—that is to say, the blood pressure is much the same in the finger as in the brachial artery. As life advances, however, there appears a marked difference between the two, until between 40 and 50 years of age there may be a difference of 88 mm. Hg. In cases of Bright's disease this difference between brachial and digital artery pressure may reach 100 mm. Hg. He thinks the explanation is to be found in the arterial hypertonic contraction. He also experimented on cats with adrenalin and found that the difference between the carotid and the paw pressure rose from 50 to 140 after adrenalin injection. His explanation is that "the constricted arteries cut off the pressure wave," that the pressure wave is different in rigid and in elastic tubes, and that when "arteries are more or less sclerosed, or in acute Bright's disease where the arteries are tonically contracted, the elasticity of the vessel is diminished and more nearly approaches the character of a rigid tube."

Experimental work is thus confirming my clinical and pathological contentions.

Illustrative Cases.

I next ask your attention to three cases which illustrate how valuable and interesting haemomanometer observations are when properly interpreted.

CASE I.—Cerebral Symptoms due to Hypertonic Contraction.

This patient was under my care in Ward 27 in September, 1910. The phenomena she then presented consisted of cerebral

symptoms whenever the arterial pressure rose, as it could do, from 200 to 260 or 290 mm. I showed her to the post-graduates, and her case is recorded in the *Lancet* of December 3rd, 1910. I had lost sight of her, so that her being here now has been quite fortuitous so far as we are concerned. She has kept fairly well, but is threatened with a return of her symptoms, and whenever these threaten there is an associated rising of her brachial pressure. At present her pressure keeps about 200 mm., but as soon as it begins to rise she develops cerebral symptoms which are promptly removed by the administration of a vaso-dilator. If left alone the pressure can rise from 200 to about 300 mm., and we are asked to believe that this is the result of a variation in this woman's heart power. As a matter of fact, what happens is that her brachial artery becomes markedly hypertonically contracted, and it is this which gives the high reading; when the artery is relaxed by erythrol the reading rapidly falls to 200 again. It is this recurrence of hypertonic contraction which is this patient's danger. It is not any variation that the hypertonus may lead to in stimulating the left ventricle to increased effort that is the essence of the clinical phenomena. It is not a heart instability, it is a vessel instability; and, if we miss that, we miss the essential factor in the whole series of phenomena which the case can present; we fail to understand the case, and what we fail to interpret aright we cannot treat aright.

CASE II.—Arterial Thickening due to Syphilis.

I next show you a man aged 40 who was admitted on account of severe and constant headache, and who had syphilis three years ago. His radial and brachial arteries were somewhat thickened, and his brachial pressure varied between 140 and 150 mm. The pulse wave was small and feeble; there was no heart enlargement discoverable by percussion; the heart impulse was feeble and the sounds were very faint, the first sound at the apex and over the right ventricle being barely recognizable. He was anaemic, with a red blood count of 2,560,000 and 46 per cent. of haemoglobin. He was put on large doses of potassium iodide. The headaches soon yielded to this treatment; at the same time the changes in his arteries and in the vigour of his circulation were of particular interest, for one does not often have the opportunity of following the changes as we had in this patient. When he had been taking the iodide for some time the arteries became quite perceptibly softer and larger, and along with this the pulse wave became larger and fuller, and the first sound of the heart came out clearly and definitely. These phenomena were as definite to my house-physicians as to me. With these marked changes in the circulatory phenomena there was, however, no change in the haemomanometer reading.

I have seen this kind of thing before, and the interesting clinical facts which are to be noted in such cases are:

(1) That the hypertonic thickening of the vessel has yielded; (2) that the power of the left ventricle has been restored, and the pulse has become larger and stronger; and (3) that there has been no change in the haemomanometer reading. No one who still claims to know anything about the pulse as measured by the finger would for a moment doubt that there had been a marked increase in heart power in this patient. Such an increase was confirmed by the change in the character of the first sound and of the impulse. Yet there was no change in haemomanometer reading; and there ought to have been, did this instrument record blood pressure and blood pressure only.

From my standpoint, the explanation is that what was gained in heart power was balanced by the relaxation of the hypertonus. That is to say, if the blood pressure rose 60 mm. it was balanced by a loss in the resistance of the vessel wall of 60 mm.

Again you see how entirely misleading this instrument will be if you take it as the measure of heart power in your clinical work, and yet how interesting and valuable it is when you learn to interpret its records aright. I shall not detain you by showing you other cases; but I should like to tell you about a medical friend whom I saw a few days ago, as he illustrates another aspect of the subject we have been considering.

CASE III.—Angina Pectoris Minor with Hypertonic Spasm.

A few months ago this friend consulted me about a recurring pain in the left chest over the precordia and posteriorly. It was difficult to be quite satisfied as to the meaning of this pain, for the heart was perfectly sound and the brachial pressure was only 125 mm.; the vessels were soft and the pulse regular and of good volume. He, moreover, was a strong cyclist, and his cycling did not make things worse, nor did it bring on the pain. With treatment he got relief for three months, but the pains had returned; they were more severe and very perturbing, as they came on at unexpected moments, and when they supervened he had to stop what he was doing. There was a suggestion of the pain going down the left arm. He was anxious that I should not think his symptoms merely fanciful. The pulse was 80 and the pressure 125 mm., the heart sounds were clear and closed. After the examination we sat talking for some

time, when he told me that an attack was coming on. His colour did not change, but he leant forward, resting his elbows on his knees, and was evidently considerably distressed. I again felt his pulse and found it distinctly smaller, and instead of beating 80 it was only beating 60. I asked him to take his coat off, and on taking his pressure found it had risen from 125 mm. to 145 mm.

This observation, to my mind, at once settled the question of diagnosis. This was a true angina pectoris minor, a condition I described years ago. My interpretation of the phenomena is that the state of the vessels, as revealed to the finger, and shown by the rise in the haemomanometer pressure from 125 to 145 mm., indicated that a hypertonic spasm had taken place; that this increased the pressure in the aorta, and thereby perturbed the heart, which, instead of being accelerated, was slowed down from 80 to 60 beats per minute. To speak of this as a neurosis almost always suggests that the symptoms are either fanciful or that the condition is mainly determined by a mental and central influence; and no men more warmly resent this view when applied to themselves than medical men do. Personally I interpret the phenomena as being determined by peripheral conditions and not by central ones. To me this is a vessel irritability, and its successful treatment lies in the removal of the cause keeping up that irritability. In this particular case the cause was probably tobacco.

Again I point out to you that the variation in haemomanometer reading was due to change in the vessel wall (the brachial artery); a mere increase of heart power represented by 20 mm. would not give rise to such symptoms. My patient could, I doubt not, stimulate his heart by cycling above that and without discomfort, but the moment a general angiospasm occurs the symptoms appear. It is always a question whether or no the coronary arteries participate in the spasm, but the practical fact is that if you counteract the general angiospasm the symptoms disappear. Your patient is cured when you have removed the tendency to this hypertonic spasm. The foundation fact is the vessel spasm, for the moment you relieve that the symptoms disappear; the spasm affects the brachial as well as the smaller arteries, and, I repeat, the haemomanometer gives you the record of it, when the vessels are normal as well as when they are sclerosed.

There are always the three factors to keep in mind—namely, blood pressure, structural change in the vessel, and hypertonus. There is no escape from the exercise of discretion and judgement when using this instrument, and those are the points; the significance and value of each have to be determined in every individual case. The instrument has opened no royal road to the determination of the all-important question of heart power, as we are called upon to judge of it clinically.

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UNDER the will of the late Mr. Alfred Bell of Newcastle, who died on November 25th last, the Royal Victoria Infirmary, Newcastle, and the Hull Royal Infirmary each receive a sum of £2,000, and Ingham Infirmary, South Shields, one of £1,000.

THE Yellow Fever Bureau *Bulletin* for January, 1912, contains a reprint of an interesting report on the yellow fever in Mérida by Dr. Mario G. Lebreto, Director of the Duque Laboratory of Investigations, Havana. After careful and prolonged study, Dr. Lebreto has come to the conclusion that there is an endemic focus of yellow fever in the Gulf of Mexico, that Mérida is to be considered as a seriously infected locality, and that because of the close contact, the lack of effective defences, and the short time since the last outbreak, Campeche is to be considered as suspicious. He believes also that persons coming from Quintana Roo should be watched by the Mexican sanitary authorities, who, by a careful study of the fevers affecting soldiers, might gain some insight into the histories of the past epidemics occurring at Mérida and Campeche. He recommends that Vera Cruz, and other ports in the Gulf where cases may arrive in the infective state, should establish a sanitary organization to observe and study all suspicious cases in order finally to determine the best methods for the complete and final eradication of the disease.

SERUM AND VACCINE THERAPY IN CONNEXION WITH DISEASES OF THE EYE.

THE MIDDLEMORE PRIZE ESSAY, BRITISH MEDICAL ASSOCIATION, 1911.

By C. W. G. BRYAN, F.R.C.S.ENG.,

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(Continued from page 592.)

II.

DISEASES OF THE EYE DUE TO PYOGENIC ORGANISMS.

In treating tuberculosis by vaccine, it is necessary to use a "stock" vaccine, whereas, in dealing with other infections, in most cases it is possible to prepare a specific vaccine from cultures of the infecting organism obtained from the patients to be treated. It is advisable to use a specific vaccine in most cases, but it seems to be unnecessary in cases of infection by staphylococcus. Here a mixed stock vaccine, prepared from cultures of *Staphylococcus aureus*, *citreus*, and *albus*, is usually efficient; if the use of such a vaccine is not attended by improvement in a reasonable time, a specific vaccine should be prepared. Again, in disease due to gonococcus, owing to the difficulty of preparing a vaccine, stock vaccine must usually be used, and gives quite satisfactory results.

Polyvalent stock vaccines have been used in infections due to pneumococci and streptococci, but the best results are obtained by the use of specific vaccines, as there are many different strains of these organisms.

In taking cultures from eye diseases for the purpose of preparing vaccines, it is advisable in most cases to use as culture media serum or blood agar, as many of the bacteria which commonly affect the eye grow well only on these media.

The vaccine is prepared by making an emulsion, by shaking, of the bacteria in 0.85 per cent. sodium chloride solution, and standardizing by mixing a portion of the emulsion with a portion of blood (which normally contains 5,000 million of red cells to the cubic centimetre), and counting fields of bacteria and red cells on a microscopic slide. In this way the number of bacteria per cubic centimetre is estimated. The emulsion is then sterilized at a temperature of 58° C. for thirty minutes, and finally diluted to a convenient strength in 0.5 per cent. phenol.

I will now deal with the vaccine treatment of the various regions which come within the province of eye surgery.

External Infections.

1. The lacrymal sac.

The lacrymal sac is liable to acute and chronic inflammations. The acute inflammations are usually due to streptococci, and, as stock vaccines are unreliable, there is not time, as a rule, for vaccine treatment. I have treated acute lacrymal abscesses with stock streptococcus vaccine, combined with ordinary surgical treatment, but have not been satisfied that the vaccine influenced the course of the disease.

The most important variety of chronic inflammation is that due to infection by pneumococcus. The chief dangers of this disease are—first, that abrasions of the cornea may become infected and severe ulcers be produced; and, secondly, that any operation carried out on an eye with such infection of the sac is attended with grave risk of sepsis. The region of the lacrymal sac is well supplied with blood, and infections react well to vaccine treatment.

Very Chronic Inflammation of the Lacrymal Sac Cured by a Specific Pneumococcus Vaccine.

W. T., aged 66. Right eye amblyopic. Attended first November, 1905, with left cataract and suppurating lacrymal sac. The sac was washed through regularly without relief, so in August, 1907, the sac was extirpated, leaving, however, a sinus. Ordinary treatment by scrapings and antiseptics did not affect the condition, cultures of pneumococcus being obtained in February and April, 1908, making operation for the left cataract impossible. Treatment by a vaccine prepared from the pneumococcus cultures was carried out from May 2nd to August 3rd. The opsonic index was regularly taken and an inoculation given every ten days, commencing with 10 million and gradually increasing to 50 million. After the latter dose, however, a negative phase was produced, followed, however, by