of specimens mounted in the beautiful formalin method devised by Mr. Priestley Smith, which is the best mode we yet know of.

Again, in many cases there is no adequate description of the specimens by which their special features can be recognized.

There can really be no lack of material considering the number of eyes which are removed every year, and it is deplorable that the waste of valuable specimens should continue.

Owing to a peculiarity in human nature, we are apt to be more interested in people's failures than in their successes. How stimulating and arresting our teaching would be, if, more than occasionally, it told of our failures and the reasons for them! If this were done more frequently it would also lead inevitably to a demonstration of how our mistakes might be overcome. For instance, what a wonderful collection might be made of both macroscopic and microscopic specimens of eyes lost after trephining or after cataract extractions. What an opportunity there might be—to give but a single instance—of working out the reason and possibly the cure for the unsatisfactory and often hopeless condition of glaucoma following dislocation of the lens.

We have few laboratories where these eye specimens can be prepared. We must have more, and make greater use of those already existing, and appoint ophthalmic pathologists who have special knowledge in the preparation, the cutting, and the investigation of eye specimens.

Pathology is, as we all know, one of the avenues of medical progress. A thorough grounding in pathology, both for teachers and students, would also act as an incentive to original work. The English medical profession is blamed, not without some reason, for the lack of original work it produces. If English ophthalmology is to fill the position we consider it should hold—and never has there been such an opportunity as the present—there is no doubt that our output of original work must be increased.

ABSTRACTS

I.—THE RETINAL CIRCULATION


In this communication Bailliart summarizes his previous researches, and at the same time carries them a stage further. He considers that the retinal circulation provides the ideal
conditions for the perfect study of the circulation of the blood within an individual organ, and he emphasizes the importance of realizing the large measure in which the circulation in one organ may vary independently of that in other parts of the system.

The retinal pulse.—If the fundus is examined under comparatively high magnification (as, for instance, with a Gulstrand ophthalmoscope), an arterial pulse can be demonstrated in the retinal arteries even under normal conditions. It is, however, better to reserve the term "retinal pulse" for the appearance of this phenomenon under abnormal conditions, as has hitherto been the custom.

When the intraocular pressure equals the diastolic pressure within the retinal artery, the pulsation witnessed will be at its maximum; this is independent of whether the rise in intraocular pressure is fortuitous (as by finger pressure), or pathological as in glaucoma. A spontaneous retinal arterial pulse is evidence that the intraocular pressure is equal to, or greater than, the local arterial diastolic pressure.

T. Henderson's pressure gauge, shown before the Ophthalmological Society in 1914, is mentioned, and credit is given to him as being the first to use this method of measuring retinal arterial pressure. Bailliart only learnt of this work recently.

Priestley Smith's articles in the British Journal of Ophthalmology are next discussed, and that writer's estimate that the systolic arterial tension in the retina is between 90 and 100 mm. Hg. is considered as too high. Bailliart mentions von Schultén's observations, quoted by Priestley Smith, and lays stress on the improbability that the retinal and carotid arterial pressures could be so nearly alike as the Finnish observer found them to be.

Next there follows a résumé of Bailliart's methods, which have already been so excellently described by Priestley Smith in the British Journal of Ophthalmology, Vol. II, pp. 487-9, that there is little left to add. Bailliart has modified his estimate of the diastolic and systolic retinal pressures, presumably in deference to Priestley Smith's criticisms as to the normal intraocular pressure; his views on this point have also been altered in the same direction by some fresh experiments he has made on the living cat. His figures are a little lower in the child and a little higher in the aged than they are in the average adult. Fasting materially lowers the retinal arterial tension, which is, on the other hand, quite unaffected by sitting as compared with lying.

Hypertension in the branches of the retinal artery.—Bailliart prefers to express this directly in figures read off on his dynamometer, rather than to reduce it to mm. of Hg., rightly holding that the former is both more accurate and more scientific as a method of stating his case. He would, therefore, say that, if the dynamometer
The Retinal Circulation

reading is above 30 grammes for the appearance of arterial pulsation, or above 80 grammes for its disappearance, we may set down the diastolic or systolic pressure as too high.

In subjects with marked hypertension, it is possible to recognize the condition by finger pressure alone, but in all doubtful cases, the dynamometer must be used.

Bailliart attaches little importance to the ophthalmoscopic evidence of increased arterial tension as described by Gunn, Bardsley, and others, but would lay much stress on certain subjective signs, which he regards as characteristic. These are: (1) attacks of complete and transitory blindness, which may affect the region supplied by the trunk of the artery, or one or more of its branches, and which speedily disappear under massage. (2) Muscae volitantes, which come and go, and which appear and disappear, independently of the presence of vitreous opacities, and even when the patient is not looking at a brightly-lit surface. (3) The appearance at the periphery of the visual field of sudden, brilliant sparks of light, which appear to circulate within the eye, and to go out as suddenly as they came. (4) A mistiness of vision which causes objects to be seen as through a haze, though no definite clinical phenomena can be found to account for the trouble; Bailliart has no doubt that these are cases of slight retinal oedema.

The causes of retinal arterial hypertension.—General arterial hypertension stands in the first rank of these. As a broad rule, it may be stated that when the general arterial blood pressure rises, the retinal follows suit, but we have also to take account of the factors which may increase the local vascular tension. Of these the most important is vasodilatation of the trunk of the retinal artery, which, while lowering the blood pressure above the point of dilatation, increases it in the capillaries and veins below. It is to be remembered that it is in the arterioles of the type of the central retinal artery, that one finds vasomotor contractility at its maximum. Then again, any obstacle to the return of blood in the region of the central vein, will drive up the local arterial pressure independently of the vagaries of the general blood pressure. In other words, the eye manifests the same kind of changes which we know are going on in all the other organs of the system, and, like any one of them, it may be influenced, from moment to moment, by many factors, one of which is the onset of mental emotion.

The complications of retinal arterial hypertension.—The most dangerous of these is haemorrhage. Although retinal arterial haemorrhage may be rare, we must bear in mind that the arterial hypertension brings about changes in the walls of the blood-vessels throughout the whole retinal system, as a consequence, in an early stage at least, of the capillary and venous hypertension which follows in the train of the rise in arterial pressure.
Aetiology.—A local rise in the retinal arterial blood pressure may be merely an evidence of an increase in the general arterial blood pressure.

The causes of the latter may fall with more powerful incidence on the retinal circulation alone; they include albuminuria, diabetes, and syphilis. Of more strictly localized conditions we must mention optic neuritis and thrombosis of the central vein.

Retinal arterial hypotension.—This is much less frequently met with than hypertension. Bailliart has twice seen it in the course of an examination by his dynamometer: the arterial pressure fell, a spontaneous pulse appeared, and then the patients fainted. It must be confessed that, from the surgeon’s point of view, this sequence of events might prove very unpleasant and even damaging. de Wecker had noticed the same phenomenon in the fainting, in the dying, and in the subjects of epilepsy, as well as in those in the algid stage of cholera.

The pressure in the trunk of the central retinal vein.—Bailliart believes that the venous pulse is often mistaken for the arterial. The latter is always abrupt, and almost instantaneous; moreover, it affects a considerable part of the arterial network on the disc; on the other hand, the former is slower and more measured, and is generally limited to a very narrow portion of the length of the vein. The vessel seems to give way for a moment, to chase its contents, first towards the centre and then towards the periphery, and finally slowly to resume its normal calibre.

A rarer form of pulse (seen in 2 per cent. of Bailliart’s cases) is brought about, not by the movement of the vein walls, but by that of the mass of blood within the vessels; it is a true piston-movement along the axis of the vein. The mass of blood is first pushed centrally, and then returns suddenly towards the periphery as if the vis a tergo, for one moment enfeebled, was yielding before a pressure momentarily superior to its own.

The mechanism of the venous pulse.—The current explanations of this phenomenon are stated, and Bailliart then proceeds to discuss the factors concerned in venous pressure: (1) There is first, the vis a tergo, originating in the drive from the heart, handed on through the capillaries, and gradually fading away until, in the jugular vein, it may be a negative quantity during the ventricular systole. (2) At the moment of the auricular systole, which just precedes that of the ventricle, the sudden arrest of the venous blood sharply increases the pressure within the jugular. (3) The respiratory movements of the thorax, too, have their influence on the venous pressure; this is positive during inspiration, and negative during expiration. The result is (1) that the pressure in the retinal veins rises to reach a maximum at the moment of auricular systole (which is synchronous
with the ventricular presystole), and (2) that it is lowest during the ventricular systole (which is synchronous with auricular diastole). But during the ventricular systole, the ocular tension is at its maximum, so that at the same moment the walls of the central vein on the disc are exposed to an increase of pressure on their external surface, and a decrease of pressure from within; it is not then surprising that they give way. Next, the ocular pressure falls during diastole, whilst the venous pressure is simultaneously rising to reach its maximum at the moment of presystole (auricular contraction). We can thus understand why the central vein at first fills gradually, and then suddenly, at the onset of presystole, undergoes for a moment a maximum dilatation. All this explains (1) the slowness of the venous pulse, (2) the contraction of the vein, which lasts throughout the systole, and (3) the slow and progressive dilatation of the vessel, during the whole of the remainder of the cardiac revolution.

If the venous pressure, even at its lowest, is higher than the ocular pressure, the pulse we have been describing cannot make its appearance; the piston pulse may, however, be seen, for, when the pressure in the central end of the vein is, during the short moment of presystole, very distinctly above the peripheral pressure, there will be a momentary arrest, or a reflux of the mass of blood within the vein; we then see the distinct and characteristic piston movement, which is most often in evidence when there is an obstruction to the return of venous blood. Lastly, if the venous pressure is constantly lower than the ocular pressure, the wall of the vein, always held down by a superior pressure on its outer side, can never dilate to its maximum; consequently, the vein will be immobile, or at the most, will show but feeble movements.

The information furnished by the examination of the retinal veins.—Bailliart states that it is on the disc alone that the inspection of the veins or arteries can furnish any information of value, for there the vessels are free from adhesions to the subjacent tissues, and so can contract and expand under variations of pressure, putting aside the piston pulse. Two cases present themselves:

1) A spontaneous venous pulse is present.—Here the venous pressure is fluctuating above and below the intraocular pressure. If we would know how far the maximum venous pressure is above the ocular pressure, all we have to do is to press on the globe until the pulse disappears, the dynamometer reading will furnish the information we require.

2) There is no venous pulse.—If, on increasing the intraocular pressure, one appears, we are dealing with an eye in which the venous pressure is steadily above the intraocular pressure. If, on the other hand, no pulse appears, we may gather that the venous exit pressure is steadily below the intraocular pressure, and that the
circulation is maintained through the flattened wall of the vessel, kept sufficiently open for the purpose by its own rigidity. If we lower the pressure of the globe by compression or massage, or if we raise the venous pressure, by making the subject hold his breath after a strong expiration, the venous pulse will appear.

**The retinal venous pressure.**—The previous estimates of this are first considered. Bailliart's observations on the presence or absence of the pulse in 66 normal subjects are recalled. Thirty-seven per cent. showed a spontaneous venous pulse; in 12 per cent. a pulse could be elicited by light pressure; in 30 per cent. no pulse could be observed or elicited by pressure; lastly, in 21 per cent., the venous pulse came and went from time to time.

Obviously, then, the venous pressure, at the exit of the vein, is always close to the intraocular pressure, round which it oscillates, influenced by the phases of the cardiac cycle, and by the systolic and diastolic variations of the intraocular pressure. Bailliart would place the intraocular pressure at around 20 mm. Hg., and the venous exit pressure at from 18 to 22 mm. Hg. He argues that, if Leonard Hill was right in placing the pressure in the torcular Herophili at 10 mm. Hg., it must surely be higher in the cavernous sinus, and still higher in the ophthalmic vein and its branches.

**Pathological variations in the venous pressure.**—Hypertension is found in the veins just as it is in the arteries, but it is a mistake to suppose that a spontaneous venous pulse is an indication of such hypertension, for it may be met with under conditions both of high and low venous pressure. A well-marked illustration of the latter is its presence, many times observed after embolism of the central artery, when the venous pressure is very low indeed. The view that by compression of the globe we can measure the venous pressure, its minimum being signalled by the appearance of a venous pulse, and its maximum by the disappearance of that pulse, is unsound, for the closure of the venous system quickly establishes a condition of uniform pressure throughout the retinal circulation from the arteries to the veins; this pressure is that of the arteries. We can, however, always prove the presence of venous hypertension and we can sometimes measure it: (1) When a venous pulse is present. Whenever it requires a dynamometer pressure of over 25 grammes to stop the venous pulse in these cases, venous hypertension is present and can be measured like arterial hypertension. (2) When there is no spontaneous venous pulse. We can find the pressure at which the pulse is made to appear and that at which it disappears, but as already shown the observation will be open to error.

Practically, we must limit ourselves to noting the pressure at which the venous pulse is extinguished, if spontaneously present, or
induced if not present. These are the only exact measurements we can make. It is sometimes very easy to recognize venous hypertension. When the maximal venous pressure is higher than the minimal arterial pressure, a venous pulse persists after the arterial pulse makes its appearance; this alternation of the two pulses, of which the venous marks the presystole and the arterial the systole, is characteristic of an obstruction to the return of venous blood; it connotes the existence of a vaso-dilatation of the capillaries and veins.

Venous hypertension is frequently met with: In thrombosis of the central vein, it constitutes from the first a diagnostic sign. It is also often met with in retinal haemorrhages. It is a mistake to suppose that it is always associated with arterial hypertension. In albuminuric retinitis, the arterial tension may be very high, and the venous tension normal. Speaking generally, retinal haemorrhages accompany venous hypertension. In glaucoma, retinal venous hypertension is met with, but not by any means constantly.

As for the causes of venous hypertension, these are (1) some obstruction more or less complete, to venous return, or (2) peripheral vaso-dilatation which throws open the capillaries and veins to the results of arterial pressure. Of venous hypotension we know but little. It is certain that, although the retinal circulation can accommodate itself, thanks to the resistance of the vessel walls, to a pressure slightly below the ocular pressure, this inferiority must not be too great, or the venous wall will close, and all circulation will stop.

The relationship between the ocular pressure and the retinal arterial pressure.—Under artificial pressure the visual field is obliterated from within outward, and complete blindness soon comes on. Just before the latter event, the subject can see a rhythmic pulse in his visual field. The appearance of this phenomenon marks the point at which the ocular pressure surpasses the diastolic arterial pressure. When the systolic level of pressure is reached, vision is extinguished. From what we thus see happen under the application of direct pressure, we can argue what will happen if the intraocular pressure rises due to pathological conditions. Nor must we forget that under the latter circumstances the vessel walls will suffer from the increased pressure to which they are submitted. There must be a relationship between the ocular and the retinal arterial pressures. To understand the problems involved we must first study this relationship under normal conditions, and then pass on to consider them under those which are pathological.

(A) The normal relationship of the ocular and retinal arterial pressures. Bailliat assumes the intraocular pressure to be one of 20 mm. Hg., but thinks the exactitude of the estimate to be of less
importance than the study of the difference between the ocular pressure and that within the retinal vessels.

Why should a comparatively slight rise in intraocular pressure unfavourably affect vision? Is it that the retinal cells cannot function satisfactorily, except within well-defined pressure limits? No! For we see them sometimes bear with impunity considerable falls of pressure. Is it not rather that under these conditions the circulatory supply of the retinal elements is interfered with?

Taking the systolic and diastolic arterial pressures as 70 and 30 mm. Hg. and the venous exit pressure at 20 mm. Hg., what is the probable capillary pressure? This must be lower than the diastolic pressure, otherwise the retinal circulation could only be carried on during part of the cardiac cycle: it must, therefore, be lower than 30 and higher than 20. As an approximation, Bailliart puts it at 25. He discusses Priestley Smith's estimate of 39, which he thinks too high.

Supposing that the intraocular pressure goes above 30, what will happen? The diastolic pulse will appear in the large arteries and blood will only circulate during the systolic period; the thin-walled capillaries, with their low vascular pressure and their feeble resistance to the pressure from outside, will ill maintain the circulation; the more peripheral they are, the more effectually will an increase of intraocular pressure cut them off from nourishing the tissues they supply. The lessons are obvious: Whenever the ocular pressure exceeds the capillary pressure, obstruction to the capillary circulation, and loss of vision follow. The further away from the papilla any area is situate, or the longer the pressure is maintained, the greater will be the damage done.

But we know that pressures well above 30 mm. Hg. may be met with without any visual trouble appearing. Bailliart explains this by stating that when the ocular pressure rises, the arterial pressure tends to rise with it, and so, up to a certain point, physiological equilibrium may be maintained by the preservation of the ratio between these rising pressures. He believes, for instance, that in different positions of the body, a constant relationship is preserved between the ocular pressure and the retinal arterial pressure, and that these fall and rise together according to the variations in the position assumed.

(b) It is a different matter when physiological limits are overstepped: the equilibrium is then broken. He discusses in turn the conditions arising therefrom.

1. Trouble due to an increase of the intraocular pressure.— If the ocular pressure rises above 30 mm. Hg., the minimal arterial pressure tends to keep a little above it, but so little that the least pressure on the globe causes arterial pulsation. The systolic pressure does not maintain its relative superiority to the diastolic,
and the two therefore come nearer together. Moreover, the arterial pressure is so little above the intraocular pressure as to make it certain that the latter surpasses the capillary pressure. [Reviewer's note.—Such a condition can obviously be only a temporary one, otherwise either all circulation would stop, or the capillary pressure would have to rise to carry it on.]

Even in the trunk of the central artery, the diastolic pressure may be lower than the ocular pressure; a pulse then appears. Such a spontaneous pulse is rarely seen in glaucoma; but, when seen, it is evidence that the blood is only circulating during a part of the cardiac cycle. Herein lies the key to the signs and symptoms of glaucoma. Bailliart repeats the fallacy that the early limitation of the nasal field is to be explained by the longer course of the temporal vessels. The reviewer has shown the futility of this argument on pages 246 to 248 of his book on "Glaucoma."

Bailliart asks: Is it the raised ocular pressure which defensively brings about a rise in arterial tension, or is it the other way about? He also raises the question of the existence of a vasomotor centre, either peripheral or central, which acts only through the vascular system. We need not follow him at any length in these speculations.

(2) Trouble due to arterial hypotension.—If, with the ocular pressure normal, or slightly below normal, the arterial retinal pressure falls sharply, we have the same rupture of equilibrium as in glaucoma. The sharp arterial hypotensions, whether general (as in syncope, cholera, and anaemia), or local (as in quinine amaurosis), bring about the same trouble as does ocular hypertension. In such cases, Graefe, by iridectomy, and Rothmund, by paracentesis, re-established the circulation and brought back normal vision.

Another very interesting group of cases is that in which we find the evidence of glaucoma without the signs of increased tension. Bailliart is sceptical as to the existence of transitory increases of pressure in these cases, and prefers to believe that there is a disturbance of the equilibrium between the ocular pressure and the arterial pressure, brought about not by an exaggeration of the former, but by a diminution of the latter. He quotes Fourrière, Schnabel, and Morax in defence of his position. The glaucomatous cup and the shallow cupping of optic atrophy are both the results of a single process, an ischaemia of the nervous tissue of the retina and papilla. In the one case the ischaemia is the result of the pressure exercised on the arterial circulation from within the globe, in the other it is an outcome of a direct diminution of the arterial supply of the retina and optic nerve. Bailliart has been interested in the study of these cases of optic atrophy with cupping, and has elicited the following facts in connection with them: The retinal arterial
pressure is always above the ocular pressure; the difference between
the diastolic pressure and the ocular pressure remains normal; the
systolic pressure is very variable, sometimes high, sometimes low,
sometimes normal, but there is one constant factor in all the cases,
namely, the feebleness of the arterial pulse-beat, or in other words,
the low amplitude of the arterial oscillations. This feature is a
characteristic of conditions of vasoconstriction, or of the commence-
ment of obliteration of the retinal vessels. What is true of the
trunk of the retinal artery must be still more so of the capillaries.
We may, therefore, assume that over a large area of the capillary
network, the vascular pressure is below the ocular pressure. He
adds that if these views are accepted, all the cases of glaucoma,
without increase of tension, should be classified as glaucoma. The
common factor would be a rupture of the normal relationship
between the retinal arterial pressure and the ocular pressure. Such
a rupture was first pointed out by Sulzer, as characteristic of an
attack of glaucoma (Ann. d'Oculistique, February, 1897, p. 81).

Much of this argument is interesting and suggestive, but to the
reviewer's mind, a needless confusion has crept into the writings on
this subject. Optic atrophy with shallow cupping is a very different
thing, to his mind, from simple chronic glaucoma with its attendant
atrophy. The cupping in the latter case is identical with that which
we meet with in typical glaucoma, and which finds a ready
explanation in the increase of pressure within the globe. He has
found on numerous occasions that patient perseverance with the
tonometer has yielded evidence of increased tension in subjects,
which might easily have been taken under a less careful examination
as supporting the cupping-without-pressure theory. Bailliat's
present thesis, though put in different and attractive language, really
goes no further than the contentions of previous writers, that arterial
sclerosis, so far from favouring an increase of blood pressure within
the eye, has in practice the opposite effect.

(3) Trouble due to arterial hypertension.—If the pressure in the
retinal artery rises, and especially if this rise is accompanied by
vasodilatation with a rise in the venous pressure, the relationship
between the retinal vascular pressure and the ocular pressure is once
again disturbed, but this time in the opposite sense, for the walls of
the vessels will be less well supported than they normally are, and
haemorrhages and exudations will therefore be rendered likely.

This condition of high vascular retinal pressure is met with in its
most marked degrees in albuminuric retinitis. Now, it is known
that this increase of pressure appears in the retinal network of
vessels before the lesions are seen, thus showing that it is not an
effect of those lesions. This is an important fact to remember: in
dealing with an affection characterised by oedema and haemorrhages,
one must take careful account of mechanical conditions so important
as this, and one must attribute to the vascular retinal pressure the rôle of at least strongly favouring the production of the lesions.

In most cases the ocular pressure remains normal, despite the great increase in the retinal arterial pressures. On the other hand, we know that glaucoma and haemorrhagic glaucoma are not exceptional complications of albuminuric retinitis. Bailliart believes that though retinal arterial hypertension may appear, and may be maintained without the occurrence of a rise in the ocular pressure, the latter is often produced secondarily. It is as though the parallelism between the two pressures has a tendency to re-establish itself when disturbed.

The treatment of retinal circulatory troubles.—These are very difficult to treat, but the more we know of their mechanism, the easier will our task become. Without belittling the importance of a rise in the general blood pressure, we must bear carefully in mind that it is the local vascular pressure which must guide and govern our therapeutics. Bailliart furnishes examples:

Example 1. For one reason or another we suspect that a patient has high tension in his retinal arteries; we measure his general blood pressure and find it raised, and so we treat him with hypotensor drugs—his arterial blood pressure falls it is true, but only as a result of vasodilatation; in his capillaries and veins the pressure will rise, and he will thus be more than ever exposed to the danger of local haemorrhages.

Example 2. Vision is steadily falling and the optic discs are showing cupping, despite the fact that the ocular tension is apparently normal; there are mists, or even attacks of blindness, which last some hours. Strychnine makes the patient worse instead of better. We have here to do with arterial hypertension, but the finest peripheral retinal vessels are in a state of spasm or of obliteration; strychnine, being a vasoconstrictor, simply increases the trouble. This patient, with a high general blood pressure, needs vasodilator drugs; anything which will open his peripheral circulation will be as good for him as it was bad for the previous patient.

The same principles must be applied to local treatment: miotics are said to be vasoconstrictors, and mydriatics vasodilators. Whether this be true or not, pilocarpin, when steadily instilled into the eye, has the most excellent effects in retinal lesions, accompanied by vascular hypertension; on the other hand, atropin is dangerous in this class of cases.

Despite the fact that we cannot observe the choroidal vessels directly, we must not forget their importance. We have means of studying them, the best of which is by the observation of the pulse-beat, as transmitted through the walls of the globe, and measured by one or another of the instruments used for determining the
tension of an eye. Bailliart hopes much from a further study of this subject.

A very interesting discussion followed the reading of this paper. Duverger and Barré have controlled Bailliart’s experiments. They hold that his methods of measurement are open to numerous errors, but think that the following general deductions may be drawn: (1) The systolic arterial pressure in the retina is about 100 mm. Hg., and the diastolic 50 to 60 mm. Hg. (2) A continued increase in the retinal arterial pressure has no influence on the ocular pressure. (3) An increase in the ocular pressure has probably no influence on the retinal arterial pressure. (4) The retinal arterial pressure is practically the same as the pressure in the brachial artery, when taken at the eye level; it seems to depend almost entirely on the general blood pressure.

Velte, too, has adopted Bailliart’s technique. He has found the difficulties considerable. His results differ from those of Bailliart, and agree with the findings of Duverger and Barré. The retinal arterial pressure does not appear to be sensibly influenced by a rise in the ocular pressure, but runs parallel with the general blood pressure.

Rochon-Duvigneaud urges that we should let our theories wait on facts and not hurry our building. Every time that he has found high retinal arterial tension it has been accompanied by a high general blood pressure. He does not believe that Bailliart has met with cases with normal general blood pressure together with increased tension of the retinal arteries. Retinal haemorrhages are the results of venous stasis and not of increased arterial tension. He cannot follow Bailliart’s argument as to the pressure within the retinal veins, especially as other factors have to be taken into account, including the viscosity of the blood and the state of the vessel walls. The arterial pulse is a very inconstant phenomenon in glaucoma, and in many cases of this disease, the retinal blood circulation is maintained all through the cardiac cycle, despite the rise in chamber pressure. He cannot agree that this is due to a purely local rise in retinal vascular pressure, and prefers the view that it is a consequence of the increase in general blood pressure which is so often met with in the glaucomatous. The reviewer would point out that reliable statistics do not indicate any causal relationship between high general blood pressure and glaucoma. As the peripheral pressure increases (due to venous stasis), the diastolic arterial pressure may likewise rise in the overfilled retinal arteries. In doing so it approaches, but can never pass, the systolic pressure. So long, however, as it remains superior to the ocular pressure, no arterial pulse will appear. We know that in glaucoma a rise in the ocular venous pressure is a constant feature of the congestive cases, and it is to the association of venous obstruction with intraocular
pressure (caused by an obstacle to the outflow of aqueous fluid from the eye) that we must look for the explanation of the absence of an arterial pulse in so many of our glaucoma cases.

The reviewer has ventured to give Bailliart's views at some length, because they appear to him to be an extraordinarily valuable chapter in the literature of glaucoma. A little more care in the arrangement of the subjects discussed, and in avoidance of needless repetition, would, he thinks, have added not a little to the merits of the contribution. He has, to a great extent, abstained from criticism, not because he accepts M. Bailliart's views in their entirety, but because his object has been to present them as shortly as possible, consistent with clarity. He would be doing that writer a grave injustice were he to omit to mention that he makes no claim to finality in the views which he has put forward, and that the modesty with which he has advanced them is only equalled by their originality and thoughtfulness.

R. H. ELLIOT.

II.—OPERATIONS


(1) Knapp, met with a case of neurofibroma at the apex of the orbit. The tumour was probably connected with the nasal branch of the ophthalmic nerve; there was also a plexiform neuroma in the lid. The tumour, which measured 22 mm. by 12 mm., was found to be adherent to the sheath of the optic nerve; this adhesion was separated, a capsule was isolated and opened, and the tumour was removed, with preservation of the eyeball.

The literature of the subject is given, and two figures serve to illustrate the paper.

R. H. ELLIOT.


(2) Aymard, of the Queen's Hospital for Facial Injuries, Sidcup, says that since his investigations regarding the vascularity of cartilage, he can, with the utmost confidence, understand how cartilage, transferred, rapidly establishes fresh communications with the blood-vessels in its new vicinity and becomes fixed to the capsule in a recent enucleation or to the surrounding tissues in
older standing cases. He illustrates his method of trephining two hemispheres from the eighth costal cartilage and pegging (with cartilage), or fixing together with catgut, these two hemispheres to make a sphere for introduction within the capsule of Tenon. At present the author only uses the method in primary enucleations where there is no sepsis, but suggests that it is worthy of extensive trial in septic cases. Apparently, the usual procedure is for one surgeon to trephine and shape the cartilage while another excises the eye. Apparently the trephine employed, which by the way is very poorly illustrated, is expected to shape as well as cut out the piece of cartilage.

Ernest Thomon.


(3) Herbert's paper is directed to prove that there is no divergence of views between the reviewer and himself on the subject of the value of the method of perchloride irrigation of the conjunctiva before a cataract extraction is undertaken. In this he is unquestionably correct, and his contention that "it is up to" any who meet with suppuration in their cataract operations, to eliminate it by the use of this method, clamours for wide recognition. The credit of originating the idea of perchloride irrigation, before major eye operations, is often given to Herbert, but the practice dates much farther back than his contribution to the subject, and appears to be due to Major Bamber, of the I.M.S., who brought the method before the Indian Medical Congress of 1894; possibly, it may go even farther back still. Many other surgeons have used this antiseptic irrigation, and far from least among such we must include the name of Lt.-Col. Henry Smith, of Jullundur and Amritsar fame. Few, however, have taken such a part in elaborating the technique and in popularising the method as Lieut.-Col. Herbert. This adds to the value of his present communication, which will well repay the careful study of all who are engaged in the operative practice of ophthalmology.

R. H. Elliot.


(4) In the present article Liegard reaffirms his views on the simplicity of substituting a horizontal suture for a vertical one and of doing both stages with one thread armed with two needles.

Standing to the right side for the right eye and using the right hand for the needle holder the surgeon passes the needle into cornea...
Operations

0.5 mm. from limbus, about 12 o'clock, and parallel to it, bringing the point out again by a slight rotatory movement close to the entrance point, this needle is discarded and the second needle is used in a similar way just over limbus, all the time the fixation forceps, with rather projecting teeth, keeping a firm grip on conjunctiva and episclera at 11 o'clock. The needles are passed in and out in one movement and the loop of the thread is situated next the caruncle. After the extraction is completed, the upper thread is pulled on and the knot made. The left eye is done also from the right side over the patient's chest, the needle being passed from without inwards so that the loop lies over towards the outer canthus. In both cases support is taken from the corresponding cheek.

Liegard claims as advantages the fixation at one spot during the insertion of both sutures, the precise and easy adjustment of the space necessary for the knife, the easy access to the eyes when passing in the sutures from below rather than over the eyebrows, the solid and constant point d'appui of the cheeks.

Firm fixation at 11 o'clock by being directly opposite the point of insertion renders this easy, reduces to a minimum injury to conjunctiva and gives more elegance and rapidity to the operation.

Two sketches of the operation and the suture material are given.

W. C. Souter.


(5) In the operative treatment of strabismus, muscular advancement is steadily replacing the older method of simple tenotomy; in a certain proportion of cases a combination of both procedures is advisable.

The weak point in advancement operations is the difficulty or impossibility of retaining the shortened tendon in its new position. The stitches in the sclera remain undisturbed, but those in the cut-end of the muscle, exerting antero-posterior traction (greater in proportion to the degree of shortening), parallel to the muscle bundles, tend to cut through the soft tissues and allow the advanced tendon to slip back. In order to counteract this slipping, Terrien advises and practises the application of an extra suture, which acts as a stay and lessens the traction on the main suture. He states that he has operated by this method in a large number of cases, and has had very satisfactory results.

The complementary stitch is applied in the way now described: but a glance at the two diagrams in Terrien's original article will give a clearer idea than any written description. In operating,
Terrien makes a horizontal incision through the conjunctiva over the rectus tendon, and proceeds in the usual way to the division of the muscle and the insertion of a silk suture through its cut-end and then through the conjunctiva and sclera close to the limbus. Before this is tightened, the extra suture is placed in position. A silk thread, armed with two needles, is used, white if the other stitch is black, to avoid confusion. One needle is passed vertically through the superficial layers of the sclera opposite, but a little below the tendinous attachment of the rectus; it picks up about 2 mm. of the sclera, then emerges, and is again inserted for a like distance and the silk pulled through. There is now a vertically placed stitch, embracing a strip of sclera rather wider than the rectus muscle. The first silk stitch (that in the muscle) is now drawn tight and knotted, bringing the cut-end of the tendon forward to its new position. The tendon lies over the complementary suture. Each end of this latter is now passed through the adjoining cut border of the conjunctiva, about 4 mm. from the edge, and the ends are tied firmly, but not very tightly. This stitch brings together the lips of the conjunctival wound and binds the rectus tendon closely to the sclera. The stitches are removed on the seventh or eighth day.

J. B. Lawford.

(6) Van Lint.—Trephining the os unguis and insertion of a rubber drain in chronic dacryocystitis. Arch. d’Ophtal., Jan., 1920.

Van Lint maintains that by a surgical technique much simpler than that of dacryocystorhinostomy a channel between the lacrimal sac and the nasal cavity can be established in cases of chronic lacrimal obstruction. He recommends the procedure to which the above title applies. The operation is easy to perform and the immediate results are “ideal.” The newly formed canal, however, is prone to close ultimately, but even then the author thinks the results are at least as good as those of extirpation of the lacrimal sac. A detailed description of the technique is given, the steps of which are, in brief: cocaine anaesthesia of the region of the sac and of the middle meatus of the nose; plugging of the middle meatus with gauze to avoid injury of the turbinate; the lacrimal sac is exposed and its anterior wall incised in its whole length; the trephine is placed in the lower end of the sac against the bony wall and the lacrimal bone perforated; removal of the plug in the meatus is followed by haemorrhage which is arrested by a strip of gauze pulled through the trephine aperture by forceps; this is followed by a rubber tube, the upper end of which is cut short in the lacrimal sac; the skin wound is sutured over the end of the drain; the nostril is plugged for 24 hours. About the eighth day
physiological serum is injected through the lacrimal punctum; the tubing is removed from the tenth to the fifteenth day. For several weeks the lacrimal passages should be syringed once a week.

J. B. Lawford.

BOOK NOTICES


Col. Maynard's "Manual of Ophthalmic Practice" is obviously the work of an experienced and practical surgeon, but it suffers from the source from which it was derived. This—as stated in the preface—was a series of lectures, in which it is usually necessary, in order to cover the ground in a comparatively short space of time, to deal with many of the subjects somewhat superficially.

The book is lavishly illustrated both with original drawings, and reproductions from other books. It is possibly difficult to obtain the services of an expert medical artist in India, but when so much expense has been devoted to colour reproduction, it is a great pity that the original illustrations in colour are so little illuminating and are not on a more uniform scale. The lack of drawing also in many of them is so glaring that one might infer that microcornea was a common occurrence among the natives of India.

We cannot help regretting that the ripe experience of so capable an ophthalmic surgeon has not been placed before the profession in a more thorough and accurate form.


We welcome the second edition of this book; the first edition appeared in 1908, and since that date much new work has been done, particularly in glaucoma. The descriptions of the operations of Elliot, Lagrange, and Herbert are excellent, as are, of course, the chapters on cataract extraction. We note that Colonel Maynard does not advocate the removal of the lens in its capsule.

We think that six pages out of 248 are not sufficient for a full description of the operations on the ocular muscles, but as squint