In 1859, we find Traube, in his *Klinische Untersuchungen* (page 969), noticing the association of amyloid degeneration of the kidney with albuminous urine. Renal casts passed in the urine during life, and plugs loaded with fatty detritus were found blocking the tubuli uriniferi after death. The kidney presented a waxy appearance; the degenerative change was nearly entirely confined to the blood-vessels; no appearances suggestive of parenchymatous inflammation were perceived, or led him to attribute the plugs or casts to active changes of or in the renal epithelium. Fatty degeneration of the renal epithelium might, therefore, be induced by other causes besides inflammation; and in amyloid, perhaps, as in some other cases, should be attributed to a kind of necrosis.

Casts, as Frerichs had shown, neither by their size, nor their quantity, nor their characters—whether full of cells, or of fat, or of granular matter, or of blood-cells—could be accepted as evidence of the actual state of the kidney; although there was a great unanimity of opinion among observers that fatty casts were signs of ill-omen, and that sediments, which contained large hyaline casts, and numerous white (or compound granular) cells, associated with botryoidal clusters of free nuclei (*vide* Dr. Basham, fourth edition, page 130), usually indicated a most intractable, because most widely diffused, form of renal degeneration.

Differentiation was the order of the day; the pendulum which Frerichs had forced back to Bright's last-issued idea, that his disease was, essentially and pathologically one, interrupted by death, and thus encountered at different stages, was swaying, irresistibly swaying, in an opposite direction.

Beer, in his work on the connective tissue of the human kidney, published in 1859, was calling attention to two distinct forms of interstitial hyperplasia, which he had observed in renal disease: the one circumglomerular, the other intertubular. The circumglomerular interstitial nephritis was, according to him, marked clinically by yellow-coloured urine, of low specific gravity, and presenting little urinary sediment; whereas the intertubular commenced with hæmaturia, and was attended by urine of high specific gravity, and possessing much sediment.

Traube writes in 1860 (*Klinische Untersuchungen*, page 969), appealing that the name of Bright's disease should be given up, because it promulgated error, and led to the confusion together under one head of a great variety of forms of renal degeneration; the parenchymatous inflammation did not constitute it; this was a first change only, which might lead to it. Interstitial nephritis, or interstitial hyperplasia, could be developed without any previous stage of parenchymatous swelling; and parenchymatous swelling was to be found in the renal changes secondary to old heart-affections. Typical parenchymatous swelling existed in the kidney of variola, and in all other acute febrile processes; but this did not necessarily go on to degenerative renal disease. At the same time, he admitted that glandular or parenchymatous swelling of the epithelium of the cortical tubes was an ordinary accompaniment of interstitial nephritis, and of amyloid disease of the vessels of the kidney.

Acts of Parliament and the Queen's pleasure apart, there are few things so difficult to shake yourself quit of as the name of a place, or of a man, or of a disease. While Traube was urging that the name of Bright's disease should be surrendered, because of its vagueness and uncertain applicability, Dr. Goodfellow, in some admirable Lectures on Diseases of the Kidney, published in 1861, was as strongly recommending its re-adoption in England, in lieu of albuminuria, for a varied but peculiar (specific) renal degeneration, which possessed certain well ascertained symptoms, and a moderately precise pathology of its own. He writes (p. 237): "It is from not separating the acute from the chronic stages, and from taking the mixed varieties as so many independent forms, that so much confusion has arisen."

Goodfellow was no blind believer in casts; he advised those who wished to make a sound diagnosis of the extent, and would forecast the issue of any case of renal disease, to go back to estimating the amount of urea excreted *per diem* by the patient; and directed attention to the grave secondary danger undergone by the blood, when the normal elimination of urea was rendered defective, in consequence of any renal inadequacy.

Dr. Goodfellow's lectures showed considerable knowledge of foreign labours, as well as original ideas. He argued (page 54), that the impurity of the blood in chronic renal disease might act, much as solutions of urea in laboratory experiments, in destroying the red blood-cells, and causing them to break up. At page 217, he compiled a valuable table of measurements of the tubuli uriniferi, and of the small arteries of the kidney; and he was the first, at all events in this country, to recognise the important part played by the arteriolæ rectæ as channels whereby the blood could pass from the renal artery by a short

cut into the renal veins, when the circulation by the longer route through the Malpighian bodies in the cortical substance, was obstructed by swelling of the tubules, or effusion of fibro-serum into the intertubular lymphatics—a point which had been insisted upon by Professor Virchow in a very interesting pamphlet, published about 1860.

THE CROONIAN LECTURES ON THE INFLUENCE OF THE CIRCULATION UPON THE NERVOUS SYSTEM.

Delivered before the Royal College of Physicians.

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LECTURE II.—PART I.

IN my last lecture, I endeavoured to describe means whereby the entry and exit of blood to and from the interior of the cranium are so arranged as to favour the maintenance of a constantly sufficient supply to the brain, whilst obviating ill results of undue stress of blood; and I then passed on to the effects produced when great atmospheric pressure forces blood into the closed cavities which contain the nervous centres; and I finally digressed to point out the weakness in the circulation of the lowest part of the spinal cord, which arises through its more precarious supply from above, and want of reinforcement from below. Whilst upon the arterial supply to the spinal cord, it occurs to me to observe a point which has struck me forcibly in reading descriptions of experiments made for the purpose of tracing the course of fibres in the cord, because the mode in which the arterial supply may influence the results of such experiments seems apt to escape notice; and this may be a part of the reason why different observers differ in their results. Thus, for instance, in one of the experiments made by a great master in this branch of investigation, a comparison is set up between the effects upon the sensibility of posterior nerve-roots when incisions are made near together across the posterior columns above and below a single pair of roots, and the effects produced when similar incisions are made far apart, including five posterior roots between them. When such incisions are close together, sensation in the single pair of roots between is lost; when the incisions are far apart, the roots not only retain their sensibility, but appear rather more sensitive than natural. If we bear in mind the posterior spinal arteries and the mode of their reinforcement by occasional branches on the nerves, we shall see that, when the incisions are close together, the posterior spinal arteries above and below are severed, and the small segment is drained of its blood, so that surely insensibility would arise from this cause alone. If, on the other hand, the incisions include as many as five nerve-roots between them, there is a very great probability indeed that a large reinforcing artery will enter the posterior spinal in so great a length, and thus maintain a blood-supply. But it may be asked, Will such an explanation serve to show why the sensibility is increased? We must not, however, assume that evidence of unnatural tenderness is a proof of increased sensibility; this, no doubt, is not the case. I have now under my care in Philip ward a man who has paralysis and insensibility to touch in his right leg, but nothing the matter with his left leg. In this man, a pinch which causes only a smile when applied to his sound limb, produces a cry and a request against its repetition when it is applied to the limb which cannot feel a touch or localise the pinch. Hence signs of pain do not prove sense of touch, and the conductors of normal sensibility cannot be traced by signs of pain. In this man, the skin of the affected limb is pinker and thicker than the skin of the sound limb. This far from unprecedented case shows that we may expect proofs of unnatural sensitiveness to coincide with considerable anæsthesia; and the unnatural sensibility to pain in the posterior roots may itself be taken as some evidence that the normal sensibilities of the part were reduced. In following out the points that have arisen with reference to the circulation of the spinal cord, I have digressed considerably from the main line of my argument, which is bent on inquiring into the soundness of the view which teaches that congestion of the brain is an essential or primary disease.

And now I will return to the effects of caisson-pressure upon the brain, if one can so speak, when we find that the pressure had no effect

at all upon the brain, except to give a general feeling of buoyancy, which was, I think, very properly ascribed to the greater quantity of oxygen that entered the blood under high pressure. The caisson-pressure offers us an example of active and unobstructed connection of the vessels of the brain with oxygenated blood, no complication with a venous state of the circulating fluid being present, so that it may be described as a forcible arterial congestion without impediment to free exit of the blood; an active arterial congestion, purely affecting the blood-supply, and free from complication with disorders of the nervous tissue.

The familiar phenomena of strangulation and of obstruction of the jugular veins, causing forced delay of exit of the venous blood, should not be allowed to complicate the question which I am approaching. But as the sensations accompanying venous obstruction in the neck give rise to a very general bias in favour of the notion of cerebral congestion at large, it may be well for me to state the bearings of this subject on the special question, whether congestion of the brain arises independently as a distinct form, or as several distinct forms of disease. It would be scarcely possible for me to mention all the many modes in which venous congestion of the brain is constantly brought before us in forms more or less pure. Perhaps the subtlest of the causes of such congestion is that which has been well investigated in the study of school-diseases. When children bend over low desks during their lessons, a proportion of them are observed to suffer from headache, and some become faint, and some bleed at the nose; and such results may be ascribed to cerebral congestion. But, in using such an expression, we must notice that the meaning of it covers at least three distinct things. The ill effects which come about through lowness of the desks in schools are set down in detail by Virchow: first, to the mental strain of the learners; second, to occasional check to respiration during close attention; and third, to obstruction of veins by bending the neck. But each of these things is quite different from the others in its mode of operation, widely different indeed; and each in its own way appeals for its special means of relief. Mental overstrain is cerebral exhaustion; lack of respiration is carbonic acid poisoning, together with deficiency of oxygen; and compressed jugulars give delayed exit of venous blood, and consequent venous congestion. The term congestion of the brain has been given in a general way to these rather complicated conditions; and there is no harm in such a use of the term, unless it induces us to forget the several distinct causes which combine to produce the ill results ascribed to it, and in the term congestion of the brain to lose sight of the mental exhaustion and carbonic acid poisoning. Indeed, if we wish to ascertain the effects of obstruction of the jugular veins and retention of venous blood, we must choose some more pure examples of such obstruction.

I will not enter more fully into the effects of impeded flow in the jugular vein, than is necessary to define its relation to the "congestion of the brain" of authors with which I have to do. Such obstruction may be acute or chronic, and may vary in degree in either case from slight obstacle to absolute closure. Many observations of the extent of effects produced by check to the flow in the jugular veins have led me to the conviction, that venous obstruction is borne with so much impunity when it is certainly present and severe, that one must not suppose such obstruction to be inducing serious results in cases when evidence of its existence is absent or trifling.

When speaking of obvious causes of obstruction to the current in the jugular vein, I refer in particular to cases of prolonged paroxysm of whooping-cough, or of the cough of emphysema, and to cases of tumour compressing or involving the jugular vein. When a person is in a seizure of either of the forms of protracted cough which I have mentioned, the evidences of venous obstruction and imperfect aëration of the blood become very apparent, often painfully so, and yet cerebral disturbances but very rarely show themselves. I have been impressed by the small number and slight degree of complaints of cerebral symptoms in such cases. Twice only have I known persons affected with old bronchitis, and habitually almost cyanosed, to stagger and fall in the extreme height of their paroxysms of cough; the description given by the friends was of extreme blueness and turgescence of veins when the staggering occurred, so that cyanosis must have largely shared in its causation. Everyone must have seen the veins rise on the forehead and a dusky colour suffuse the face of persons coughing violently, when, meantime, nothing noticeable happened to the comfort of the brain. What I am seeking thus to establish is the proposition, that congestive strangulation of veins is borne with impunity up to visible and obvious extents, and therefore symptoms cannot be ascribed to it when the visible and obvious conditions of venous overfulness are wanting. Also, I believe that general experience will be with me in recalling how, in examples of tumours in the cervical region pressing upon the jugular veins, and even involving and closing one of them, the comfort of the

brain is surprisingly little invaded. One of the most clear-headed and intelligent patients I have seen had the neck enlarged so that, shortly below the chin, it exceeded the girth of the head and face, and these were livid with venous obstruction; yet she denied to my questions every sign of cerebral annoyance. When such ascertained venous congestion, producing obvious retention of blood, fails to cause serious symptoms, unascertained and unproven congestion cannot be called in as a supposition to explain serious symptoms.

I have so far endeavoured to adduce examples, first, of extreme arterial intracranial blood-pressure; and, second, of visible and obvious intracranial venous blood-pressure; so that the absence of serious ill effects, under conditions of ascertained great or evident blood-pressure within the skull, may be within our minds in approaching the consideration of the disorders described under the name of congestion of the brain.

There is still one step remaining before I can proceed to the task of examining into the clinical symptoms described as due to this disease; and that is, to make clear how far it is possible to show, by *post mortem* examination, that a person has died from congestion of the brain. We must spend some time upon this question, because, following and supporting the clinical descriptions of congestion of the brain, we find a morbid anatomy of congestion of the brain; just as after the clinical description of acute pneumonia we find the morbid anatomy of acute pneumonia. And the morbid anatomy of acute pneumonia does verify and explain the presence and the symptoms of pneumonia up to a certain point, failing only in showing why that particular person died at that particular time, and leaving it to clinical observation to answer that question, which is the vitally important question in each case. The *post mortem* examination of pneumonia never tells us why that particular death occurred, but it leaves us in no doubt about the characters of the case; one can be quite sure that pneumonia did exist. But will any morbid anatomy of congestion of the brain afford us the same kind of proof; or do anything of the kind? The question is, Can you, in any individual case, prove by *post mortem* appearances that death was caused by congestion of the brain, or even that a state of congestion of the brain, or overfulness of its blood-vessels, preceded death? I believe these questions must be answered firmly in the negative. In establishing such a negative, one has to meet with a very strong prejudice rooted in the most tenacious of grounds—the ground of convenience and of ancient and universal acceptance. After examining a body dead from brain-symptoms, when all you were able to see in your examination was only that the veins were very full of blood, it is very convenient to be able to say that death was caused by congestion of the brain, and it sounds much better skilled evidence than to say that death was caused by insensibility; so that the doctrine of congestion of the brain is convenient; and the universality of its acceptance may be illustrated by the naive earnestness with which authors of great works adopt it. One author of a great work says, on one page, that patients never die in the first stage of congestion of the spinal membranes; and on another page, he says, that *post mortem* examination shows in the first stage redness of the membranes; but if they do not die, how is the redness known? It is very difficult to deal with a delusion thus deeply rooted in faith and prejudice—a delusion which is much favoured by occasional very striking appearances at imperfectly conducted *post mortem* examinations. Thus, in the Clinical Society's *Transactions*, there is a very remarkable spinal case—probably a case of acute myelitis, but introduced to the Society as congestion of the spinal membranes; and, indeed, the membranes were seen to be frightfully congested. But Mr. Hilton pointed out long ago the necessity of observing whether the cervical veins are divided, before you make a remark upon the congestion of the veins of the nervous centres. And, indeed, if we do not divide these veins, we will be sure to see frightful congestion when we look at the membranes of the brain and spinal cord; but the observers of this case had only opened the cranium and spine when examining these membranes.

I mention these examples only to illustrate how readily the mind of a physician adopts, on insufficient evidence, the assumption of a congested state of the brain; but, in some of our most justly and highly valued text-books, we find, amongst a series of excellent, learned, and ably written articles on particular diseases, that a long chapter on congestion of the brain finds a place; and this chapter gathers groups of symptoms of a disease called congestion of the brain, and collects signs of morbid anatomy of the disease so named; and the gathering of symptoms includes a sweeping together of all such things as are obscure and associated with stupor, and have an acute course; and the collection of anatomical signs includes a sweeping up of all the marks of change in the blood-vessels or texture of the brain, however evidently chronic or ancient. And the collection of chronic and ancient changes stands, then, to the gathering of acute and obscure symptoms as the morbid anatomy of a definite fatal disease, congestion of the brain.

And so in our great books we find this chapter on congestion of the brain, made up of obscure fragments out of the history of all sorts of diseased states. In the work which contains the chapter, we generally find that there is already a chapter on epilepsy, and another on sunstroke, and another on alcoholism; and then, in the chapter on congestion of the brain, we have a treatise on epilepsy that is not quite epilepsy, and sunstroke that is not quite sunstroke, and alcoholism which has changed so that it is now deserving to be called not alcoholism, but congestion of the brain. But, when we ask for evidence that these hazy states of quasi-epilepsy, quasi-sunstroke, etc., are really due to congestion of the brain, we meet with a very difficult state of affairs, very prejudicial to clear dealing with the subject; I mean we are met with a foregone conclusion, a sort of traditional certainty about the matter, as if you ought not to ask for evidence. Believing as I do, that there is no such definite fatal disease, I find this a very unsatisfactory state of affairs; and I was very glad to see, in a recent and very valuable work on medicine, that the author of that work, himself a pathologist of great experience in morbid anatomy, speaks thus of this topic of "congestion of the brain": "So many nervous phenomena are commonly referred to congestion or anæmia of the nervous centres, that we can scarcely presume to pass over them in silence." One could have wished that the conviction thus evinced had led this distinguished physician to deal more summarily with a doctrine upon which he thus confesses himself forced by common reference to touch.

As one finds categorical descriptions of a morbid anatomy of this disease, congestion of the brain, standing on the highest authority in our text-books, I cannot ignore them, or merely say there are no such signs; but must deal with the several anatomical appearances which are advanced, as showing *post mortem* that the brain was congested during life, my position being that there is no valid evidence of the kind. Hence I must now trouble you with an examination of the *post mortem* appearances which are alleged to prove congestion of the brain. We find writers giving four conditions as showing congestion. I will take them *seriatim*. The first condition advanced is a swollen state of the brain, so that it seems, after removal of the calvaria, almost too large for the cavity which contained it. The dura mater seems tightly stretched over it; and, on reflecting this, the convolutions appear broad and flattened, and the sulci less obvious. Now here we have evidence of swelling of the brain; but, as to our power of drawing any conclusion from such a swollen state, it is curious that the description makes no allusion to hypertrophy of the brain, which is treated of elsewhere in the same work, and is known only by its causing a general enlargement of the whole brain. I have never seen such hypertrophy, but the flattening described has very frequently come under my observation; and my impression is, that in no instance did I ever meet with such flattening, unless some obvious cause of expansion of the brain has been present, in the form usually of an increase of the intraventricular fluid, or else of apoplectic bleeding, or of tumour, in which case the swelling and flattening are more localised. The next evidence mentioned is the distension of veins and capillaries with blood. The veins are tortuous and varicose; the grey matter is dark; both this and the white matter show abundance of bloody points and gorged vessels; and this description finishes by saying, "It is extremely difficult to draw the line, and say what is morbid and what consistent with health". Now it is better, nay it is necessary, to say firmly that it is not only extremely difficult, but it is simply impossible, to draw the line, and say what is consistent with health. Indeed, by bringing in the idea of health when the person is dead, we confuse the issue; and I should not mention this, except to remark that the visible state of affairs at the *post mortem* examination is the point that concerns the morbid anatomist, and not the inferential state of affairs during life and health; for this is not any longer at all in the question; which question is, whether the mode of dying, the position of the body after death, or the manner of making the inspection, will not determine the appearance of extreme overfulness of the vessels in the brain of the dead person, independently of the conditions which were antecedent to all this, and led to his having so to die. Can we infer, from the amount of blood in the brain after death, the amount during life? Perhaps one could not find a more decisive answer to this question than the answer given by Kussmaul and Tenner. These observers were investigating the effects of ligature of the cervical vessels, and it was most important for their purposes that they should be able to estimate the resulting anæmia of the brain by *post mortem* examination. Their experiments were very numerous. They were testing the results of a simple mode of treatment of the brain; they had it in their power to place the bodies in any position they liked, or in any circumstances of temperature, etc.; and they knew accurately the conditions actually allowed. This is what they say as the result of their investigations. "We could never deduce any results from the *post mortem* examinations undertaken with

a view to determine the state of fulness before death of the most important parts of the vascular system *viâ* the arteries and arterial capillaries; and, even in the most favourable instances, when similar inquiries were directed towards the veins, our results could only be looked upon as approximate. It appears, therefore, to us to be especially necessary to study carefully the state of the brain-circulation in asphyxia, narcosis, intoxication, etherisation, etc., in the living animal, according to Donders's method (that is, by insertion of a watch-glass into the skull)." Now, if this is the result when the conditions before and after death were determined and known, how can it be said that the amount of blood found after death in the human brain will show the amount present before death, when the conditions before and after death were unknown? Again, why should we take the amount of blood found *post mortem* in the brain as a criterion of the amount present there during the disorder that lead to death, when the amount of blood present in other parts after death cannot be taken to indicate the amount present before the act of dying? It may be necessary here to examine the exact bearing of evidence in some cases where we appear to accept fulness of blood after death as evidence of fulness of blood before dying. Let us take the most striking example we can choose, in which we might be led to suppose that we are in the habit of drawing such a conclusion, and thus do ourselves an injustice. Now it is true that, where organs are situated close to seats of long continued and positive hæmal obstruction, as, for instance, the liver, or stomach, or lungs, when seen after death from heart-disease, the amount of blood in the vessels of these parts forms a striking feature at the *post mortem* examination. We observe great overfulness of the veins, and we know that overfulness existed during life. But it is not because of the amount of blood found there after death, that we know there was so much blood there during life; for the amount of blood present *post mortem* is really only the result of the mode of dying. Death in mitral disease is due to stoppage of the heart under intense engorgement, so that the great vessels near the heart are very full in the moment of death, and remain so afterwards in the ordinary position of the body; and from this cause there is much blood in the wall of the stomach; and, meantime, our knowledge of the ischæmic state produced by the cardiac obstruction makes us aware that the same condition existed during life; also the presence of excess of tough mucus or superficial ulceration may give evidence to the same effect. But, as to the particular question, whether a great amount of blood in the stomach-wall after death will serve to show that there was a great amount of blood in it before dying; if we were asked that question, we must, I believe, answer it firmly in the negative. Great amounts after death may have been preceded by great amounts before the person began to die, as heart-disease shows us; but the stomach may be deeply congested by sudden disease or accident, causing asphyxia merely as a manner of dying; so that you cannot, because the stomach is purple after death, say that it was so before dying commenced. The importance of this subject in many aspects of medical practice induces me to follow it out fully, because we find it on all hands assumed, and by the highest authorities stated, that redness of the brain shows congestion or even inflammation of it. Thus, in a very thorough and able article on Tubercular Meningitis, we find it said of the membranes of the brain, that they show "that first great sign of inflammation, redness"; and, even in works devoted especially to morbid histology, the same fallacy is encouraged. Rindfleisch, speaking of acute hyperæmia of the brain, says: "A slight flush of the medullary substance, a faint tinge of red mingled with the usual grey hue of the cortex cerebri, must be taken as very significant indications; wherever these are lacking, we must not jump to the conclusion that they did not exist during life". Of course, the question is not of jumping to negative conclusions, but founding sound positive conclusions; and nothing can be more misleading or wrong than such a statement as that a faint tinge of red, mingled with the usual grey hue of the cortex, must be taken as a very significant indication. One is tempted to think the author had not looked carefully into the variations of colour to be observed in brains, as they ordinarily appear before the pathologist: faint tinges of red are constantly to be seen in the brains of persons dead from different diseases; and the blood is often very curiously and irregularly distributed, under entirely indifferent conditions, marbling and mottling the grey matter with pink and purplish tinges. A section of the cortical grey matter, or, more especially, of the corpus striatum and thalamus, will convince any one of this in almost any case. So also it is misleading to speak of that great sign of inflammation, redness, as appearing *post mortem*. During life, redness, when associated with swelling, heat, and pain, is at one with these associates in proving the existence of inflammation. But, after death, the redness, which was due to the enlarged scope of vascular play, ceases, because vascular play has ceased with life. The consequence is

not doubtful; and it is quite certain that, after death, the redness goes from inflamed and congested parts. Such curious issues may turn up on this point, that I think it very necessary to clearly recognise that no degree of redness or pinkness, or overfulness of blood about the brain or its membranes, can prove that there was any morbid state of circulation within the head before the act of dying. Death by asphyxia increases the amount of blood in the head; but a dependent position of the head after death, if only for a short time, will cause a similar increase. It should be taught that it is a sign of ignorance to say, in a coroner's court, that congestion of the brain was found, on *post mortem* examination, to be the cause of a person's death.

Passing on, now, to the next point advanced as morbid anatomy of cortical congestion, one finds the description lays down tortuosity and varicosity of the larger veins of the brain as evidence of such congestion. Now, if by this is only meant the bending of the veins through overfulness, it is but a sign of that overfulness; and Kusmaul and Tenner have disposed of overfulness for us. But, if it be really meant that the veins are varicose, I think moderation of the description is desirable; at least, in the several thousands of inspections conducted by me, I never saw a varicose vein upon the brain. It is true, that we often find the veins thickish and opaque near their endings, but the veins have their share in the general thickening of fibrous parts within the skull in old age. I will take this thickening, along with *l'état criblé*, which is given as another proof of congestion of the brain: *L'état criblé* is a perforated look of section of brain, usually in the corpus striatum, and due to cutting across of dilated channels, which are dilatations of the channels in which the arteries run. The thickish vein and perforated-looking appearance are signs of senile wasting of the brain—such as results in elderly people from a hard life, and results sometimes in younger people from a still harder life, in which they may have poisoned themselves with alcohol, or, by too much continued stress of emotional existence, have been their own poison to the same effect. In the process of growing old the brain shrinks, both as a whole so as to leave space between it and the skull, and interstitially so as to allow spaces to form around its internal vessels, giving *l'état criblé* on section. The skull is thus not sufficiently filled, and then everything movable and within reach is drawn upon to fill the vacuum. The call tells first on the cerebro-spinal liquid, whose function is to supplement the brain in filling the skull. But, when the natural amount of this liquid is insufficient, the blood must enter and be retained to fill the rest of the space. But the excess of venous blood thus retained lends itself to encourage the growth of the lower forms of tissue, especially the connective tissues. Hence, as a consequence, you find thickening of the membranes; and this appears specially in what I called the tidal area, where the locking system in the veins tells most effectively, and where Pacchionian glands in time appear, the vein-walls thickening a little in the meanwhile. Even the cranium itself, having its dura mater kept full of blood, thickens inwards, so tending to reduce the cavity. But, as to the question before us, which concerns itself only with the disease called congestion of the brain, what we have to see is that all these things are most gradual and slow effects of chronic shrinking of the brain, producing secondary effects most gradually and slowly. Thus it all comes gradually about as a progressive system of events, constituting the character of a period of life, and explaining the creeping approach of age; but in no way serviceable to furnish explanation of sudden insensibilities, and other phenomena, described as acute disease under the title of congestion of the brain.

And now let us look at the remainder of the morbid anatomy advanced in support of the supposition, that sudden serious illness is caused by rush of blood to the head. The description lastly sets down, as such evidence, a quantity of blood or hæmatin around the vessels, twisted and varicose capillaries, evidence of bursting of capillaries, orange coloured spots and specks of crystalline hæmatoidin, and especial stress is incidentally laid on the observation of blood-pigment around the vessels in the brains of excitable lunatics. Now there is no doubt that blood-pigment is very frequently met with about unsatisfactory-looking capillaries, when the brain of any adult is searched through with care. At least, I was led to this conclusion when, during the first few years of my office as Pathologist to Guy's Hospital, I used to take away about a cubic inch from the neighbourhood of the island of Reil in every brain I inspected. For, after the vessels of these portions of brain were washed and examined, the result was such as to assure me that, when anyone over twenty-five years of age replies in a satisfactory way to an inquiry as to his health, he expresses the truth of the matter only so far as he is aware; and his answer overlooks the condition of a considerable number of the capillaries of his brain, much in the same way as it overlooks the state of his skin at some pimple, or perchance the caries of some tooth. In fact, were a pathologist with a great microscope to spy through all our brains as we sit here in states of satis-

faction, he would certainly see a great deal in the way of tortuous capillaries and dots of yellow pigment; a great deal that under the microscope would look very alarming. But so constant are moderate amounts of the changes now in question, that one cannot justly ascribe any special significance at all to moderate degrees of them; and surely experience would suggest great caution in allowing importance to the observation of blood-pigment in the brains of excitable lunatics, because, if the amount of pigment be little, it is not more than other people have; but if it were much, then would not the question arise, whether the excitable lunatics had struck their heads, or perchance received blows in the course of the care they required to have taken of them? Numerous broken ribs are not unfrequent morbid signs in asylum anatomy. A great deal of blood may be effused in the brain through blows from which people recover. A fatal hæmorrhage into the brain is nearly always a large hæmorrhage; and many people recover from blows which, more or less, approach a fatal result without reaching it. Those who recover must, then, have the moderate amounts of hæmorrhage. I have seen the middle and anterior lobes of the brain stuck fast to the skull, and imbedded in old yellow pigment and hæmatoidin, showing that most extensive crushing of the brain had been survived; so that sharp blows or falls upon the head, such as everybody not more than commonly fortunate has to suffer at least several times in his life, are not without effect in doing more or less to throw out spots of blood here and there for future microscopic observation to regard as evidences of congestion of the brain. But, besides accidental injury, there are other mischances to the blood through fever with purpuric tendencies, or through morbid states of the blood generally; and to these rather than to any such disease as congestion of the brain, or determination of blood to the head, we must be prepared to ascribe those changes in the capillary vessels, and the little spots of blood-pigment, that are found on careful microscopic examination of the brain. They are the results of all the accidents, little and great, that have made their mark, large or small, on the brain in the course of its life. It must be remembered that blood-pigment is probably never removed when once it has been formed, but it remains at the spot as a permanent register of the accident that produced it. I trust that I have sufficiently shown that we have no anatomical grounds for believing that there is any malady whose cause is overfilling of the vessels of the brain with blood.

[To be continued.]

ON AN IMPROVED MODE OF EPILATION IN RINGWORM. As the subject of the treatment of ringworm (*Tinea tonsurans*) is just now attracting special attention, I wish to draw attention to a method of epilation which I have recently practised, and which I believe to be more effective, more expeditious, and less objectionable than epilation by forceps. It is generally acknowledged that epilation is desirable. It is admitted by every one, that epilation by means of the forceps is, at the time when it is most required, that is to say, when the hairs are the most diseased, impracticable, on account of the extreme brittleness of the hairs.

My plan is this, and it is a very simple one. When scabs exist over a patch of ringworm, I peel the scab carefully off in one piece. When scabs do not exist, I produce them artificially, and then carefully peel them off in one piece. Thereupon the under surface of the scab is seen to be thickly studded with the white frosted hair-stumps which are peculiar to ringworm, presenting the appearance of the chin of a grey-bearded man who has not been shaved for a week. Now these stumps are considerably longer than any bits that can be pulled out or rather broken off by means of the epilating forceps; it might be supposed that it is impossible to peel a brittle scab off in one piece, and that it would be painful to attempt it. But the scab may be expeditiously taken off whole and without any pain, if it be removed by means of the small sharp steel spoon that I am in the habit of using for performing "erasion" in cases of lupus. It is sold by Messrs Weiss of the Strand. The scab adheres to the skin *only all round its edge*. By applying the side edge of the spoon to the edge of the scab and then slightly rotating the spoon, the edge may be completely detached all round without hurting the skin in the least degree. Then commencing again at one edge of the now loosened scab, and advancing the spoon along the under surface of the scab, constantly making use of the lever movement which is effected by repeated slight rotations of the spoon, the whole scab may be rapidly removed in one unbroken piece with the effect that I have described.

I should add that I first remove the hair of the whole of the scalp by means of my human hair-clipper, which is sold by Messrs. Adie of Pall Mall, and by the various instrument-makers, and which I believe is now in general use as an aid in the treatment of diseases and injuries of the scalp.

BALMANNO SQUIRE.