A LECTURE ON THE PROGNOSIS OF CEREBRAL HÆMORRHAGE.

By JULIUS ALTHAUS, M.D., M.R.C.P.,
Physician to the Hospital for Diseases of the Nervous System; etc.

GENTLEMEN,—Not fewer than twelve thousand persons die annually in England and Wales of cerebral hæmorrhage; and we may assume that at least twice as many people living in these parts suffer from the effects of it. It constitutes, after convulsions, the most fatal of all diseases of the nervous system, carrying off year by year, with unerring certainty, more victims than either paralysis, epilepsy, or insanity. If we consider it only numerically, therefore, cerebral hæmorrhage must be ranked with the most important diseases which we are called upon to treat in practice; but, as it threatens the life chiefly of the aged and the middle-aged, that is, practically speaking, the heads of families, its importance appears by this circumstance to be considerably increased, for the social position of entire families is often completely altered by its occurrence.

In spite of its frequency and importance, however, the anatomical and clinical features of cerebral hæmorrhage have only of late been more accurately ascertained. The old ideas about its being due to the rupture of an atheromatous blood-vessel are still found in the textbooks; and many highly significant symptoms, which are not only of great pathological interest, but also of considerable value for determining the prognosis of the affection, are not yet familiar to practitioners, from attention not having been sufficiently directed to their occurrence and meaning.

I have, on a previous occasion, fully gone into the anatomical part of the subject; and I will, therefore, now only remind you that cerebral hæmorrhage may take place—

1. *By rupture of the large cerebral arteries*, such as the middle cerebral and basilar, after these have undergone aneurismal dilatation.
2. *By rupture of the capillary vessels*, which is generally caused by injury to the head, either direct or by contre-coup; but also occurs in the course of that chronic inflammation of the grey matter which is found in some forms of mental disease; and may be consequent upon embolism and tumours which grow in the cerebral substance and corrode the coats of the blood-vessels. It is also occasionally observed in leukaemia, where it is owing to accumulation of white corpuscles in the capillary vessels, which thus gradually become distended, blocked up, and ruptured.
3. *In consequence of contracted granular kidney*, which leads to blood-poisoning, and consequent over-action and hypertrophy of the coats of the arterioles, and hypertrophy of the left ventricle. In cases where the hypertrophy of the coats of the cerebral arterioles is not sufficient to counterbalance the excessive impulsive force of the left ventricle, cerebral hæmorrhage from rupture of these arterioles may be the consequence.
4. By far the most important form of cerebral hæmorrhage, however, is that which occurs by rupture of the cerebral arterioles, after these have undergone the change known as miliary aneurismal dilatation. *Miliary aneurisms* were first described by Virchow; but their frequent occurrence, and their importance for the pathogenesis of the common form of cerebral hæmorrhage, was first pointed out by Charcot and Bouchard. It is this latter form, which occurs in the immense majority of cases of what is commonly called *apoplexy*, and constitutes a disease of itself, which has special clinical and pathological features. To be able to make a reliable prognosis in such cases is quite as important for the physician as it is to treat them; and this is absolutely impossible without knowing what significance to attach to certain symptoms, which, by their presence or absence, or their degree of intensity, will serve to render the prognosis hopeful or the reverse.

Your first duty, on being called to a patient suffering from the symptoms of cerebral hæmorrhage, is to make a diagnosis of the case, which in most instances is not difficult. Immediately afterwards, however, the question will be asked of you, whether the attack is likely to prove fatal or not; and, in case the patient should have recovered from the apoplectic condition which in general accompanies cerebral hæmorrhage, whether he is likely to recover the faculties of speech, motion,

and sensation, which may remain lost after the immediate danger to life has ceased, or whether he will remain in the condition of

"Paralysis, grim foe to men of mind,
That lies in ambush for them treacherously,
And lays them prostrate down, as with a mace."

The quantity of blood which may escape from one or several miliary aneurisms varies from a few drops to four and even six ounces; and the less blood is effused the better will be, *ceteris paribus*, the patient's prospects. Where the effusion is very small, the only symptom may be numbness, and some awkwardness in using the hand, and in walking; but, in the large majority of cases, so much blood is poured out into the cerebral substance as to cause the condition of apoplexy.

Cerebral hæmorrhage never kills with the same rapidity as some forms of heart-disease; and what French authors call "*apoplexie foudroyante*" does not really deserve to be compared with the rapidity of a lightning-stroke, for even in those cases which are most rapidly fatal, viz., where the bleeding takes place from the rupture of an ordinary aneurism of one of the basilar arteries, the least interval between the commencement of the symptoms and the fatal issue has been seven minutes, and it will commonly take from fifteen to thirty minutes. Compare with this the case of my late colleague at this institution, Dr. Thompson Dickson, who suffered from disease of the aortic valves, and who one day, while he was driving with his wife and talking to her, suddenly leant forward, and was found to be dead. Syncope, indeed, may kill as quickly as lightning, but you will see that the term "*apoplexie foudroyante*" is a misnomer.

In the large majority of fatal cases, from six to twelve hours elapse between the beginning of the illness and its end; and that this should be so is easily accounted for by the circumstance that the size of the miliary aneurisms is rarely above that of a pin's head, and that it is, therefore, physically impossible that a quantity of blood sufficient to kill should escape from them in a very short time. It is well known that the brain is able to bear the presence of a small quantity of blood in some of its parts, without resenting it violently; and the anæmia of the organ, which is caused by the hæmorrhage, must have reached a considerable degree before it proves to be incompatible with life.

A most important element in the prognosis of apoplexy is *time*; and although a number of cases only prove fatal on the fourth or fifth day after the commencement of the symptoms, either from collapse or from cerebral fever, yet it is the fact that, the longer the attack lasts without death taking place, the more hope there is for recovery, as far as life is concerned.

In cases which are to end fatally early, that is, within twelve hours from the commencement, you will be guided in your prognosis by the high degree of intensity of all the symptoms of apoplexy. Amongst these, I would direct attention, foremost of all, to those signs which are given by *thermometric observation*; and you should never be without your clinical thermometer at the bedside of such a patient. Quite in the commencement of the attack, and particularly where the hæmorrhage is not abundant, the temperature remains normal; but, after ten or fifteen minutes, the thermometer in the rectum or the arm-pit shows signs of falling. Respiration may still be quite regular, and the pulse may beat quietly at the rate of 70 or 76, yet you will see the mercury receding to 97 deg., 96 deg., and even 95 deg. Where this fall takes place rapidly, and reaches the lowest degree just mentioned, the prognosis is bad; while, if the fall is only slight, say one degree or one and a half, and is protracted in its production, the prognosis is generally favourable.

What is the cause of this considerable fall of temperature? Bournerville and Charcot, who were the first to point out these changes, are quite silent on this point. I believe that we must look upon this fall as a symptom of irritation or spasm of the controlling centres of heat-production; and that it has the same clinical significance as convulsions and rigidity of the muscles of the extremities, and conjoint lateral deviation of the head and eyes, which are also prominent and significant symptoms of the apoplectic seizure. Where there are severe convulsive attacks, lasting for many minutes, and where the head and eyes are persistently turned away from the paralysed side, you may conclude that the hæmorrhage is progressing more or less rapidly, and that the blood is irritating and undermining the central ganglia, previously to its destroying their texture.

During this period of the attack, you have, therefore, partly symptoms of spasm and partly of paralysis—spasm where there is irritation, paralysis where there is compression or destruction of nervous matter. Consciousness and speech are at first impaired, but, after a time, lost; the energy of the special senses, and of common sensation, is diminished and gradually vanishes. A strong light, loud sounds, powerfully smelling or sapid substances, no longer convey any impressions to the anæmic brain, and the patient cannot be roused by any of the usual

odes of reviving consciousness. The face is distorted, and its expression utterly vacant; the pupil dilates; reflex action appears abolished, and the urine and feces are voided involuntarily. The sphincter ani does no longer offer any resistance to the introduction of a finger or an instrument. The automatic movements of circulation and respiration, which at first continued much in the same manner as they do during a heavy sleep, become affected in their turn. Inspiration is short, superficial, and irregular; the soft palate is heard to flap to and fro, producing stertor; and, from having been accelerated, respiration becomes stertored and intermittent. Mucus accumulates in the air-passages, and laryngeal, tracheal, and bronchial *râles* are heard. A frothy liquid, which is a mixture of saliva, buccal mucus, and air, is seen to run down the chin. The pulse is large, hard, and incompressible, more especially in the carotids—not from congestion, as has been erroneously supposed, but from the resistance encountered by the current of the blood, which cannot enter the intracranial blood-vessels, compressed as they are by the effusion.

Patients have occasionally recovered after presenting all, or nearly all, of the severe symptoms just mentioned; and none of them are, therefore, of the same prognostic importance as a great fall of temperature.

After a time, which varies from thirty minutes to thirty-six hours, the spasm in the centre ceases, either from the hæmorrhage being arrested, or from the blood breaking through into the lateral ventricles and into the fourth. The symptoms then become purely paralytic in their character; the lateral deviation of the head and eyes disappears; convulsions and rigidity cease; and the body-heat begins to rise more or less rapidly. Where this rise is extensive, it has the same unfavourable meaning as the fall in the commencement; and the mercury, now at 103 deg., 104 deg., or even 105 deg., corresponds in prognostic significance to the low readings of the first period. More especially where the temperature runs up rapidly, it is a sure sign of collapse, which is soon followed by death.

On the other hand, we find cases in which the thermometer, after a comparatively slight fall, rises only little, if at all, above the normal mean, but remains stationary at 98 deg. or 99 deg. This means that the hæmorrhage has been arrested; the opening in the miliary aneurism having been, as it were, sealed up by contraction of the clot, before the blood could have broken into the lateral ventricles. There is, therefore, now only a comparatively small cavity filled with blood, which may be expected, in course of time, to undergo its appointed changes. There is no lateral deviation of the head and eyes, no rigidity or convulsion of the limbs; the body does not appear so completely relaxed as before; the difference between the sound and the paralysed side—if there be paralysis—becomes more marked, one being quite relaxed, while the other offers some resistance on being moved, or carries out semi-voluntary movements; the patient begins to talk again in a dreamy fashion, and the coma gradually lightens into consciousness. The pulse becomes steadier, and respiration more regular, although the contraction of that side of the diaphragm which corresponds to the paralysed side is more feeble than that of the other side.

The prognosis in this stage, which may last for two or three days, still remains doubtful; for although the patient has escaped death from collapse, he is still liable to be carried off by the consecutive cerebral fever, which, in cases that are to end fatally, is apt to come on after the partial recovery just described has taken place. You have to be particularly careful, at this period of the malady, not to give too favourable an opinion about the prospects of the patient, for blame may be attached to you if you pronounce the patient out of immediate danger, and he die the next day of cerebral fever.

At this time, when the patient's fate hangs, as it were, in the balance, nothing will assist you more in forming a correct view of his condition than an *inspection of the nates*; for the first symptom of cerebral fever is almost invariably acute bed-sore in the buttock of the paralysed side.

An erythematous spot, or macula, appearing on the second, third, or fourth day on the part just mentioned, almost invariably heralds a fatal termination of the case; for this change in the nutrition of the skin and subjacent parts shows paralysis of the trophic intracranial centres, which precedes paralysis of the volitional centre only by a short time. The pathology of the process is the same as that of ulceration of the cornea after section of the fifth nerve. The cerebral macula has an irregular shape and varies in size; from being pink at first, it gradually changes into purple. Pressure will cause it to disappear, showing that here is only hyperæmia in the beginning. Probably, at your next examination of the parts, you will notice a great change; for rapidity of progress, a superacute course, is characteristic of this affection. The hyperæmia is succeeded by effusion of serum, and phlyctenæ are formed, which contain a liquid that is at first colourless, but shortly becomes sanguinolent and livid. The raised cuticle then gives way,

and an open sore is left, which has a scarlet surface, and appears covered with livid granulations. Gangrene is now fully established, and, if the patient survive long enough, inflammation sets in, by which the gangrenous parts are to be eliminated.

This acute bed-sore is quite different from that which occurs habitually, in consequence of prolonged pressure on the back and other parts, in the course of protracted disease, where the patients are bedridden, and where there is frequent involuntary evacuation of the urine and feces, which aids in the production of gangrene. Charcot, who has studied this condition more particularly, has found that it cannot be prevented by turning the patient over to the non-paralysed side, so as to avoid pressure on the buttock of the paralysed side; nor by frequent catheterism, whereby any contact of urine with the surface is avoided. Moreover, the bed-sore which occurs in protracted cases of spinal and other diseases, affects more the sacrum than the buttock, which constitutes another difference between the two.

In some cases, appearances similar to those just described are observed on the heel, ankle, and knee of the paralysed leg; and where the effusion has burrowed its way into the lateral ventricles, phlyctenæ may appear on both nates.

A rise in the temperature may be expected with certainty after the cerebral macula has begun to form; and the mercury will, in a comparatively short time, run up to 103 deg., 104 deg., and even 105 deg. At the same time, other symptoms manifest themselves which point to the impending dissolution. The patient throws himself about in a restless manner, and a low muttering delirium sets in, which is occasionally broken by loud moanings. The pulse becomes small and compressible, and runs up to 120 or more beats. Respiration is hurried and superficial, at the rate of 40 to 60 in the minute; and the abdomen is drawn in during inspiration, showing commencing paralysis of the phrenic nerve. The extremities become cyanotic, and completely relaxed. The face, and sometimes the whole body, is bathed in clammy perspiration; the neck is flabby, and the head will retain any position in which it is placed. Pulmonary complications sometimes set in a short time before death, and the temperature occasionally reaches its maximum shortly after the fatal issue.

Cerebral fever rarely occurs later than the fourth day of the attack; and if the patient, therefore, survive the fifth or sixth day without there being phlyctenæ and undue elevation of temperature, the prognosis as to the continuance of life becomes favourable. Nevertheless, you must impress the fact upon the patient's friends, that the condition of his arterial system renders further attacks probable; and it will depend upon the general features of the case, taken collectively, and your treatment of the case afterwards, whether such attacks are likely to come on sooner or later.

Amongst the various points which influence the issue of such attacks as just described, the *age* of the patient is a most important one. Clinical experience has shown that the young recover more easily from the complaint than the old; and the results of my researches on the mortality from this disease in England and Wales during the last forty years, enable us to give considerable precision to this point. A large number of infants die of apoplexy in the first year of age; but these are mostly cases of meningeal, and not of cerebral hæmorrhage. Of the latter, there are hardly any instances between the first and fifteenth year of life; after fifteen, they are "few and far between"; but at thirty-five there is a perceptible increase, and the numbers then gradually swell, until they reach an immense maximum between seventy and seventy-five years of age. Between seventy-five and eighty, the mortality from this complaint is still very large, while, after eighty, a rapid fall sets in; but, considering how few people are still alive at eighty, and the subsequent periods of life, the fatality of cerebral hæmorrhage does actually increase rather than diminish as age advances. I am, therefore, able to state in general terms that cerebral hæmorrhage is of slight significance up to thirty years of age; that its fatality increases *pari passu* with years; and that *the greater the age, the less is the probability of recovery from cerebral hæmorrhage*.

While, therefore, age must, in every individual case of this kind which may come under your observation, largely influence your opinion about the patient's prospects, you should know that *sex* has no such influence at all. It is true, that it has hitherto been generally assumed that males are more liable to die of apoplexy than females; but my investigations of this point have conclusively shown that such is not the case; that the sexes die in almost equal proportions of the disease; and that the slight excess which is found to exist is for women and not for men, the proportion in two hundred thousand consecutive cases being 1,000 for males to 1,009 for females. From this you will perceive that, for the purpose of prognosis, sex is devoid of practical importance.

The *constitutional condition* of the patient has, on the contrary, a

most important bearing on prognosis. Where cerebral hæmorrhage occurs from leukaemia or contracted granular kidney, the prognosis is unfavourable. Gout and syphilis are likewise undesirable complications, while the absence of constitutional faults will, *ceteris paribus*, render the patient's prospects more hopeful.

Finally, let me tell you that *treatment* may incline the balance towards recovery or death. The treatment by venesection, which was formerly much in favour, was thoroughly irrational, and generally followed by disastrous results; indeed, many patients have died of the remedy rather than of the disease. Venesection has lately fallen into disuse; but as it is still recommended in some text-books for this condition, I wish to impress upon you most strongly the fact, that the condition of the brain during cerebral hæmorrhage is not one of congestion, as was formerly believed, but of anæmia; that the organ not only loses blood largely, but is also, from compression of its arterioles through the clot, unable to receive a fresh supply of the reviving fluid; that death in this disease takes place chiefly from anæmia; and that, by resorting to phlebotomy, you simply increase cerebral anæmia still further, and thereby hasten the fatal result. *Eschew the lancet, therefore, as a deadly instrument in these cases.*

A simply expectant plan of treatment is recommended by the most recent writers on the disease; and there can be no doubt that abstaining from all active interference is far better than to bleed your patient. Molière, on his death-bed, cried out to his doctors: "*Laissez-moi mourir, mais ne me tuez pas!*" and the expectant plan of treatment certainly does not kill the patient, it only allows him to die. In spite, however, of recent authorities for doing nothing, a more active mode of treating cerebral hæmorrhage seems to me to be called for.

Your object must be to arrest the further effusion of blood from the ruptured coats of the milary aneurisms, by causing the vessels to contract. Now, many styptics must be inapplicable for these cases, because the patient cannot swallow, and even if medicines were introduced into his stomach, it seems most doubtful whether they would be absorbed. Nor can the rectum be used for the purpose of affecting the circulation, as there is frequently paralysis of the sphincter ani, and inability of the bowel to retain its contents. The hypodermic mode of administering medicines seems, therefore, to recommend itself, particularly in these cases; and the remedy I think most appropriate for them is ergotine.

There are two kinds of ergotine known to chemists, viz., Wiggers's and Bonjean's. The former is insoluble in water, ether, and dilute acids, but soluble in alcohol, strong acetic acid, and caustic potash; and, on account of these peculiarities, it is not suitable for subcutaneous injection. Bonjean's ergotine, on the other hand, is easily soluble in water, and it is this therefore which you should use. I am in the habit of injecting a grain of it every hour, or, where the symptoms are very urgent, even every half hour, into the subcutaneous cellular tissue; and although the experience of a single observer, in a disease like the one now under consideration, cannot count for much, yet I feel justified in recommending you to follow this practice, as being likely to save many lives.

The popular notion that *it is the third stroke which kills*, is quite fallacious, for, in a large number of cases, the first proves fatal; nevertheless, it is a fact that many patients succumb to a third attack. Others survive a third, and even further strokes, although in so enfeebled a state that they are left, as it were, only shadows of their former selves. There is gradual but plainly perceptible decay of the mental and physical faculties, owing to consecutive atrophy of the brain, and more especially of its cortical substance. Senile marasmus thus becomes established, and as there is much less resistance to injurious external influences than there was previously, a slight incident, such as a cold affecting the bronchial tubes, or intestinal catarrh, will rapidly destroy the patient's life.

It is impossible to answer the question definitively which will be sometimes asked of you, viz., when do you think that the patient will have another attack? A wide margin has always to be left here, and only the ignorant will speak with decision about this point. Where the patient at the time of the attack is still comparatively young and vigorous, he may still enjoy many years of good health afterwards, while no such prospect can be held out to the old and decrepit.

Do patients ever completely recover from an attack of cerebral hæmorrhage?

This question used formerly to be unhesitatingly answered in the affirmative. Even so recent an observer as Durand-Fardel states that, of twenty-seven cases of hemiplegia from hæmorrhage into the corpus striatum, which came under his observation, nine were cured, two nearly so, four remained weak in the affected limbs, one retained difficult articulation, one became imbecile, and ten remained hemiplegic. This proportion of recoveries, however, does not really occur; and if we were to accept

it without reserve, it would imply a far greater vitality of the previous generation, or more effective modes of treatment employed by our predecessors. You will be nearer the truth in assuming that our search for symptoms of disease is now keener than previously, and that we consequently discover them more frequently. Having had more than four hundred cases of hemiplegia, from hæmorrhage into the corpus striatum under my care in hospital and private practice, I am enabled to state that complete recovery is the exception; and that even in those patients who professed to enjoy good health after a seizure, the memory was, as a rule, less ready, the speech less fluent, the power of application less enduring, and the sense of touch and the co-ordination of movement less quick than before the attack. Indeed, we must consider it highly gratifying if a fair amount of capacity for mental and physical exertion be regained by those who have suffered from the rupture of milary aneurisms in the cerebral substance.

Nor should it be a matter of surprise that such patients do not, as a rule, entirely regain their faculties. The blood which has been effused must, in the nature of things, destroy a number of nerve-cells which can never be regenerated, and whose place will ultimately be taken by connective tissue. The eventual loss of function will be proportional to the extent and importance of the destroyed parts, and to the degree to which their place can be taken by allied structures in the neighbourhood of the lesion. A somewhat analogous case is that of a man who has had a portion of his lung consolidated by tubercular deposit, and who, although the activity of the disease may have been arrested, and retrogressive changes taken place in the tubercle which renders it innocuous to the system, can never be so strong as he might have been with the entire organ in full functional activity, although he may certainly enjoy a measure of health and strength.

The extent to which recovery of function may take place depends—

1. Upon the quantity of blood which has been effused. This varies from a few drops to several ounces; and the less escapes the better will be, *ceteris paribus*, the patient's prospects.

2. Upon the portion of the brain into which the effusion has taken place. Sensation is more easily re-established than motion; and hæmorrhage into the thalamus opticus seems to give better prospects of recovery than when the blood tears up the corpus striatum. Again, hemiplegia affecting the right side of the body is not only more serious in its aspect than that of the left, because it is almost invariably combined with loss or great impairment of speech, which, when lost, is never entirely recovered; but it also appears that patients recover the use of the left more readily than that of the right side of the body; to which must be added the circumstance that the left arm and hand are nearly so essential to the patient as the corresponding limbs of the right side.

3. Upon the manner in which the effused blood is ultimately disposed of in the intracranial centre. You know that where the patient survives the stroke, the hæmorrhage is arrested after a variable space of time; the clot of blood contracts; the serum is absorbed; the solid parts shrink; and the vacuum which is caused by this contraction is filled up by the effusion of a liquid, which is at first homogeneous, but gradually assumes the characters of connective tissue, and ultimately forms a capsule which encloses what remains of the clot. Thus a kind of cyst is formed, which lies imbedded in the cerebral matter, and the size of which varies from that of a pea or cherry-stone to that of a small apple. The manner in which this apoplectic cyst is formed varies considerably, according to the constitutional powers of the patient; and a small clot in an aged person with enfeebled systemic energy, may cause more loss of function than a clot considerably larger in a comparatively young man of otherwise unimpaired vigour. In old decrepit persons there is less contraction of the clot, and more liability to inflammatory irritation in the period of healing, whereby softening in the neighbourhood is induced, with its well-known consequences. In some cases, subacute inflammatory irritation never completely subsides in the neighbourhood of the original lesion, and this is indicated by a high degree of rigidity and contraction of the paralysed muscles, with spasms and general systemic irritability, which is sooner or later followed by exhaustion and collapse.

In favourable cases, the formation of the apoplectic cyst is generally finished in about three months after the attack; and, in accordance with this, we find that the paralysis diminishes gradually up to that time, and remains more or less stationary after that period unless appropriately treated. In other cases, the paralytic symptoms increase, and the period varying from six to twelve months, and this must be looked upon as most unfavourable in a prognostic point of view; for it shows the commencement and progress of that peculiar form of granular disintegration of nervous matter which Türck and Cornil have shown to occur in the parts contiguous to the clot, and which may spread beyond the striated body and the optic chamber to the corresponding half of the

pions and the pyramid, down to the lateral column of the spinal cord. When this condition has been fully developed, no kind of treatment is of any avail; and it appears, therefore, for this reason alone, that your therapeutical efforts should not be postponed a day longer than you can help it. Ergot, in the form of the liquid extract, for the cure of any military aneurisms which may be in the process of formation; phosphorus, for improving the nutrition and function of the injured nerve-cells in the neighbourhood of the lesion; galvanisation and faradisation, for the stimulation of the paralysed parts, are our sheet-anchors in the treatment of this condition; and, if sagaciously used, will do infinitely more good than the merely expectant plan of treatment, which has hitherto been somewhat too much the fashion in these cases.

A FEW PERSONAL FACTS CONCERNING EXPERIMENTS ON ANIMALS OPPOSED TO THE FALSE FANCIES OF THE PERSECUTORS OF VIVISECTION.

By CHARLES J. B. WILLIAMS, M.D., F.R.S.,

Physician Extraordinary to the Queen; Consulting Physician to the Brompton Hospital for Consumption from its foundation; Ten Years Professor of Medicine, University College, London; Late President of the Royal Medical and Chirurgical Society; and First President of the Pathological and New Sydenham Societies.

1. IN February 1835, assisted by several able physicians and surgeons, I conducted an investigation of the causes of the sounds of the heart. Two young donkeys were the subjects of the experiments. They were killed by the insertion of woorara poison (twenty and fifteen grains) into a small wound in the thigh. When they ceased to breathe, and every sign of sensation and voluntary motion was extinct, the chest was cut open, and the heart was found still beating; by the aid of artificial respiration, it continued to beat regularly for upwards of an hour, during which time I made the series of observations on the sounds, and on the means of stopping or altering them, which gave the first complete knowledge of those sounds. This knowledge has ever since supplied the profession with the means of distinguishing the signs of the heart's action in health and in disease. It is difficult to overrate the value of this discovery in the practice of medicine; yet it was obtained at the cost of the lives of only two young animals, with only the suffering of a trifling wound, much less painful than one of the common blows with which these poor brutes are belaboured in their common work. The large dose of woorara destroyed all possibility of suffering; in fact, however doubtful may be the action of small quantities of this poison, it is certain that these animals were dead in all respects except the heart's action, and this was automatically sustained for awhile by artificial respiration.

2. In a series of researches, in which I proved the muscularity of the bronchial tubes and shewed the modes of acting on it in cases of asthma, I made use only of recently dead animals; and the intelligent physiologist knows that there are often residues of life which may be thus profitably questioned by experiment long after the cessation of all feeling. Yet, such cases are ignorantly or mendaciously branded as instances of animal torture.

3. In 1841, I was long engaged in studying the circulation of the blood in the web of the frog's foot; and, among other results, I was enabled to trace the process of determination of blood to enlargement of the arteries on the application of an irritant. Yet, so gentle were the means which I used, and so little hurtful to the feelings of the animal, that I needed no ligature to the toes, but simply spread the web on glass with a wet camel's hair brush; and, by patiently waiting, could best observe the size and current of the vessels during the periods of tranquillity when the animal seemed quite at ease. The nature of the inquiry excluded all strong or painful irritants, and the best results were obtained when there were no signs of uneasiness. These observations have been referred to by Professors Virchow and Burdon Sanderson as the first to demonstrate the true cause of determination of blood in health and in disease, a chief element in the processes of growth and inflammation.

These and other important results of investigation by a private individual, seeking after truth, with full consideration to brute as well as to human suffering, would never have been obtained had that law been in force with which it is now proposed to shackle and insult the members of an enlightened and humane profession.

ON VIVISECTION AND SCIENTIFIC SURGERY.

By SAMPSON GAMGEE, F.R.S. (Edin.),

Surgeon to the Queen's Hospital; President elect of the Birmingham and Midland Counties Branch of the British Medical Association.

THE Parliamentary Committee of the Association has worked so well, in eliciting the opinion of the profession on the Vivisection Bill, and the eminent men who have spoken for us have done so with such weight of authority and discreteness of utterance, as to supply very convincing proof of the power to which the British Medical Association has attained.

Yielding to none of my brother associates for sincerity in acknowledging the obligation imposed on us by those who have fought our battle, I submit that, in the contest for freedom of experimental investigation, the great influence which experiments on living animals have exercised on scientific surgery has been somewhat overlooked.

When Lord Cardwell* put the question "Have the great performers of these painful experiments on animals been generally great surgeons?" Sir William Fergusson is reported to have answered, "No, I am not aware of any great surgeons having been very great experimenters on the lower animals".

Omitting the obvious instance of John Hunter, and his immediate followers in this country, what were Scarpa, Dupuytren, Travers, Syme, Ribès, Jobert, Sédillot, and Porta, unless great surgeons and great experimenters on the lower animals?

In the whole subject of the arrest of hæmorrhage by pressure, ligature, and torsion, in the selection of the best materials for sutures, in the treatment of wounds of the intestines, in the discovery and demonstration of the pathology of pyæmia and allied states of blood-poisoning, in the repair of tissues, experiments on living animals have preceded, and been the foundation of, progress in scientific surgery.

Some proof of this proposition is to be found in the Report of the Royal Commission on Vivisection, but much more evidence might be adduced. While the Hunters in this country were raising surgery towards the dignity of a science, the French school was labouring successfully in the same direction, through the happy coincidence which made Bichat the pupil of Desault, and the editor of his works. It is needless to say how the very brief and brilliant career of Xavier Bichat affected the sciences of anatomy and physiology. If any one doubt how directly his teaching influenced the study of surgery by experiments on living animals, and by clinical observation, let him consult such a work as that of his pupil Ribès' *Mémoires et Observations d'Anatomie, de Physiologie et de Chirurgie* (Paris 1841).

Dupuytren's clinique was one of the greatest schools of practical surgery the world has ever known; not merely pupils in the ordinary sense, but surgeons from all countries crowded round the great teacher in the Hôtel Dieu. Admitting the paramount influence of his intellectual endowments and of his iron will, there is no doubt that Dupuytren's success, as a promoter of surgical science, was largely due to the completeness of his medical education, particularly in anatomy, in chemistry, and in experimental physiology: he worked in Thénard's laboratory, and repeatedly had recourse to experiments on living animals.†

As a pioneer to new truths, as an anatomist and physiologist, as a draughtsman and linguist, as a dissector and a mechanician, as an operator and a clinical teacher, Antonio Scarpa has had few rivals in the history of medicine. What a school he was reared in! A favourite pupil of Morgagni, who, when stricken with blindness, made him his amanuensis, and closed his long and glorious life in his arms, Scarpa, as a pathologist, proved himself a worthy follower in the work *De Sedibus et Causis Morborum*. The pupil in Paris of Vicq-d'Azyr and Frère Côme, in London of Pott, the Hunters, and Cruikshank, the companion of Volta in his visit to the Universities of Germany when Wrisberg, Blumenbach, Gmelin, and Richter professed at Göttingen, Scarpa was educated in the highest school of science, and pre-eminently qualified

* Report of Royal Commission on the Practice of subjecting Live Animals to Experiments for Scientific Purposes. London: 1876. Question 1026, p. 49.

† Eloge du Baron Dupuytren in *Histoire des Membres de l'Académie Royale de Médecine*, par E. Pariset et E. F. Dubois; Paris. 1850, tome second, p. 103, et seq. See also *Parallèle de Delpech et de Dupuytren in Tribut à la Chirurgie*, par E. F. Bouisson; Paris et Montpellier, 1861, tome second, p. 1, et seq. *Propositions sur quelques points d'Anatomie de Physiologie et d'Anatomie Pathologique*, par G. Dupuytren, an. xii, 1803.