tropia; so, no doubt, to carry the illustration further, there are various degrees of congenital ill-development of the highest cerebral centres. Hypermetropia from ordinary use of the eyes becomes manifest at different ages, according to its degree (roughly speaking, one-eighth at eight years of age, one-twentieth at twenty years of age, and so on); so it is possible that insanity becomes manifest at different ages, even from ordinary wear and tear of a civilised life, according to the degree of ill development of the highest cerebral centres.

I should extend the principle to all organs. The inheritance of phthisis is, I submit, an inheritance of small lungs—of lungs easily overworked.

Leaving the consideration of anomalies of refraction, I pass to a very important disease, in which loss of accommodation is a symptom. I suppose it is to ophthalmologists that we are indebted for our knowledge of what so-called diphtherial "amaurosis" really is. It is one of the most singular forms of palsy. Diphtherial palsy altogether seems unlike any other. I believe that diphtherial amaurosis gives us a clue to its nature. Diphtherial amaurosis is that part of diphtherial paralysis which is most easily studied. The state of things can be studied with absolute precision, and therefore I suggest that, in a scientific investigation of the nature of that very complex thing diphtherial paralysis, we should begin here. Whilst urging on physicians the ophthalmological study of diphtherial paralysis, I would particularly urge on ophthalmic surgeons a careful study of the researches of Greenhow into this very singular disease.

In diphtherial amaurosis, the power of accommodation of the eyes is diminished or lost, and thus the patient (supposing him to be emmetropic) cannot read or see small objects, but can see in the distance. Obviously, the term amaurosis is improper; the deep parts of the eyes are normal. So far as I know, paralysis of the ciliary muscles on the two sides is only found in a high degree after diphtheria. After any debilitating illness in a hypermetropic person, we may have a condition which is symptomatically a minor degree of it: the patient's hypermetropia, heretofore latent, becomes manifest.

The peculiarity of diphtherial amaurosis is, that the paralysis is of parts supplied through a ganglion of the sympathetic chain. There is not paralysis nor paresis of the third nerve, but of parts of it which are supplied through the lenticular ganglion. In other regions, we see the same thing. The defective articulation is owing to paralysis of muscles of the palate supplied through Meckel's and the otic ganglia. The very slow pulse which, as Jenner and Greenhow have pointed out, is found in some cases, may be explained by affection of the cervical ganglia of the sympathetic. I grant fully that I cannot show that a similar state of things applies to the rest of diphtherial paralysis. Indeed, we could not reasonably suppose that the pathological process in diphtherial paralysis has any special malice against sympathetic ganglia as such; we can only suppose that cells of certain histological constitution easily give way, and, at the most, that those of these which first give way are most largely represented in the higher ganglia of the sympathetic.

There is one other direction of inquiry. In the case of hearing, there is evidently an accommodative apparatus analogous to that in the eye. Omitting the muscles of the pinna, which in man are rudimentary, there are, inside the tympanum, the tensor tympani and the stapedius muscles; we have to take into account also the tensor palati, because it has to do, if not with what I call the accommodation of the ear, at any rate with the functions of hearing. The otic ganglion, Troeltsch says, is of the same importance to the ear as the lenticular ganglion is to the eye. It is, then, I think, legitimate to inquire if we do not find in diphtherial palsy occasionally affection of hearing analogous to the defect of sight. One could only expect slight defect, because the muscular apparatus of the ear has not so much to do with hearing as the ciliary muscle has with sight. Besides, if the hypothesis be correct, that the otic ganglion in such a case is affected, there would be no palsy of the stapedius muscle; and, more than all, the tensor tympani and the tensor palati would not be entirely paralysed, as they receive a supply directly from the internal pterygoid branch of the fifth nerve, as well as from the otic ganglion. Moreover, the tensor tympani is the accommodator for high sounds; and thus, on the hypothesis, we should only expect loss of power to recognise high sounds. So far, then, from expecting to find "deafness" in diphtherial paralysis, I should expect only slight interference with the power of appreciating high-pitched sounds. I have met with but one case of slight affection of hearing in diphtherial paralysis. A medical man said it was not enough to impair his hearing for ordinary purposes, but, to use his expression, "enough to render music unintelligible". It was just the kind and degree of defect one would expect to find. Slight defects of hearing are not likely to be noted by unskilled persons. It is a remarkable

fact that double hearing has never been noted except in musicians (Roosa), although, of course, it may occur in anybody. It would seem to me that the investigation of the states of hearing in diphtherial paralysis could only be possible in an intelligent and also educated patient, and when the investigator is an aural surgeon. For there is the obvious difficulty, as Greenhow insists, that, if there be deafness, it may be owing to some affection of the Eustachian tube, a relic of the active diphtheritic process. In cases of Menière's disease, and in other ear-affections, the aural surgeon notes the patient's power of perceiving particular notes; and I see no reason why the state of hearing in cases of diphtherial paralysis should not be equally carefully noted.

Before leaving this subject, I would mention that the clearest account of the peculiar difficulty of articulation which occurs in paralysis of the palate is given by Donders in an appendix to his exposition of diphtheritic paralysis of the ciliary muscles; it is not an uncommon thing for paresis of the lips to be suspected when the palate is solely affected.

[To be continued.]

THE LUMLEIAN LECTURES ON THE MUSCULAR ARTERIOLES: THEIR STRUCTURE AND FUNCTION IN HEALTH AND IN CERTAIN MORBID STATES.

Delivered at the Royal College of Physicians of London.

By GEORGE JOHNSON, M.D., F.R.S.,

Fellow of the Royal College of Physicians; Professor of Clinical Medicine and Senior Physician to King's College Hospital; etc.

LECTURE III.—*Concluded.*

Renal Asthma: Symptoms and Proximate Cause.—The Dry and Inactive Skin of Bright's Disease: its Proximate Cause.—Is Diuresis a Result of High Arterial Tension?—Is High Arterial Tension a Cause of Albuminuria?—Uræmic Convulsions and other Nervous Symptoms: their Relation to Epilepsy: their Proximate Cause: Principles of Treatment.—Two Forms of Impaired Vision in Bright's Disease: the Proximate Cause of each.—Cholera Collapse and Pulmonary Embolism compared.—Cholera Collapse and Spasmodic Asthma compared.—Conclusion.

THERE is good reason to believe that some of the more formidable nervous symptoms which result from uræmia—in particular uræmic convulsions and a form of transient amaurosis—are directly due to cerebral anæmia consequent on sudden extreme contraction of the muscular arterioles.

There can, of course, be no question that uræmic convulsions are of an epileptic character. A large amount of evidence points to the conclusion that both the loss of consciousness and the convulsions of epilepsy are the results of sudden and extreme anæmia of the brain. In man, and in most, if not in all, warm-blooded animals, a rapid and very copious hæmorrhage usually causes convulsions. Kussmaul and Tenner state (*On the Nature and Origin of Epileptiform Convulsions caused by Profuse Bleeding*, New Sydenham Society, 1859) that in numerous cases of dogs, cats, and rabbits, they observed, without a single exception, violent and general convulsions preceding death by loss of blood. In order to produce this result, the hæmorrhage must be rapid. If it occur slowly, so that the vital powers are gradually exhausted, death then occurs with swooning, drowsiness, and delirium without convulsions.

The same experimenters found that an interruption of the supply of blood to the head of a rabbit, by ligature or compression of the arteries of the neck, produces epileptic convulsions as surely as hæmorrhage does. In about one hundred rabbits they ligatured or compressed the carotids and subclavians, from which, be it remembered, the vertebral arteries proceed; and in every case, except that of one very old lean and feeble animal, convulsions occurred.

In order to excite convulsions, they found it necessary to close all the four arteries which supply the brain. If but one carotid or one vertebral artery remained pervious, the animal was enfeebled and more or less paralysed, but not convulsed. And again, if, during the height of the convulsion, the ligature were removed from one carotid, the convulsions generally ceased immediately, and there was a sudden change from the most frightful spasm to complete relaxation of the muscles. The description of the convulsions thus artificially produced with, as it

seems to me, needless reiteration, in the lower animals, shows that they were essentially the same as epileptic convulsions in the human subject. There was the dilated pupil, the tonic spasm, quickly followed by clonic convulsion so violent as to throw the animal forward to a distance of one or two feet, and sometimes even over the shoulders of the operator. These experiments obviously could not be performed on the human subject; but Drs. Kussmaul and Tenner approached as near to this as they dared by compressing the carotids of six men. The result was that in all the face turned pale; the pupils first contracted and then dilated; the respiration became slow, deep, and sighing; then there was giddiness, staggering, and unconsciousness, and the men would have fallen if they had not been supported. They say that, "in two subjects of weak intellect and moderately anæmic, in whom, notwithstanding the above symptoms, the compression was continued, a choking sensation, attended by vomiting and general convulsions, came on, which, however, did not attain an aggravated form; for, on withholding the compression, they disappeared in a few seconds". (*Op. cit.*, page 28.) Compressing the carotids does not, of course, entirely cut off, but only greatly lessens the supply of arterial blood to the brain; but these experiments render it probable that sudden occlusion of all the arteries supplying the brain would as certainly excite epileptic convulsions in man as in the lower animals. And this conclusion is confirmed by observing the results of certain diseases and accidents in the human subject. Thus convulsions occur almost invariably as a result of sudden suffocation or acute apnoea. It has commonly been supposed that the convulsions thus occurring are caused by the noxious influence of black blood upon the brain: It is far more probable that they are caused by the sudden and extreme anæmia of the brain, consequent on the impeded flow of blood through the lungs into the systemic heart and arteries, as explained in my first lecture. The epileptiform convulsions which often result from the inhalation of nitrous oxide gas admit of the same explanation. It is quite certain that, in Kussmaul and Tenner's rabbits, with closed carotids and subclavians, no black blood could reach the brain, yet the convulsions were apparently identical with those which result from suddenly fatal apnoea, whether in the lower animals or in the human subject.

A few years since, the following case came under the observation of my friend Dr. Lavies and myself. A gentleman, about sixty years of age, had been confined to his bed for three weeks with symptoms which pointed to great feebleness of the heart, and probably to fatty degeneration of its walls. There was dyspnoea on exertion, and sometimes on awaking after a long sleep; the heart's impulse and the radial pulse were feeble; there was some oedema of the legs, and over the bases of the lungs there were moist crepitating sounds, probably the result of oedema there. He awoke in the middle of one night, told the nurse that he felt quite comfortable, asked the time, and began to repeat her reply "Oh, half-past —", when he suddenly stopped, and the nurse, turning to him immediately, saw that his face was livid and he was in strong convulsions. In a few seconds, and before any one could answer her call for assistance, the patient was dead. The body was examined, in the presence of Dr. Lavies and myself, by my friend and former colleague Dr. Kelly. The walls of the heart were thin, soft, and fat. The right ventricle was dilated, and contained firm decolorised thrombus, extending from the apex of the ventricle through the tricuspid orifice into the auricle, to the outer wall of which it had evidently been attached and moulded, but, becoming separated from the auricular wall, it had fallen over the tricuspid orifice and completely closed it. Thus, the circulation must have been completely and instantaneously arrested. The result was lividity of the face from venous fulness, and epileptiform convulsions from cerebral arterial anæmia. In this case, as in the case of the rabbits with ligatured arteries, it is evident that the convulsions were caused, not by black blood, but simply by the absence of circulating blood in the cerebral vessels.

When animals are killed by air being forcibly blown into a vein, the breathing becomes hurried, the animal suddenly falls down, and usually dies in convulsions; the contents of the bladder and rectum being frequently expelled at the time of death. Dr. John Reid states that, "in very few cases only is death from this cause not preceded by convulsions". (*Physiological, Anatomical, and Pathological Researches.*)

The immediate cause of death in these cases is the arrest of the frothy mixture of air and blood by the contraction of the pulmonary arterioles, the air seldom reaching the left side of the heart; and as a result of this arrest there is, of course, sudden extreme anæmia of the brain, and of every other organ supplied by the systemic arteries. In man, it appears that death from the accidental admission of atmospheric air into a vein during an operation, is less frequently preceded by convulsions. Probably the chief reason of the less frequent occurrence of convulsions from this cause in the human subject is, that the amount of air

accidentally admitted is less, and death consequently is less rapid than when air is forcibly blown into the vein of an animal. It would probably be found, on a careful inquiry, that the occurrence of convulsions in these cases depends upon the circulation being suddenly and completely arrested.

It has been noted, in some cases of suddenly fatal pulmonary embolism, that death has been preceded by convulsions; and Virchow observed, amongst the results of artificial embolism of the pulmonary artery in animals, convulsions and dilatation of the pupil. (*Des Embolies Pulmonaires*, par B. Ball, page 129.)

We find, then, a large amount of evidence pointing to the conclusion that sudden and extreme anæmia of the brain will cause epileptiform convulsions, and a theory of epilepsy has been framed in accordance with these facts; the theory being that the cerebral anæmia, which is the immediate cause of the convulsion, is the result of spasm of the cerebral arterioles. It may be said with truth that this is only one step towards an explanation of the phenomena, and that the cause of the arterial spasm remains to be determined. We will presently revert to this question.

It is, I think, pretty generally admitted that this theory of cerebral anæmia from arterial spasm is quite consistent with the phenomena of epilepsy. It is a matter of general observation that, at the very commencement of an epileptic fit, the face is pallid. There is obviously anæmia of the superficial vessels, and with this there is probably associated anæmia of the intracranial vessels which supply the brain itself. The pallor is in most cases soon succeeded by lividity, owing to the venous engorgement which results from impeded respiration and pulmonary circulation. It is very remarkable that, while the face is pallid, the heart is beating strongly and the carotids throbbing violently. These phenomena would be explained by extreme contraction of the muscular arterioles, resisting the escape of blood from the arterial trunks into the capillaries.

Kussmaul and Tenner endeavoured to support the theory of arterial spasm by experiment, and to some extent they succeeded. In each of two white rabbits, they ligatured the two subclavians and one carotid; the cervical sympathetic, on the other side, was then exposed and galvanised, with a view to excite contraction of the arterioles by the stimulus conveyed through the vaso-motor nerves. In two animals, no effect was produced; but in the third, the background of the eye became completely pale; the pupil dilated, so that the iris could scarcely be seen; the neck was drawn back, and violent convulsions occurred. The electrodes being removed, the spasms ceased, the pupil contracted, and the background of the eye became red; but the animal continued in a swooning condition. After some minutes, electricity applied to the sympathetic nerve produced the same effect as at first. A third attempt to excite convulsion did not succeed.

The authors suggest that these experiments deserve repetition, with a view of rendering certain what at present is probable, namely, "that epileptic convulsions can be brought about by contraction of the blood-vessels induced by the vaso-motor nerves".

According to this theory, then, epilepsy is the result of sudden anæmia of the brain; and this anæmia, when not caused by a sudden and profuse hæmorrhage, or by some impediment to the circulation outside the cranium, is due to an extreme contraction of the muscular arterioles. This arterial contraction may be determined by two main classes of causes:

1. By a purely nervous reflex influence, such as, for example, may be excited by anger or terror, by the irritation of the gums during dentition, by a calculus in the kidney, the ureter, or the gall duct, or by worms in the intestines.

2. In the second class of cases, a blood-poison is the exciting cause of the arterial spasm and the resulting epileptic convulsion. This includes all cases in which convulsions result from retained excreta, of which uræmic convulsions are a typical example.

From the preceding narrative of facts, it appears to be highly probable that uræmic convulsions are directly due to a sudden and extreme anæmia of the brain, resulting from contraction of the cerebral arterioles, and that the arterial contraction is excited by the influence of impure blood upon the vaso-motor nerves and centre.

This theory, moreover, indicates two modes in which uræmic convulsions may be prevented, namely: first, by means directed towards removing the morbid quality of the blood; and, second, by remedies which lessen the reflex excitability of the nervous centre.

Time would not permit, even if it were desirable to enter into the details of treatment; but I am anxious to direct attention to one or two points of practice. It is a well-known fact that the inhalation of chloroform or ether-vapour invariably puts a stop to uræmic convulsions, and often wards off an attack after premonitory symptoms, such as convulsive twitchings of certain muscles, have occurred. It has

sometimes been supposed that the anæsthetic acts by relaxing the cerebral arteries; but an observation of Kussmaul and Tenner points to a different explanation. These experimenters found that, if animals are etherised, no convulsions occur when they are bled to death or when their intracranial circulation is arrested by ligatures. It appears, therefore, that the anæsthetic vapours prevent or stop convulsions by lessening the reflex excitability of the nervous centre.

The undoubted influence of repeated full doses of bromide of potassium, in warding off uræmic convulsions, is also probably to be explained by its soothing sedative influence on the nervous centres. The bromide is a very useful remedy for the painful muscular cramps which are of common occurrence in the advanced stages of all forms of renal degeneration. These cramps, which are especially frequent and severe in the lower extremities and during the night, are no doubt to be classed with the results of uræmic poisoning, and in not a few cases they are the precursors of more formidable nervous disorder. They may, in some cases, be entirely prevented by a draught containing twenty grains of bromide of potassium, with five grains of carbonate of ammonia, at bed-time.

No doubt, some of the many nervous disorders that result from uræmic poisoning are due to the noxious influence of the morbidly altered blood upon the nervous tissue, while others are more probably explained by sudden partial interruption of the blood-supply to certain parts of the nervous system. This statement may be illustrated by a reference to the two forms of impaired vision, which are very frequently associated with advanced renal degeneration. In one class of cases, dimness of sight comes on more or less gradually, affecting one or both eyes, and is permanent. This form of impaired vision is found to be associated with peculiar structural changes in the retina, results of the so-called *albuminuric retinitis*. In the other class of cases, the impairment of vision may be so sudden in its onset that, in a few minutes or even seconds, there is complete blindness, which usually passes away as suddenly as it came. These attacks of sudden and transient blindness may recur again and again. That they are closely allied to epileptiform attacks, is shown by the fact that they are sometimes immediately followed by general convulsions. The most probable explanation of this sudden transient form of amaurosis is that which attributes it to sudden anæmia of the retina, or of the central origin of the optic nerves, the result of arterial contraction, excited by the morbid quality of the blood. It is, in fact, a form of circumscribed partial epilepsy, "epilepsy of the retina", as it is sometimes designated.

There are various forms of nervous disorder of uræmic origin which probably admit of a similar explanation: sudden and transient impairment of motor power or irregular spasmodic movements limited to a particular set of muscles; various disordered sensations in limited portions of the skin; sudden perversions of taste, or smell, or sight, or hearing, sudden impairment of speech, vertigo, confusion of thought, temporary mental excitement and delirium. One or more of these symptoms may occur singly or variously associated in different cases, the onset and the departure being often equally sudden. In explanation of these phenomena, Dr. Hughlings Jackson has, with much ingenuity, suggested that they may result from a sudden temporary interruption of the blood-current through one or more branches of the cerebral arteries by spasm of their muscular walls; so that the brain-tissue within a circumscribed "arterial region", having its nutritive supply arrested or limited, would suffer a suspension or impairment of its proper functions. Our increasing experience of the various forms of nervous disorder which may result from so purely mechanical a cause as embolism of cerebral vessels lends support to this theory. An arrest of the circulation through a portion of the brain involves immediate suspension of function in that part, with perhaps a disorderly action in subordinate or correlated parts. Thus, amongst other symptoms of nervous disorder, maniacal delirium, with mental illusions, and acute chorea have been found associated with, and probably have been directly caused by, mechanical plugging of minute cerebral vessels; the plugging being a result of embolic particles of fibrin detached from the so-called warty vegetations on a damaged cardiac valve. In like manner, sudden and complete blindness may result from embolism of the *arteria centralis retinæ*, partial and patchy blindness from embolism in one of its branches. The results of the mechanical plugging of vessels are thus seen to bear so strong a resemblance to those which are due to uræmic poisoning as to afford much support to the theory of arterial contraction as the immediate cause of some at least of the characteristic symptoms.

There is another class of cases in which the theory of obstructed circulation being the result of arterial spasm receives confirmation from the fact that very similar phenomena result from a demonstrable mechanical block of the same system of vessels. I refer now to the striking

resemblance between the symptoms of cholera-collapse and the results of embolism or thrombosis in the pulmonary artery.

It will be known to most of those whom I have now the honour to address, that for a number of years I have maintained that the impeded circulation through the lungs, which obviously exists during the collapse stage of cholera, is explicable only on the hypothesis of abnormally energetic contraction of the pulmonary arterioles. And I now desire to direct attention to the very striking resemblance between the symptoms of choleraic collapse and those which have been observed in some cases of obstruction of the pulmonary artery by fibrinous clots.

I have references to several cases of pulmonary embolism in which the symptoms bore a more or less striking resemblance to those of the collapse of cholera; but the most complete record of such a case is one which was published by Dr. Alfred Carpenter (*Lancet*, September 23rd, 1871). In that case, as Dr. Carpenter remarks, "the only symptoms wanting to make it apparently one of cholera were alvine discharges and cramps of the limbs". The symptoms actually noted, and which in the choleraic cases have very commonly been supposed to result from the dehydration of the blood by the intestinal discharges, were the following: blueness of the surface; icy coldness of the uncovered parts of the body; cold clammy perspiration; coldness of the breath; sinking of the eyes; feebleness of the voice; a feeble thready pulse; with quick breathing, excessive thirst, and almost complete suppression of urine, two ounces of urine only having been passed one day, and on another day less than two ounces. After death, the right side of the heart was found fully distended with dark-coloured blood, while the left side was empty. The pulmonary artery at its origin was partially obstructed by a clot of fibrinous matter, which sent branches into the ramifications of the artery for several inches; these did not block up the passages entirely, but floated like semi-cylinders in the current. It is obvious that, if the trunk of the artery and its main branches had been completely obstructed, death must have been as instantaneous as in the case of cardiac thrombosis which I mentioned in the early part of this lecture; and it can scarcely be denied that the symptoms which resulted from this partial obstruction of the arterial trunk bear a striking resemblance to those of choleraic collapse. Such a case, therefore, may fairly be cited as evidence in support of the theory of arterial contraction being the main cause of the impeded pulmonary circulation during the collapse stage of cholera.

Again, it is not without interest to remark upon the very striking resemblance between the symptoms of choleraic collapse and a severe fit of spasmodic asthma. For the purpose of illustrating this, I will take Dr. Hyde Salter's graphic description of the asthmatic paroxysm. He says: "If the bronchial spasm is protracted and intense, the heat of the body falls; the oxygenation of the blood is so imperfectly performed, from the sparing supply of air, that it is inadequate to the maintenance of the normal temperature; the extremities especially get cold and blue and shrink. I have known the whole body deathly cold and resist all efforts to warm it for several hours. But, while the temperature is thus depressed, the perspiration produced by the violent respiratory efforts may be profuse, so that the sufferer is at the same time cold and sweating. It is this union of coldness and sweat, combined with the dusky and pallor of the skin, that gives to the asthmatic so much the appearance of a dying man. The pulse during severe asthma is always small, and small in proportion to the intensity of the dyspnoea; it is so feeble sometimes that it can hardly be felt." I scarcely need insist upon the many points of resemblance between these symptoms and those of cholera. What, then, is there in common between these two forms of collapse? Obviously not a drain of fluid from the blood, which was at one time looked upon almost universally as the main cause of choleraic collapse; not, I repeat, a drain of fluid from the blood, but a partial arrest of the pulmonary circulation. In both classes of cases, there is evidence of an impeded pulmonary circulation, the result of spasm of the muscular arterioles. In cholera, the arterial contraction is a primary result of the irritant action of the poisoned blood upon the vessels and the vaso-motor nerves; while in asthma the arterial spasm is a secondary result of a partial apnoea occasioned by primary spasm of the bronchi. Using the words asphyxia and apnoea in their strictly literal sense to express pulselessness and breathlessness, we may say that in cholera-collapse there is a primary asphyxia, and a secondary apnoea consequent on the blood-stasis in the arterioles before it can reach the capillaries to be aerated. On the other hand, in asthma there is a primary apnoea caused by bronchial spasm which cuts off the air from the pulmonary vesicles, and a secondary asphyxia the result of arterial contraction.

In conclusion, Sir, I venture to express a hope that the brief survey which we have taken of some of the pathological phenomena with

which the muscular arterioles and the vaso-motor nervous system are intimately and obviously concerned has not been without interest even for this learned audience, to whom I desire to offer my cordial thanks for the attentive hearing with which they have favoured me.

ON DR. BILLING'S VALVULAR EXPLANATION OF THE HEART-SOUNDS.

By W. J. LITTLE, M.D.,
Late Senior Physician to the London Hospital, etc.

AMONGST the controversial communications on the causes of the sounds of the heart which have recently appeared in the columns of the BRITISH MEDICAL JOURNAL is one dated September 30th, 1876, from Dr. C. J. B. Williams, in which the following statement occurs:—"I reassert that those observations" (viz., Dr. Williams's experiments on the ass stupefied by woorara) "made in February 1835, supplied the first complete knowledge of the sounds of the heart, which has given to the profession the means of distinguishing the physical signs in health and disease." In the same article, it is said: "My experiments established the view that the *first** sound of the heart is produced by the tightening of the ventricular walls and valves in the systole, and the *second* sound by the tightening of the arterial valves by the recoil on them of the column of blood in the arteries by the diastole"; also: "These observations seem to me sufficiently to prove that the essential cause of the first sound is in the walls of the ventricles." He further adds: "I admit that the auricular valves have their full share with the ventricular walls in producing the systolic sound, and I ascribe the *flapping* commencement of this sound especially to them."

Dr. Williams here appears to lay claim to being the first to demonstrate that the first sound is *in part* caused by the action of the auriculo-ventricular valves, and that the second sound is *wholly* caused by the action of the arterial valves. There is some difficulty in reconciling the apparent meaning of the sentences which I have quoted, when they are compared with each other and with what Dr. Williams wrote in 1835. At that period, he assigned more importance to *bruit musculaire* than he now does, as shown by the following extracts from his work on *Diseases of the Chest*, 3rd edition, 1835, page 175:—"That the first sound is not dependent on the closing of the auriculo-ventricular valves (as imagined by M. Rouanet and others) is evident from Exp. 1, Obs. 4, 6, 7, 8, 9, in which the closure of these valves was partially or completely prevented, yet the first sound still continued.....That the first sound is produced by the muscular contraction itself† may be considered as proved by Obs. 8 and 9 of Exp. 1, in which every other possible source of sound was excluded and the first sound still accompanied the systolic action of the ventricles." There appears also some confusion of language in one of the above extracts, where Dr. Williams speaks of tightening of the valves, and in another place of the *flapping* sound of the valves. Surely Dr. Williams would consider himself somewhat at sea, if he imagined the valves to flap in the blood in the manner that the sail of a ship which is loose at one side flaps about during a gust of wind.

I have waited until the probably approaching end of the controversy, in the hope that some of the contributors to it, and especially Dr. Williams, would have alluded to the labours of Dr. Billing, who, before the date of Dr. Williams's experiments, showed, as the result of anatomical, physiological, acoustic, and clinical research, that the first sound of the heart is produced by *tension* of the auriculo-ventricular valves, and the second sound by the *tension* of the arterial valves. Dr. Williams's experiments were made known in 1835. Dr. Billing's views and explanations were given in his lectures on the *Theory and Practice of Medicine* at the London Hospital during the period of my attendance on those lectures, and whilst I was acting as his clinical clerk in the wards previously to August 1831. I was also present as a visitor at the Hunterian Society in February 1832, when Dr. Billing read a paper on the Valvular Origin of the Sounds of the Heart. This paper was published *in extenso* in the *Lancet* of May 1832. I well remember seeing and hearing Dr. Billing illustrate the mode of production of the heart-sounds by means of a strip of paper held by one hand at each end of it and then suddenly rendering it tense. I refer those readers who desire to investigate the matter to Dr. Billing's *First Principles of Medicine*, 6th edition, 1868, page 37.

Whatever honour may spring from the discovery of the valvular origin of the sounds of the healthy heart—and, in my humble opinion,

this honour is considerable—is due to Dr. Billing, my old teacher, colleague, and friend. I hold that clinical study is a firmer basis of knowledge on this subject than experiments on dying animals, especially when, in addition to removal of the anterior wall of the chest and listening through the stethoscope applied to the naked heart, the operator varies his investigation by "pushing the auricles by the end of a finger into each auriculo-ventricular opening, cutting open the auricle, partially destroying the mitral valve, causing the blood to well out at each pulsation, inserting a hook into the pulmonary artery and drawing back its valves". It is not hereby meant to indicate that any of these methods of inquiry were meaningless or unjustifiable; quite the contrary. Having formerly had some experience in vivisections, knowing the difficulty of carrying them out under circumstances in any way analogous to the normal condition of things, I set a smaller value on the results of Dr. Williams's experiments, confirmatory in a great degree as they are of Dr. Billing's previously published views, than Dr. Williams very naturally places upon them. I express the conviction that Dr. Billing is alone entitled to the merit of the discovery of the sounds of the heart being dependent upon valvular tension.

It is an agreeable thing for the old pupils of Dr. Billing to know that many able later investigators have wholly or partially confirmed his views. Of these may be named Rouanet (September 1832), Bryan (January 1833), Von Kiwisch, Valentin (1850), and Halford (1852). It would seem, from the present controversy in the BRITISH MEDICAL JOURNAL, that the minds of many men are still unsettled as to the presumed existence of coexisting simultaneous causes of sound going on in the heart—movements of fluids, *bruit musculaire*, contact of heart with the walls of the chest, etc. I avoid wearying my readers, and, therefore, refer them to the full sources of information contained in Dr. Billing's book already quoted.

It was a grand merit on the part of Dr. Billing, when so many able men—Hope, Latham, Watson, Clark, Williams, and others, his contemporaries in this country—were, like himself, assiduously employing auscultation, to have alone enunciated and demonstrated the valvular origin of the sounds in 1831, at so early a period after the publication of Laennec's immortal discovery of *Auscultation and Percussion*, and at a period when the facts and explanations of Laennec were undisputed as to this matter by men so gifted as Sir John Forbes, the translator of Laennec's work, and Dr. Thomas Davies, the earliest followers, I believe, of Laennec in this country.

THE RECENT CATASTROPHE AT TYNEWYDD COLLIERY, NEAR PONT-Y-PRIDD.

By H. N. DAVIES, L.R.C.P.L. & ED., Surgeon to the Colliery.

THE subjoined narrative of the recent accident at Tynewydd Colliery is so remarkable in some of its features, especially those having reference to the prolongation and sustentation of life in air considerably compressed, that I have great pleasure in sending it for publication in the BRITISH MEDICAL JOURNAL.

I shall not in this report submit any opinions as to the physiological effects of compressed air on the waste of tissue, or in arresting evaporation, and thus preserving the heat of the body and maintaining the temperature of the cell in which the rescued men were entombed.

It is, perhaps, necessary to remind those of the profession who are unused to colliery operations that, when coal is to be dug, a shaft is sunk to the depth at which the mineral is to be found. From the bottom of this shaft radiate one, two, or three principal roadways; and it is on both sides of these roadways, and at right angles more or less with them, that headings are driven. On each side of these headings, openings are made into the coal, called stalls. Tramways run along the main roads and branch off into each stall. Further, it is to be remarked that, with respect to this and many other collieries in South Wales, the headings run up from the roadways, at a dip or angle more or less acute with the plane of the roadway. It can, therefore, be easily understood that, a mine being flooded, it is possible that, though all practical egress from parts of the interior is closed, the air driven into the upper headings and stalls will ultimately assert its limit of compressibility, and prevent the further advance of the water. This compression will, of course, increase with the weight of the superincumbent water above the level of the remote stalls, to which reference has already been made.

With this imperfect summary of the conditions as to the general structure of the colliery, which I think necessary before the gravity of the facts can be fairly appreciated, I at once come to the stall which en-

* The italics are mine.

† The italics in this paragraph are in the original.