may make a pilgrimage to his old school and seek to lay before his revered teacher the matters that have vexed his soul. Alas! the illusion is soon dispelled—the yawning indifference with which the recital of his experiences is received chills his enthusiasm and shakes his faith in his teacher.

Such, indeed, had been my experience, and when I took courage and sought to publish my early observations I fared no better, my early endeavours being invariably disposed of in the learned editor's waste-paper basket. At last an editor friend, being short of copy, published my observations. No doubt they were not of much value, and my idea of their importance was probably excessive. Nobody seemed to take the slightest notice of them. Still, there were a few facts which were then for the first time described, and observations of late years have confirmed them. Nevertheless, at the time of their publication they were simply ignored, and I thought that no one troubled about them until one day in a bookseller's shop I accidentally met Dr. Gibson. At this first interview he referred to my work, and I admit that I felt flattered that one of the brighter spirits, who was then rising in the horizon of fame as a capable clinician, should have been interested in my work. This gratification was all the more real because he had grasped my meaning and my intentions and discussed them with a fullness of knowledge that showed that his interest was not mere surface courtesy. To me, an obscure general practitioner, this interview with a brilliant teacher gave great encouragement to prosecute the work upon which I was engaged. Through all the years that have passed since then I always knew that I had one capable reader to appeal to. His criticisms were always valuable and by no means always complimentary, for he brought a rare judgement to bear upon the subjects under discussion, and he had a power of expression at once forcible and direct. By such discussions new ideas were developed, which helped both of us to find out the truth. To him, as to his writings, I always turned when I wished to know the history of any matter or when I wanted the most reliable and the most recent views, for as I have said, I know of no one who had read so much and who had digested his reading so well. You can understand how grateful a place he holds in my memory, and how, as a slight return for the great help he ever was to me, I have attempted to show the position in medicine that Dr. Gibson held.

So far I have spoken of Dr. Gibson as the distinguished physician, but he was equally remarkable in other ways. It is scarcely necessary that I should dwell upon the fascinating personality of the man—the manly bearing, gracious manner, and the bright and cheery smile which endeared him to all. Those who were intimate with him knew him as a most delightful companion whose wealth of amusing anecdotes and whose versatility and remarkable facility of expression rendered his company the most fascinating. To us he was ever the lover of truth and hater of humbug. Those who were in trouble or in suffering recognized the sincerity and depth of feeling of this large-hearted man. I frequently come across patients who have been under him, and they all express the deepest devotion and gratitude for the manner in which he dealt with them. This manner was no mere pretence, but was the expression of a man of profound sympathy, as many of his colleagues can testify, for he would spare himself no labour to help a friend in trouble, and his purse was always at the call of distress. I have seen him in many phases, but none impressed me so much as the real manifestations of sympathy he extended to us when the hand of sorrow was heavily laid on my household. This testimony also many others can bear, and we feel the world is poorer, and I feel that Edinburgh is no longer such an attractive place to me, since George Gibson is no longer here.

THE Association of German Scientists and Medical Practitioners will hold its eighty-sixth annual meeting this year at Hanover in September (20th to 26th).

THE Medical Faculty of Turin has decided to erect a memorial to the distinguished physiologist, Angelo Mosso, in the institute in which he taught for many years. The memorial will be unveiled on November 14th, 1914, the fourth anniversary of Mosso's death. Contributions should be sent to Professor Alberto Aggazotti, Corso Raffaello, n. 30, Torino.

A Clinical Lecture

ON

PHENOMENA ATTRIBUTABLE TO SPASM OF CEREBRAL VESSELS (ANGIOSPASM).

BY

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I PROPOSE to speak to you to-day on some points bearing upon the blood circulation in the brain and of phenomena due to disturbances of that circulation. Owing to the position of the brain within the cranial cavity, local disturbances of the circulation are difficult of proof; they can only be assumed from the clinical phenomena which may be present and from our knowledge of the dependence of local function upon an efficient local blood supply.

The special condition I propose to speak about is spasm—constriction of the arterial channels in localized areas of the brain; the term "cerebral angiospasm" is given to the condition. Although the cerebral vessels cannot be seen, the vessels of the retina can be examined by means of the ophthalmoscope, and it has been clearly established that angiospasm occurs in these vessels. Various observers have noted that a temporary loss of vision of one half of the retinae may be associated with marked diminution in the size of the retinal vessels corresponding to the blind field, and that vision returns with the relaxation of the constricted vessels and the re-establishment of the normal blood supply to the part. From these observations alone it is fair to postulate that corresponding constriction can take place in the arteries in localized areas of the brain.

Migraine.

This can be illustrated from a common and well-known disorder, namely, migraine.

It is a well-established clinical fact that in migraine such disturbance of vision as has just been referred to is by no means uncommon, and that it can be shown to be associated with the change in the retinal vessels referred to. In migraine, however, there are other temporary phenomena which must have their origin in the brain—of these perhaps one of the commonest is partial or complete loss of power of one side of the face, of one limb, or of both limbs on one side. Other temporary phenomena are characterized by disordered sensation or temporary loss of sensibility; the distribution of such phenomena being usually, if not invariably, one-sided, and affecting either side. These phenomena and the explanation which I have given you as to their causation are now pretty generally recognized and accepted.

Loss of Memory.

There is, however, another set of phenomena which I venture to think may be explained on the same hypothesis, namely, the loss of memory of recent actions which ordinarily imply not only active volition but conscious volition; this set of phenomena is illustrated by what frequently occurs in old people. Old people tend from time to time to get restless and sleepless, and noisy or active during the night. In an attack of this kind, an old person will get out of bed, disturb the other inmates of the same room by interference with the bedclothes and by other acts; they will go to the fire and interfere with it; light matches without any apparent object in view; and the following morning have absolutely no recollection of what had happened during the night; they may even state that they have had a good night and slept well. Periods of mental excitement and incoherence come on in the same way, and may be followed by mental coherence during the day; and here also there is no recollection of the restlessness and incoherence of the night. Cases of this kind may show, during the periods referred to, a definite constriction of systemic arteries; and when this occurs it is found that the most suitable remedy for removing the symptoms is the administration of a vasodilator. So impressed have I been with this experience that, whenever such cerebral symptoms occur as I have referred to, and if the condition of the arterial pulse even

suggests arterial constriction, the routine practice is to administer a vaso-dilator; and it is quite remarkable the number of patients who respond to this treatment and whose attacks may be thereby arrested. The two drugs which I use mainly are phenacetin and erythrol-tetranitrate. From the character of the phenomena and the effect of treatment, it seems to me reasonable to explain such cases on the lines I have indicated.

I beg you to note, before passing from this point, the importance of the proposition that a person who is awake and able to move about, and is able to do acts which ordinarily imply conscious volition, may have recurring periods during which he is absolutely unconscious of his acts and doings.

The case of Walter S. which follows is, I believe, a striking example of this general proposition. In this particular case there were, apparently, definite epileptic phenomena; yet to designate the phenomena to which I am specially referring as epileptoid does not help much; whilst a similar forgetfulness in the nocturnal disturbances of old people cannot be regarded as epileptoid. The presumption is that the explanation of the phenomena in old people is applicable also to the phenomena in this case.

CASE I.

W. S., aged 45, shale-miner, was admitted on October 30th, 1913, complaining of fits of unconsciousness. This patient had been previously admitted to the hospital on February 14th, 1913, and was discharged on March 14th. At that date he was admitted because of sudden fits of unconsciousness. He stated then that he suddenly became unconscious without any previous warning, and that these attacks had been present for over two months; no twitching or muscular movement of any kind took place during the attacks, and he was not pale or cyanosed during them. They took place at intervals of about a week. While in hospital he had two fits which lasted about one minute, from which he slowly recovered, and was quite conscious in about five minutes. A positive Wassermann reaction was in accordance with the specific history given by the patient.

When admitted on October 30th the patient stated that since January, 1913, he had been subject to attacks of mental abstraction, during which he was unconscious of his actions. The attacks lasted for from two to five minutes, but he neither knows he is having an attack nor has he any warning that an attack is coming on. After an attack he is told of his having done things which he has no recollection of having done; the things he does are often "foolish"—as, for instance, he would empty a full pipe and fill the pipe with brown paper without having any recollection of doing so; in one attack he caught one of his children, and threw the child out of the door. When I questioned him about what he had done to his child he was evidently much perturbed emotionally by the matter being recalled to him, and assured me that he had no recollection whatever of having done such an act. He has one or two attacks weekly, and they are not preceded by any aura.

Circulatory System.—The left border of the heart at the apex was slightly outside the nipple line. There was a systolic murmur at the base of the heart. The pulse was regular in rate and in rhythm. There was a marked difference between the two radial pulses, the right being much more forcible than the left. The vessel pressure was 126 mm. An x-ray examination showed an aneurysmal dilatation of the first part of the aorta.

Respiratory System.—The respiratory system showed nothing abnormal.

Nervous System.—The organic and skin reflexes were normal. The plantar reflexes showed normal flexion. The tendon reflexes in the arms were brisk. The knee-jerk was somewhat exaggerated on the right side and somewhat diminished on the left. Both pupils reacted normally to light and to accommodation; there was slight horizontal nystagmus. The other systems presented no abnormal phenomena.

On November 3rd, about 11 p.m., the patient was unconscious and presented a series of clonic contractions of the muscles of the extremities which lasted about two minutes. The patient frothed at the mouth—mucus being blood-stained—but he did not bite his tongue; he slept afterwards.

On November 7th, at 10 p.m., the patient was noticed to be looking queer, and on examination it was found that he was lying on his back, with his eyes wide open and turned upwards; on being spoken to several times he made no response; in a few minutes, however, he recovered and went to sleep. Several attacks occurred during the following days, and on November 12th the patient left hospital against the advice of the house-physician.

Mental Obscuration with Attacks of greater Obscuration and Inability to Work.

The next type of cerebral phenomenon to which I would draw your attention is characterized by a complaint of sleepiness, of entire want of energy, and of marked forgetfulness. Such symptoms are the result of grave

disturbance of the circulation in certain brain areas. Such phenomena may, indeed, indicate more than mere anaemia of brain areas, for it is found that when there has been marked impairment of cerebral function, such as is implied in persistent forgetfulness, it commonly indicates destruction by softening of the area implicated. The softening is the result of cutting off of the blood supply to the area affected, and I believe this often begins in local vessel spasm and is succeeded by local thrombosis. When such phenomena are temporary the fact implies temporary diminution in the supply of nutriment to the area involved. The evanescent character of the phenomena, associated as it frequently is with tendency to recurrence, must be regarded as due to variations in the supply of blood to the affected area; while the permanent loss of function implies permanent arrest of blood supply and, as an inevitable consequence, necrosis and softening of the area affected. Even when such softening has occurred, there may be added to the permanent symptoms accessory phenomena which appear and disappear from time to time. Such accessory phenomena may be a feeling of being dazed and of entire inability to attend to business of any kind. Such accessory phenomena may, as has been said, pass off in the course of some days, and may only reappear at intervals of weeks or months. They are also, in my opinion, to be explained by the occurrence of impaired blood supply to regions which may adjoin the softened area, although they may, of course, have a totally separate site. It is on this hypothesis that satisfactory treatment of such cases is carried out. The aim of treatment is to reduce the irritability of the vessel wall which leads to such local spasm. In some cases no doubt such temporary diminution of blood supply as is here pictured may be the result of periods of special heart enfeeblement associated with marked atheroma of cerebral vessels; but in my experience this only accounts for the phenomena in a limited number of cases, the phenomena being more commonly met with in cases in which there is no question whatever of such heart enfeeblement as would lead to insufficient supply of blood. The following case of James B. illustrates these contentions.

CASE II.

J. B., aged 62, an umbrella-maker, was sent by Dr. Calder. He was accompanied by his wife. He complained of being continually sleepy, having no energy, and being very forgetful. This condition had lasted nearly a year, and had been preceded by severe epistaxis. The bowels moved daily. He had been a hearty eater, but had never taken alcohol.

His wife informed us that during the past year he had had three attacks of being dazed and unfit for work; the two first had lasted for ten days, while the one he was at present suffering from began a fortnight previously. She stated that his mind was never quite clear between the attacks. On questioning him he said that he used to read a good deal, but that he had given up reading for the past year. The arterial pressure was 220 mm. Hg, and there was a small amount of albumin in the urine.

My interpretation of the phenomena in this case is as follows: About a year ago this patient had an attack in which he was dazed and unfit for work; the attack lasted for ten days, and was preceded by severe epistaxis. The arterial pressure when he came to hospital was 220 mm., and there was a trace of albumin in the urine. What happened at the time of his attack was that his systemic arteries were experiencing a wave of hypertonic constriction; that a corresponding condition was present in at least part of his brain, producing symptoms referable to mental areas, as indicated by his being dazed and stupid, and that this had lasted long enough to allow of softening to occur in one of those areas. He made a partial recovery as the brain vessel spasm subsided, but the recovery was only partial, as the area of softening remained. He was able to attend in some degree to his business, for he had no motor paralysis, but his memory was impaired and he had lost his former energy. Since that first attack he had had two other attacks in which he became dazed and was quite unable to attempt to go to his place of business. The attack from which he was suffering when we saw him had lasted longer than the two first. These attacks mean, in my judgement, a recurrence of the constriction of brain vessels, and each attack, if it leads to a fresh area of softening, will leave him on a lower mental level than before.

This, to my mind, is not only the picture of what has been happening in this particular brain, but it represents

common occurrences and experiences, and the great advantage of having a correct moving and living picture is that in many cases of the kind the brain angiospasm can be stopped and your patient saved from cerebral softening.

Temporary Losses of Ability to Perform Ordinary Work.

Another set of phenomena is illustrated in the case of a patient, J. W., to whom fuller reference is made later. This patient was 31 years of age, and was a joiner by trade. His mother stated that for nine years he had from time to time suffered from "stupid" attacks, characterized by inability to perform the most ordinary procedures necessary for doing his work as a joiner; he would, for instance, be incapable of taking the simplest measurements necessary to the carrying out of simple pieces of work. A similar mental inability to deal with other matters showed itself. The attacks lasted for two or three days, and gradually wore off, and as they wore off he began again to understand things and to be able to do his work. He also showed that, while he recognized his mother as "mother," he could not understand how it was she was his mother. Notwithstanding this strange obscuration of his mental processes, he was able to go about as usual, and would even go to his workshop with a view of working; but, even when put to the simplest of operations, he could not carry them out. These attacks have come and gone, some of them having been short attacks, others of them having lasted longer. In this case, again, it seems to me difficult to explain the phenomena otherwise than by attributing them to local impairment of nutrition in the areas affected, and that local vessel spasm alone enables us to form a tangible view of the phenomena. Merely to give the phenomena the designation of being epileptoid, again, seems to me to lead us nowhere. The following is the more detailed account of this case:

CASE III.

J. W., aged 31, was sent to me on February 11th, 1914, by Dr. Bowie with a note saying that the patient had an attack about a year and a half ago similar to that from which he was at present recovering.

We found it impossible to get a satisfactory history from the patient himself. His mother, who accompanied him, gave us the following history, namely, that about nine years ago he began to have attacks in which he was stupid in ordinary work; he could not even take ordinary measurements. The attacks lasted for two or three days and gradually wore off, and as they wore off he again began to understand things and to be able to do his work. His father died in July, 1912, and in the following October he took an unusually bad turn and did not go to work until January. His mother informed us that during that time he walked about and saw her, but would say to her that he did not understand she was his mother; he at that time could not be made to understand anything in connexion with any papers that required to be signed bearing upon his father's affairs. He gradually got over that attack and was pretty well during all 1913, but on the Monday before he was sent to see me he went to the joiner's shop in which he worked, but was quite unable to do the simplest of the operations necessary for his work.

Epileptoid Attacks simulating Fainting Attacks.

The following is an example of epileptoid attacks resembling fainting:

CASE IV.

L. S., a young woman, was sent to me by Dr. Deuchars, with a history of continually having fainting attacks, the nature of which was not apparent, and she was sent to me for opinion. Her employer was much interested in her, and was anxious to know if he could do anything for the girl.

The story we got from the patient of her attacks is that in the forenoon she feels sick, loses control of her muscles, and sometimes falls. These attacks come on quite suddenly with a feeling of extreme nausea and pain in the head, in the right temple region. She never vomits. She has on two occasions suffered from concussion as the result of knocking her head upon the floor. The lesser attacks last for about a quarter of an hour, as she has been told. She is so upset by the attacks that she has to rest the whole day thereafter. The attacks are more apt to come on when menstruation is due, but they are not confined to that time. The girl's colour was good, and there was no appearance of anaemia. The heart sounds were closed and distinct. The pressure was 114 mm. The bowels were kept regular by means of a laxative, and there was no albumin in the urine.

You have had lectures on the grave and the minor forms of epilepsy, and I only indicate to you at present that epileptoid phenomena sometimes respond to vaso-dilators, and that there is a vessel theory of epilepsy.

Vertigo due to Brain Angiospasm.

The following two cases illustrate this relationship:

CASE V.

J. W., aged 49, a labourer to an electrical company, recommended by Dr. Hamilton. Admitted January 23rd, 1914, complaining of giddiness of ten weeks' duration. The patient drinks a good deal of beer, and smokes 4 oz. of tobacco weekly. He has a comfortable home, and has a family of eight children alive and well.

History.—Ten weeks ago he began to suffer from giddiness, which came on after stooping and was very severe. In one day he had three attacks of a sensation of coldness and numbness on the left side of the body, including the face, the trunk, and the limbs. He at the same time experienced a feeling of nausea and a salt taste in the mouth. On one occasion he fell. His bowels, which as a rule act well every day without medicine, had been acting sluggishly prior to the onset of the attacks referred to; there was no vomiting, but he said his sight was bad and he had a slight headache; the giddiness was worst four days after the attack of numbness.

After two weeks' illness he was admitted to the Hawick Hospital, where he stayed for five weeks. Since then he has been two weeks at home, when at the request of Dr. Hamilton he was admitted to the Royal Infirmary. His sight had improved a little, but he was still subject to threatenings of attacks similar to those described. He said, however, that he was able to avert them by lying down, when after ten minutes he felt perfectly well again. He had these attacks as a rule once or twice a day, but sometimes missed a day. He stated that the attacks began with a gripping sensation round the left upper arm.

Condition on Admission.

Nervous System.—Intellectual and mental functions satisfactory. He has slight headache in the frontal region and also pain shooting along the vertex. He still suffers from giddiness, which is so marked sometimes that turning in bed to use the spittoon may make him feel as if he would fall out of bed. Voluntary movement is in no way affected. Sensibility to touch, pain, and temperature are normal. Reflexes are normal; there is no inco-ordination and no Rombergism; although the patient says that when first tested for this by his own doctor he was very unsteady.

Special Senses.—The pupils are normal and react to light and accommodation. Vision is good; the patient can read, and the discs, as reported on by Dr. Sim, show a little obscuration of the margins within physiological limits, and the vessels did not appear to be in any way abnormal. The patient was a little deaf on the right side. Dr. Logan Turner, who kindly examined his ear condition, reported that the patient had evidence of old-standing middle-ear disease; that he had a large perforation and some moisture on the right side, and this ear had discharged for many years. Dr. Turner further stated that the tests went to show that the deafness was entirely of the middle-ear type and that the inner ear was not affected.

Circulatory System.—The pulse varied from 68 to 80 or more and was regular in force and type. The arterial pressure was 140 mm. The apex of the heart was in the sixth interspace, 1 in. outside the nipple line; the sounds were pure.

Respiratory System.—There is a history of winter cough for some years. The lungs were normal.

The abdominal organs were normal. The urine was of acid reaction, specific gravity 1025, and showed no abnormal constituents.

He was treated with 10 grains of potassium iodide three times a day. On February 6th the dose was increased to 20 grains three times a day. On February 9th he had greatly improved, the headache and the giddiness having completely disappeared, and he was allowed to get out of bed. He was discharged on February 14th, having showed no recurrence of his symptoms. On February 10th his arterial pressure was 110 mm. and his pulse was between 72 and 80.

The preceding facts suggested that the phenomena might be the result of brain angiospasm. The fact that the vertigo had been preceded by such marked disturbance of sensation on one side of the body from the face downwards, and that this abnormal sensation had appeared and disappeared several times in the course of one day, suggested this view. The action of potassium iodide in counteracting the tendency to vessel spasm seems to me from clinical experience to be definitely established. In this case the effect of the drug on the patient was eminently satisfactory, and my contention with regard to its action is supported by the fact that with the disappearance of the cerebral phenomena the pressure was found to have fallen from 140 mm. to 110 mm.

CASE VI.

Mr. W., aged 67, a solicitor, was sent to me on February 12th, 1914. He complained of attacks of headache, referred to the back of the head, and of giddiness, which might or might not be accompanied with vomiting. These attacks came on if he applied his mind very closely to his work, if his work worried him, or if he became excited in discussion or controversy. The

giddiness might be so pronounced as to compel him to stop work and to go home in a cab. Rest led to disappearance of these symptoms.

The pulse was 60, and the arterial pressure was 110 mm. On lying down the pulse was definitely fuller than when sitting. The vessels were soft. The heart at the apex was in the mammary line; the sounds were fairly good and closed. The abdomen presented no abnormality; to regulate the bowels a penta was taken regularly. The urine presented no abnormal characters.

In this patient the headache and giddiness were primarily cerebral, and were to be regarded as the result of disordered cerebral circulation, due to too close mental application, or to emotional influences. There could be no doubt that there was an imperative call for complete mental rest, away from the patient's ordinary environment. Here, again, on the hypothesis of local cerebral angioplasm being the cause of the phenomena, the danger was not that in one of these attacks cerebral haemorrhage would occur, but that the angiospasm might be so pronounced and of sufficient duration to lead to cerebral softening in the more purely mental areas of the brain.

A Lecture

ON

OXYGEN WANT AND THE MEANS OF ITS RELIEF.

By A. G. AULD, M.D., M.R.C.P.,

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THE important advances made within recent years in the physiology of the respiration have furnished us, amongst other things, with more precise conceptions as to the rôle of the blood gases, the nature and production of oxygen want, the adaptations provided by the body for the relief of this want, and their assistance by art. To the laborious investigations of Haldane and his collaborators in particular we owe in great measure the light which has been shed on this subject, albeit many difficult problems still await solution. Now, oxygen want constitutes in itself a true disease of the respiration; therefore in this lecture it is not proposed to discuss the gross results of prolonged want of oxygen such as are found in the heart, the nervous system, the alimentary system, and the kidneys, but to confine attention to the respiration factors proper.

The Gases of the Blood.

The governing factor in the respiration is the carbon dioxide of the arterial blood. As observed by Haldane, the respiratory centre is probably as sensitive to carbon dioxide as the retina is to rays of light. Its activity is entirely dependent upon it, and should the tension of this gas fall below a certain point apnoea will result. When Head made his observations it was supposed that the vagi not only regulated the rhythm, but to a considerable extent also activated the centre. A vagus apnoea was supposed to result from summation of stimuli. But there appears to be little room for this theory nowadays. The tension or partial pressure of the carbon dioxide in the blood and alveolar air is maintained constant under normal conditions (Haldane and Priestley); the slightest increase exciting the respiratory centre and the excess of gas being given off by the increased breathing. It is now considered that what the centre really responds to is the hydrogen ion concentration. So sensitive is this reaction that a rise of 0.22 per cent. in the alveolar carbon dioxide percentage—that is, of 1.6 mm. in the pressure (the normal pressure being about 40 mm.)—will raise the breathing by 100 per cent. This corresponds to a difference in the hydrogen ion concentration so minute as to be beyond detection. Now, any acid other than carbonic, if added to the blood, increases the hydrogen ions in proportionate amount, and the effect of these, added to the hydrogen ions of the carbonic acid already present, is to excite the centre and cause hyperpnoea. To put this differently, the added acid lowers the threshold of excitation of the centre to carbon dioxide, so that it reacts to a correspondingly lower tension of the gas. This factor is of the greatest

importance and constantly comes into play in the physiology and pathology of the respiration. It is due to defective oxidation in the tissues.

Let us recognize that the respiration is essentially carried on in the tissues. The lungs, the circulation, and the kidneys are receiving, transmitting, and adjusting agents. It is the gas exchanges in the tissues which determine those in the lungs. In either case they are not necessarily carried out according to the law of the diffusion of gases. The endothelial lymph cells, the endothelium of the capillaries, and the epithelium of the lungs have secretory and selective functions. Normally they permit diffusion to take place in accordance with the gas laws, but under abnormal or exceptional conditions they exercise their powers of selection and secretion. The experiments of Bohr in 1891, attributing this function to the pulmonary epithelium, has been amply supported by recent research. Strassburg also found that the tension of carbon dioxide may be lower in the lymph spaces than in the venous blood. This secretory action, as in the case of the swim-bladder of fishes, may be largely dependent on the integrity of the pertaining nerves.

In the next place let us briefly consider the all-important matter of the tension or partial pressure of the gases of the blood. It is the tension that matters, not the amount. The amount may vary widely, and yet the pressure remain the same. In the case of the carbon dioxide, this regulation is very intricate and only partially understood. Of the total quantity of the gas taken up by the blood, from 36 to 46 volumes are in chemical combination, and only two and a half volumes are in solution in the plasma and corpuscles. It is only this small amount of the free gas in solution that determines the tension, the large quantity in loose chemical combination forming a store, so that as soon as the gas in solution decreases, an equivalent amount is dissociated to take its place, and vice versa, if the gas in solution rises above normal, the excess is taken into chemical combination, until matters are again adjusted by the breathing. This regulation is carried out chiefly through the serum-globulin. When the tension rises the globulin is displaced from its adsorption combination with the alkali (sodium) by the mass action of the gas, and alkali carbonate or bicarbonate is formed; when the tension falls the globulin again enters into possession. As the carbon dioxide tension rises the alkalinity of the plasma also rises. Very high tension, according to Gürber, causes expulsion of some of the hydrochloric acid from the sodium by mass action, with migration of the acid to the corpuscles. When acids other than carbonic—that is, those due to defective oxidation in the tissues—enter the blood, the carbon dioxide is dissociated to a certain extent and pumped out by the breathing. Such acids, however, except under extreme conditions, are taken up by the blood only to a limited extent, as its potential alkalinity is maintained within certain definite and necessary limits. As acid products of catabolism increase, the organism utilizes ammonia (which otherwise would pass to the liver to form urea) to combine with them. Hence the quantity of ammonia present in the urine is an index of the acidity of the blood. Did the organism not possess this means of defence, acid poisoning would quickly supervene in many conditions. On the other hand, as will shortly be seen, the addition of a certain amount of acid to the blood acts in a compensatory or protective fashion.

The partial pressure of carbon dioxide, as has long been recognized, exerts a very important influence on the absorption and dissociation of oxygen; the higher tension of carbon dioxide in the capillaries facilitating the dissociation of oxygen to the tissues, and the fall in tension in the lungs facilitating the absorption of oxygen. On the other hand, it has consistently been held since the experiments of Bohr in 1890, that the absorption and dissociation of carbon dioxide were uninfluenced by the presence of oxygen, and the ready combination of the former gas with methaemoglobin lent support to this belief. Just lately, however, an important communication has been made by Christiansen, Douglas, and Haldane,¹ in which it is contended that saturation of the haemoglobin with oxygen has a very marked effect in diminishing the absorption and increasing the dissociation of carbon dioxide as compared with blood deprived of oxygen. In the light of this statement, the blood gases reciprocally exert exactly similar effects, in accordance with the needs of the body.