AN ESSAY
ON
ASPHYXIA

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(APNŒA)

BY

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TO THE LOVED AND HONOURED MEMORY OF

SIR THOMAS WATSON, BART.

A GREAT PHYSICIAN

AND ONE OF THE WISEST AND BEST OF MEN

PREFACE

THE subject of Asphyxia has had for me an especial interest since the now far-off time when my friend and former teacher Dr. Todd directed my attention to the experiments of Dr. John Reid, whom he highly esteemed as an eminently accurate and trustworthy observer.

I have been surprised to find that some recent text-books on Physiology, in opposition to previous writers of eminence, give what I believe to be an incorrect description of the state of the heart's cavities immediately after death from asphyxia, and with this an erroneous explanation of the manner in which the circulation is finally arrested. The subject is one not only of high scientific interest, but of great practical importance; inasmuch as a true physiological interpretation of the phenomena affords valuable assistance in the solution of some complex pathological problems. Therefore, in the hope of exciting more general interest in the subject, I have been induced to publish the following essay in three sections.

A clear perception of the vital mechanism by which the blood is propelled and its distribution regulated being

essential for a true theory of asphyxia, I have, in the first section, briefly set forth the now generally accepted doctrines with regard to the physiology of the circulation.

In the second part I have subjected some conflicting statements and theories respecting asphyxia to a critical examination, and I have given what I believe to be the correct physiological explanation of the facts.

In the concluding section I have instituted a comparison between various pathological conditions and the phenomena of asphyxia.

I have not ventured without much thought and careful consideration to express my dissent from the teaching of some eminent physiologists whom I have quoted; and I have only to add that, my motto being *In lucem aspiro*, I shall be truly thankful to be informed of any error, whether of statement or of inference, if such should be found in the following pages.

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AN ESSAY

ON THE

PHYSIOLOGY OF ASPHYXIA (APNŒA)

SECTION I

THE PHYSIOLOGY OF THE CIRCULATION

If this essay should come into the hands of anyone not thoroughly acquainted with the most recent doctrines regarding the forces concerned in carrying on the circulation of the blood, the following brief statement may assist him to understand the arguments for and against certain theories which will presently be discussed.

Influence of heart and large arteries

There is now a very general agreement amongst physiologists with respect to the influence which the heart, the large elastic arteries, and the terminal muscular-walled arterioles respectively exert upon the circulation. The force which propels the blood through the systemic arteries is derived entirely from the contraction of the muscular walls of the left ventricle of the heart. The elastic walls of the large arteries, distended by the injecting force of the heart, react upon and force the blood onwards during the diastole of the ventricle. This resilient force in the walls of the arteries is as obviously derived from the contraction of the muscular heart as the elastic force in an archer's bow has its

source in the contraction of the muscles which bend the bow.

The resiliency of the arterial walls, reacting upon the blood during the diastole of the ventricle, gradually converts the intermitting jet of blood from the heart into a continuous stream in the minute arteries and capillaries.

Action of muscular arterioles

The smallest ramifications of the arteries, whose middle coat is entirely muscular, are conveniently designated *muscular arterioles*. These actively contractile arterioles, under the influence of the vasomotor nerves, regulate the blood supply to the various tissues and organs. Their action is analogous to that of stopcocks. By the contraction of their muscular walls their canals are narrowed; the onward blood-stream is in a corresponding degree lessened, and the pressure of blood in the larger arteries behind is increased. 'This contraction may go so far as, in some cases, to reduce the cavity of the vessel almost to nothing, and to render it practically impervious.'[1] On the contrary, relaxation of the walls of the arterioles enlarges their canals, thus permitting a fuller stream of blood to pass onwards into the capillaries, and so lessening the tension and blood pressure in the arterial trunks and larger branches.

The muscular arterioles therefore, through their stopcock action, exert a regulating but not a propulsive influence on the blood current. Professor Huxley says: 'While the small arteries lose the function, which the capillaries possess, of directly irrigating the tissues by transudation, they gain that of regulating the supply of fluid to the irrigators or capillaries themselves.

[1] Huxley's *Elementary Physiology*, 1886, p. 25.

The contraction or dilatation of the arteries which supply a set of capillaries comes to the same result as lowering or raising the sluice gates of a system of irrigation canals.'[1]

The discovery of the structure and the function of the arterioles is the most important addition to our knowledge of the circulation which has been made since the time of Harvey. A clear perception of the means by which the movement of blood through the terminal vessels is regulated, and by which it may be arrested, is essential for a thorough comprehension of the physiology of the circulation and for the right interpretation of many pathological phenomena.

Capillaries

The *capillaries* have no muscular fibre in their walls, and they have therefore no power of active vital contraction. They become distended and dilated when the muscular arterioles are much relaxed, and they return to their original size when the arterioles contract and lessen the blood stream; but the contraction of the capillaries is apparently the result of simple elastic resiliency after distension and not of an active vital contraction. The capillary obstruction which occurs during the process of inflammation, when the white corpuscles especially adhere to the walls of the vessels, is, of course, entirely different from a normal physiological impediment.

Veins

The propulsive force of the heart is transmitted through the capillaries into the *veins*, and so assists to drive the blood towards the right side of the heart.

The small veins have in their middle coat some circular muscular fibres, the contraction of which tends to propel the blood into the venous trunks. The return

[1] Huxley's *Elementary Physiology*, 1886, p. 25.

of the blood to the heart is aided by the pressure of the actively contracting voluntary muscles (the valves preventing a reflux towards the capillaries), and also by the suction force of the chest during each act of inspiration. The terminations of the venæ cavæ and the pulmonary veins in the right and left auricle respectively are surrounded by muscular fibres continuous with those of the auricles; and these terminal veins may be seen to contract and dilate alternately, so as to drive the blood into the auricles.

Pulmonary circulation

The pulmonary circulation is effected by the same propelling and regulating influences as the systemic; but as less force suffices to send the blood through the lungs than is required to propel it through the entire system, so the walls of the right ventricle are thinner and less powerful than those of the left, and, in the same proportion, the coats of the pulmonary artery are thinner and less powerfully elastic than those of the aorta; the pulmonary arterioles likewise contain in their middle coat less muscular tissue than the systemic. The propelling and the regulating forces are so exactly balanced that in the normal condition they work harmoniously together; but we shall hereafter see that in certain pathological states the pulmonary arterioles contract so powerfully as to entirely arrest the flow of blood through the lungs, the resisting force of all the pulmonary arterioles, when contracting simultaneously, being greater than the propelling power of the right ventricle.

Mechanical model

The influence of the heart, the large elastic arteries, and the muscular arterioles respectively may be shown by a very simple piece of mechanism. A pump is made of a hollow indiarubber ball with two orifices, to one of

which is attached an indiarubber tube six inches long, and to the other an elastic indiarubber tube about four feet long and having at its distal end a brass stopcock. The central end of each tube is guarded by a bullet valve. The end of the short tube being dipped into a basin of water, while the elastic ball is alternately compressed and relaxed by the hand, the intermitting jet of water from the hollow ball, representing the heart, is converted into a continuous stream by the long tube, thus acting like the large elastic arteries. Placing the finger on the tube near the pump, at each compression of the pump the tube is felt to expand and then to recoil. This distension is the pulse, the result of a wave of alternate expansion and contraction of the tube, projected from the indiarubber heart.

The size of the continuous jet from the metallic orifice is regulated by the more or less open condition of the stopcock, which here imitates the function of the muscular arterioles.[1]

The smaller the orifice of the stopcock the greater is the resistance offered to the escape of the fluid, and the greater the force required to work the pump.

Effect of a rigid tube

If now we substitute for the elastic tube one with rigid walls, the stream of water from the orifice of the stopcock is no longer continuous, but an intermitting jet; so if the opening in the stopcock be large enough to

[1] This apparatus is described and figured by Dr. Rutherford in a very interesting lecture on the circulation, in the *Lancet*, Feb. 17, 1872. In that lecture he says he 'caught the idea' of representing the muscular arterioles by a stopcock from my observation of the altered condition of the small arteries in Bright's disease. This condition—namely, hypertrophy of the muscular coat of the arterioles—is a result of what I have ventured to call their *stopcock* action, and it will be referred to hereafter.

allow the water to escape as fast as the pump forces it into the tube the outflow will be interrupted. This wide-open state of the stopcock represents a greatly dilated condition of the arterioles, when the pulse may extend through the capillaries into the veins. For the conversion of the intermitting jet from the pump into a continuous stream from the stopcock, the orifice in the latter must be so small as to allow the fluid to accumulate in and distend the tube, the elasticity of which continues to drive on the fluid while the pump, representing the heart, is dilating to receive a fresh supply.

SECTION II

THE PHYSIOLOGY OF ASPHYXIA (APNŒA)

Conflicting statements

On a comparison of some physiological text-books which have been published within the last fifteen years with corresponding works of earlier date, it will be found that the former directly and decidedly contradict the latter with regard to the relative amount of blood on the two sides of the heart immediately after death from what is commonly called asphyxia. The term *apnœa* may, however, be more appropriately used to designate deprivation of air, and I shall so employ it in this essay.[1]

Todd and Bowman

In the 'Physiological Anatomy and Physiology of Man,' published by Todd and Bowman in 1856, the phenomena of apnœa—there called asphyxia—are thus described (p. 375): 'When the access of air to the lungs is excluded the circulation ceases at the pulmonary capillaries, and on examination after death *the left auricle and ventricle are found quite empty, and the right cavities of the heart gorged with blood.*[2] The repletion of the latter cavities and the emptying of the former

[1] Physiologists apply the term *apnœa* to the temporary suspension of breathing resulting from artificial hyperoxygenation of the blood. This may be called 'physiological apnœa,' but pathologists require the term to designate an impediment to or complete arrest of breathing, while the term asphyxia (pulselessness) is applied to a partial or complete arrest of the circulation.

[2] The italics are mine.

indicate the position at which the obstruction to the circulation took place.'

The only part of this brief but clear description which is not in accordance with the results of later research is that which assumes that the blood is arrested in the capillaries. We shall presently see that the blood is arrested by the constriction of the minutest muscular-walled branches of the pulmonary artery.

Dr. Taylor

Dr. Taylor, in the first edition of his 'Principles and Practice of Medical Jurisprudence,' published in 1865, says (p. 117): 'As the circulation of the blood in asphyxia is primarily arrested in the lungs, the pulmonary artery, the right cavities of the heart, and the venæ cavæ are found gorged with blood. The pulmonary veins, the left cavities of the heart, and the aorta are either empty or contain but little blood.' In the second edition of the same work, published in 1873, Dr. Taylor says (vol. ii. p. 13): 'In asphyxia the right cavities are generally found to contain blood, while the left cavities are either empty or contain much less than the right.' He then quotes some statistics published by Dr. Ogston, who found the right cavities empty twice in fifty-three inspections, while the left cavities were empty in every case.[1]

Drs. Guy and Ferrier

Drs. Guy and Ferrier, in their 'Principles of Forensic Medicine,' published in 1875 (p. 261), state that after death from apnœa 'the veins of the heart are distinctly

[1] I shall show hereafter that in the very rare cases in which, after death from an obstructed circulation through the lungs, the right cavities are found to contain little or no blood, this is due to the fact that the blood has escaped from the right side of the heart, in consequence of a large vein having been wounded in opening the chest.

traced on its surface; its right cavities and the large venous trunks are gorged with black, thick, liquid blood, but its left cavities are found nearly or quite empty.'

It will be seen that the authors hitherto quoted describe the relative amount of blood in the right and left cavities of the heart after death from apnœa in almost identical terms. I now proceed to show that later writers contradict the preceding statements as to the comparative emptiness of the left cavities.

In 1875 Drs. Klein, Burdon Sanderson, Michael Foster, and Lauder Brunton published conjointly a 'Handbook for the Physiological Laboratory,' under the editorship of Dr. Sanderson, who is also the author of the chapter on 'Respiration,' from which I am now about to quote.

Dr. Sanderson describes the great enlargement of the heart which occurs during the final struggle in cases of asphyxia, when the great veins are so distended that 'if cut into they spirt like arteries.' He afterwards says (p. 323): 'If the heart is rapidly exposed immediately after death by asphyxia, and a strong ligature tightened round the roots of the great vessels, the organ may be readily cut out without allowing any blood to escape from its cavities. The quantity of blood contained in the right and left side respectively may be measured by carefully opening the ventricles and allowing their contents to flow into separate measure glasses. It is always found that all the cavities of the heart are filled to distension, the quantities in the right and left cavities respectively usually being to each other in the proportion of about two to three.' Dr. Sanderson

According to Dr. Sanderson, then, so far from the

left side of the heart being comparatively empty when examined immediately after death from asphyxia (apnœa), as described by the writers before quoted, it contains more blood than the right side. Before I give my reasons for dissenting from Dr. Sanderson's doctrine I proceed to indicate that it has been accepted by subsequent writers on the subject.

Mr. Power

In the ninth edition of Carpenter's 'Physiology,' edited by Mr. Power, the phenomena of apnœa are described for the most part in Dr. Sanderson's words, including the following sentence: 'Its contractions [the heart's] become more and more ineffectual till they finally cease, leaving the arteries empty, the veins distended, and its own cavities relaxed and full of blood' (p. 390).

Dr. Stevenson

The third edition of Dr. Taylor's 'Principles and Practice of Forensic Medicine,' published in 1883, after the author's death, was revised by Dr. Stevenson, who, in direct opposition to the description which I have before quoted from Dr. Taylor's first two editions, states that in animals killed by asphyxia 'the cardiac chambers are all gorged with blood, the left as well as the right' (vol. i. pp. 164–5).

Dr. M. Foster

Dr. Michael Foster, in his 'Text-Book of Physiology' (3rd edit. p. 352), makes the following statement: 'If the chest of an animal be opened under artificial respiration, and asphyxia brought on by cessation of the respiration, it will be seen that the heart during the second and third stages becomes completely gorged with venous blood, all the cavities as well as the large veins being distended to the utmost. If the heart be watched to the close of the events it will be seen that the feebler strokes which come on towards the end of the third stage are quite

unable to empty its cavities, and when the last beat has passed away its parts are still choked with blood. The veins spirt out when pricked, and it may frequently be observed that the beats recommence when the over-distension of the heart's cavities is relieved by puncture of the great vessels.'

Rigor mortis hypothesis

The preceding description is practically identical with Dr. Sanderson's. Then to explain the fact that in post-mortem examinations the left cavities of the heart are commonly found empty, Dr. Foster says: 'When rigor mortis sets in after death by asphyxia the left side of the heart is more or less emptied of its contents, but not so the right side. Hence in an ordinary post-mortem examination in cases of death by asphyxia, while the left side is found comparatively empty, the right side appears gorged.'

Dr. Foster

Professor Gerald Yeo

Professor Gerald Yeo, in his 'Manual of Physiology,' says, 'Both sides of the heart and the great veins are engorged with blood in the last stage of asphyxia; the cardiac muscle, being exhausted, from want of oxygen, is unable to pump the blood out of the veins or to empty its cavities. Owing to the force of the rigor mortis of the left ventricle, and the greater capacity of the systemic veins, the left side is found comparatively empty some time after death, and at post-mortem examinations the right side alone is found overfilled.'

Drs. Landois and Stirling

Again, in Landois's 'Human Physiology,' translated, with additions, by Dr. Stirling, we find the following statement as to the condition of the heart's cavities after death from asphyxia (2nd edit. p. 284): 'The right side of the heart, the pulmonary artery, the venæ cavæ, and the veins of the neck are engorged with dark venous

blood. The left side is comparatively empty, because the rigor mortis of the left side of the heart and the elastic recoil of the systemic arteries force the blood towards the systemic veins.'

It will be seen that the three physiologists last quoted with one accord, and in almost identical terms, explain the comparative emptiness of the left cavities by rigor mortis of the ventricle. This hypothesis has been framed to explain the unquestionable fact, which is not even noticed by Dr. Sanderson, that at the post-mortem examination in cases of death from apnœa the left cavities are always found comparatively empty and never distended. I shall have little difficulty in proving that this explanation is inconsistent with well-ascertained facts.

Right heart and veins distended

There is one anatomical fact respecting which all writers on the subject of apnœa are agreed—namely, that the right cavities of the heart and the systemic venous trunks are not only *full* of blood, but they are *distended*. Dr. Sanderson, as we have seen, says the veins 'are so distended that if cut into they spirt like arteries,' and Dr. Foster says 'the veins spirt out when pricked.' The distension of the right cavities of the heart after death from apnœa was first demonstrated two centuries and a half ago by the immortal discoverer of the circulation of the blood.

Harvey on execution by hanging

Harvey says ('Second Disquisition on the Circulation of the Blood,' Sydenham Society's translation, p. 127): 'I have several times opened the breast and pericardium of a man within two hours after his execution by hanging, and before the colour had totally left the face, and in presence of many witnesses have demonstrated the right auricle and the lungs distended with blood, the

auricle in particular being as large as a large man's fist, and so full of blood that it looked as if it would burst. This great distension, however, had disappeared the next day, the body having stiffened and become cold, and the blood having made its way through various channels.'

Harvey here makes no reference to the condition of the left cavities of the heart, but this defect has been supplied by many later observers.

Dr. Massey's case of hanging

Dr. Massey, of Nottingham, has published the following report of the appearances found in the chest of a man four hours after his execution by hanging ('Lancet,' Nov. 9, 1867): 'On removing the sternum and cartilages of the ribs, the lungs were not to be seen, but were found to occupy a very small space at the back of the chest, resembling the contents of a fœtal thorax, the pericardial sac alone being seen. The colour of the lungs was of a darker hue than natural, especially at the bases. On cutting out the lungs a quantity of black liquid blood flowed. The structure was natural, but there was loss of crepitancy, and but very little air was contained in them. The right auricle of the heart was gorged to the greatest state of distension with blood, and the inferior cava was in the like condition. On opening the auricle a great quantity of black fluid blood gushed out. The right ventricle also contained a large amount of blood. The left auricle and ventricle were quite empty.'

It is evident from the post-mortem appearances that in this case and in the cases recorded by Harvey, death was the result of the exclusion of air from the lungs. With the modern method of execution by hanging it is customary to allow a drop of about six feet from the

platform, the effect of which is usually to cause dislocation or fracture in the cervical region of the spine, with instant death from the simultaneous arrest of the respiration and circulation. In such cases the appearances after death would of course be quite different.

It may perhaps be objected to the record of Dr. Massey's case that the left cavities had been emptied by rigor mortis during the four hours which intervened between the man's death and the inspection. This objection, however, would not be applicable to the results of the following experiment.

Experiment on a dog

In October 1867 a dog weighing 19¼ lbs.[1] was killed in my presence by a ligature on the trachea. The animal continued to struggle convulsively for about five minutes. As soon as these movements had ceased the chest was opened. The pericardium was so filled and stretched by the distended heart that it was at first supposed that the sac of the pericardium had been opened, so as to lay bare the heart. The right cavities of the heart were full and tense, the left comparatively empty and flaccid. In particular the two auricles presented a marked contrast; the right auricle stood out in a globular form and had a tense and elastic feel like an indiarubber ball distended with air, while the left auricle was flaccid and had its surface wrinkled. A ligature having been placed round the large vessels, the heart was removed and its cavities opened, when two ounces of blood gushed out of the distended right cavities, while two drachms and a half only flowed slowly from the left

[1] In my volume of *Medical Lectures and Essays* the weight of the dog is erroneously given as 14¼ lbs.

side, the relative proportion being sixteen to two and a half.

After division of the large vessels twelve ounces of blood escaped into the cavity of the chest, chiefly from the venæ cavæ and the pulmonary artery. The lungs, which were pale and anæmic, had collapsed to an extreme degree.

It will be seen that the condition of the heart's cavities and of the lungs was identical with that observed by Dr. Massey in the man who had been executed by hanging. In this experiment there could, at any rate, be no question of the left cavities of the heart having been emptied by rigor mortis.

Result of delaying inspection

The recently propounded doctrine that the left cavities are full immediately after death, but are subsequently emptied by the rigor mortis of the ventricle, is inconsistent with the fact that the sooner the chest is opened after death the greater is the distension of the right cavities and the emptiness of the left. When the inspection has been delayed for twenty-four hours or more, not only is it found that the right cavities are much less distended, as was first noted by Harvey, but the vital contraction of the pulmonary arterioles having ceased, some of the blood has been driven onward by the elastic resiliency of the distended pulmonary artery and the right cavities of the heart, so as to engorge the pulmonary capillaries, which immediately after death are always empty; while another portion passes on through the pulmonary veins to the left side of the heart.[1]

[1] It is really surprising that recent writers on asphyxia should, in direct opposition to earlier physiologists of great eminence, assert that

Explanation of right cardiac and venous distension

Now the most important and the most debated question regarding the theory of apnœa is this: What is the explanation of the great distension of the right cavities of the heart and of the systemic venous system? Two conflicting explanations of the phenomena have been given. First, those physiologists who find that, when the chest is opened soon after death from apnœa, the extreme engorgement of the right cavities is associated with comparative emptiness of the left, maintain that this contrasted condition of the two sides of the heart can be explained only by some impediment to the passage of the blood through the lungs. On the other hand, Dr. Sanderson and those who agree with him in affirming that after death from apnœa 'it is always found that all the cavities of the heart are filled to distension,' entirely ignore the evidence of obstruction in the lungs, and endeavour to explain the phenomena by the theory that the contractile power of the heart's walls is gradually impaired, and ultimately destroyed, by the circulation of venous blood through its tissues. Dr. Sanderson says: 'The heart itself being weakened by defect of oxygen, the organ soon passes into the state of diastolic relaxation, before described. Its contractions become more and more ineffectual until they finally cease, leaving the arteries empty, the veins distended, its own cavities relaxed and full of blood.'

Dr. Sanderson's

Dr. Foster's

Dr. M. Foster, to the same effect, says: 'The cardiac tissues, which at first probably are stimulated, after a while become exhausted by the action of the venous

the left cavities of the heart are distended immediately after death, when the contrary can be proved in a few minutes by so simple an experiment as that above recorded.

blood, and the strokes of the heart become feebler as well as slower.'

Professor Yeo's

And again Professor Gerald Yeo affirms that 'the cardiac muscle being exhausted for want of oxygen, is unable to pump the blood out of the veins or to empty its own cavities.'

An old theory revived

This explanation of the cessation of the heart's contraction is the revival of a theory long ago propounded by Bichat, the inadequacy of which to explain some of the well-known phenomena of apnœa will presently be shown.

I propose now to indicate briefly the successive steps by which what I believe to be the true explanation of the distension of the right side of the heart has been arrived at.

Dr. Reid's researches

One of the most instructive papers on the subject of apnœa was published many years ago by Dr. John Reid,[1] who was the first to discover and publish the fact that there is, for a time, an increase of pressure in the systemic arteries when, in consequence of suspended breathing, venous blood passes into those vessels. He expressed his belief that the subsequent rapid fall of the blood pressure was the result of a diminished flow through the lungs, the impediment—as he supposed—in the pulmonary capillaries being also the cause of the distension of the right side of the heart and venous system. With reference to the hypothesis which as-

[1] See his paper 'On the Order of Succession in which the Vital Actions are arrested in Asphyxia,' which was first published in 1841, and republished in his collected *Physiological, Anatomical, and Pathological Researches*, 1848. In this paper Dr. Reid gives a complete history of the attempts which had been made by previous observers to explain the phenomena of so-called asphyxia.

sumed that the circulation is arrested in the capillaries, it should be borne in mind that Dr. Reid's paper was published long before the function of the arterioles had been discovered by the researches of Bernard, Brown-Séquard, and others.

As his experiments were conducted, the observation of the blood pressure was somewhat interfered with by the sudden variations which resulted from the convulsive strugglings of the suffocated animals.

Mr. Erichsen's experiments

Mr. Erichsen[1] subsequently, in an elaborate series of highly instructive experiments, got rid of the disturbing element of muscular contraction by pithing the animals, which were then kept alive for a time by artificial respiration. Mr. Erichsen by this means obtained results which were strictly in accordance with those of Dr. Reid; the main points being, that with the suspension of the respiration and the consequent passage of black blood into the systemic arteries, there is, for a time, an increased blood pressure in those arteries, the result of some resistance in the terminal vessels; then after a period of two or three minutes there is a rapid decrease of pressure, in consequence of the impeded and finally arrested circulation through the lungs. He maintained that the existence of obstruction in the lungs is sufficiently proved by 'the tension of the pulmonary artery and the accumulation of blood in the right cavities of the heart, as compared with the state of the pulmonary veins and the left cavities of the heart.'

Why right heart is sometimes empty

When, in very rare exceptional cases, the right cavities are found comparatively empty after death, this is with-

[1] *Edinburgh Med. and Surg. Journal*, January 1845.

out doubt a result of one or more large veins having been wounded in opening the chest.

Dr. Sutton, with reference to the great engorgement of the right cavities of the heart and the systemic veins which is usually found after death in the collapse stage of cholera, says ('London Hospital Reports,' vol. iv. p. 493): 'When the large veins of the neck were accidentally wounded as in the act of raising the sternum, the blood escaped from the veins, and the right ventricle was emptied in two or three minutes;' and he refers to one case in which this actually occurred from the wounded jugular veins (p. 448).

In the 'British Medical Journal' (May 7, 1870) Mr. Worley has recorded an interesting case of clot in the pulmonary artery which had caused death after ten minutes of dyspnœa, screaming and struggling. Although a clot was found 'completely filling' the pulmonary artery, *all* the cavities of the heart were quite empty. As from the mode of death it is quite certain that the right cavities must have been greatly distended during the last few minutes of life and at the moment of death, the only probable explanation of their being found empty, is that one or more large veins had been wounded in opening the chest; and it is obvious that the disgorgement of the cavities would be rapid in proportion to their previous fullness and the consequent tension of their walls. The spirting of blood from a wound of an engorged vein after death from apnœa, mentioned by Drs. Sanderson and Foster, must soon empty the venous side of the heart, or at any rate greatly reduce the amount of blood contained therein. It is a well-known fact that when the right ventricle is much distended, the blood

regurgitates through the imperfectly closed tricuspid orifice.

Experiments on young puppies

Mr. Erichsen in the course of his elaborate investigation made some interesting observations on those young mammals which have the eyes closed at the time of birth, and in which for some days after birth the foramen ovale and ductus arteriosus remain open. The experiments of Buffon, Legallois, and Edwards showed that these young animals survive the exclusion of air for a much longer period than the same class of animals a few days older, in which the foramen ovale and ductus arteriosus are closed. In the former class it is evident that death would not occur from obstruction of the pulmonary circulation, the two sides of the heart communicating in a more direct way than through the pulmonary vessels. Accordingly, not only do these animals live longer after the exclusion of air, but the amount of black blood on the two sides of the heart is found after death to be nearly equal.

Mr. Erichsen describes the following experiment: 'A puppy four days old was strangled; struggles ceased in nine minutes. The thorax was then laid open and the heart exposed; the blood that flowed from the cut mammary and intercostal arteries was perfectly black. At the expiration of an hour and twenty minutes the ventricles had ceased to act; the auricles continued to act for nearly three hours and a half. On examination about two hours after the action of the heart had ceased, it was found that the right cavities and the pulmonary artery were full of black blood, but by no means distended, certainly not so much as in older animals that have been asphyxiated. The left cavities also contained

a considerable quantity of black blood, but not quite so much as the right side; there was likewise some blood in the aorta. The ductus arteriosus and foramen ovale were quite pervious.'

'Another puppy of the same age was strangled; the spasmodic movements continued for sixteen minutes; the chest was opened four hours after the trachea had been tied. All movement of the heart had then ceased, and the same appearances, as nearly as possible, were found as in the former case, the difference in the quantity of blood in the two sides of the heart being but small. The same experiment repeated on other puppies of the same age was attended with similar results.'

The unconsciousness and the convulsions are, no doubt, results of the circulation of venous blood through the nervous centres, while the principal cause of the arrest of the circulation in young animals with a patulous foramen ovale and ductus arteriosus appears to be, as Mr. Erichsen says, 'the gradual diminution of the force of the heart's contractions in consequence of the circulation of black blood through its muscular fibre.'

Dr. David Williams

Dr. Reid quotes an interesting experiment made by Dr. David Williams, of Liverpool, who states that, 'When the chest is laid open immediately after the trachea is tied during the acme of inspiration, the pulmonary veins soon become empty, while the pulmonary artery continues full.[1] He concluded that the blood is obstructed in its passage through the lungs, and that the obstruction arises from a deprivation of pure atmospheric air.'

In connection with this observation it is interesting

[1] Dr. Williams's paper is entitled 'On the Cause and Effects of an Obstruction of Blood in the Lungs.' *Edin. Med. and Surg. Journal*, xix. 524, 1823.

Effect of readmission of air

to note one result of Dr. Reid's experiments as recorded in the following statement: 'When atmospheric air was allowed to enter the lungs after the mercury had sunk to the lowest level in the instrument' (i.e. the mercurial dynamometer, in a systemic artery), 'no sooner had the air acted upon the blood in the lungs, than the mercury instantly sprang up several inches; and when the blood had become more perfectly arterialised, it again stood lower, and the range was more limited.'

This interesting and instructive observation has often been verified by subsequent experimenters, and, as I shall presently show, it is verified in every case of rapid recovery from anæsthesia by nitrous oxide gas.

Dr. Rutherford's experiments

But the most complete and entirely satisfactory experiments tending to throw light upon the phenomena of apnœa are those which have been performed upon animals under the paralysing influence of curara. I am indebted to my friend and former colleague Dr. Rutherford, now the distinguished Professor of the Institutes of Medicine in the University of Edinburgh, for the opportunity of witnessing some experiments performed in 1873, the results of which I will endeavour as briefly as possible to describe. I may state at once that the results, although in some respects more complete and conclusive than those obtained by Dr. John Reid and Mr. Erichsen, are entirely in accordance with their observations.

Into the trachea of a dog a tube was tied and connected with a bellows for the performance of artificial respiration. The voluntary muscles were then paralysed by the injection of curara, and the animal was kept alive by artificial respiration. The sternum and por-

tions of the ribs were removed and the pericardium was opened, so as to expose the whole of the anterior surface of the heart. One common carotid artery was divided, and a dynamometer-tube connected with a mercurial kymograph was introduced into the proximal end. In making all these preparations, much time and labour and great skill were required. Artificial respiration was now suspended, and immediately the colour of the left auricle changed from crimson to purple, the dark venous blood showing through the thin walls of the auricle, and the kymograph indicated a continuous increase of pressure in the systemic arteries. The variations of arterial pressure were registered by a pen on a revolving cylinder. After the increase of pressure had continued for about a minute, the *left* cavities of the heart became much distended; the auricle, in particular, became expanded into a tense globular ball with a smooth surface (fig. 1, p. 24). In the next period, the pressure in the arteries began to fall, and, about the same time, the right cavities of the heart, which had hitherto remained of the normal size and form, began to expand, while the distension of the left began rapidly to subside. Meanwhile, the right cavities became more and more distended, and now the *right* auricle assumed the appearance of a tense globular ball, while the left auricle had become nearly empty and flaccid. The right ventricle also became so distended that it projected above the level of the left (fig. 2, p. 25).

Effect of readmission of air

This was the condition of the heart's cavities when the animal died by the final arrest of the circulation through the lungs; but more than once, when the circulation was nearly at a standstill, artificial respiration

was resumed, and then all the phenomena rapidly changed. The blood, which had accumulated in the pulmonary artery and the right side of the heart, at once passed freely through the lungs, the distension of the right cavities of the heart subsided, and the systemic arterial pressure became first excessive, while the blood was partly venous, and then normal, when the blood

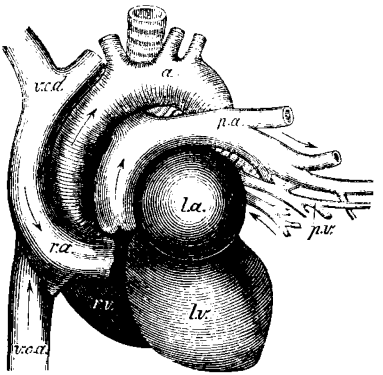

FIG. 1.—Represents the distension of the left cavities of the heart and aorta in the first stage of apnœa (asphyxia). *l.a.* left auricle. *l.v.* left ventricle. Both greatly distended, the former like a smooth indiarubber ball. *a.* aorta distended. *p.a.* pulmonary artery. *p.v.* pulmonary vein. *r.a.* right auricle. *r.v.* right ventricle. *v.c.d.* descending vena cava. *v.c.a.* ascending vena cava. The right cavities of the heart, the pulmonary artery, and the systemic veins are in a state of normal fullness. The right ventricle is partly overlapped by the distended left.

had become thoroughly oxygenised, and its passage through the terminal vessels was no longer resisted.

We have now to consider the minute mechanism of the process by which first the systemic and then the pulmonary circulation is impeded after the respiration is suspended.

Theory of Alison and Reid.

The theory originally propounded by Dr. Alison, and afterwards accepted by Dr. Reid and other physiologists, was that when respiration has been suspended the

blood is impeded in its passage through the *capillaries* and finally arrested there. As regards the lungs, their condition immediately after death from apnœa affords conclusive evidence that the blood has *not* been arrested in the capillaries. If the blood stagnated in those vessels, capillary engorgement would be a necessary and constant result, whereas, on the contrary, while the trunk and

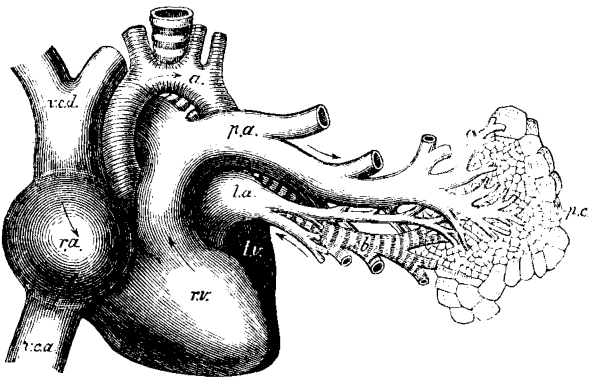

FIG. 2.—Represents the distension of the right cavities of the heart, of the pulmonary artery, and the large systemic veins in the final stage of apnœa (asphyxia). The letters have the same significance as in fig. 1. In addition, *p.c.* indicates the anæmic condition of the pulmonary capillaries. *b.* left bronchus. The right auricle and ventricle and the pulmonary artery are fully distended, the auricle having the form and smoothness of a distended ball, while the left cavities of the heart and the aorta are collapsed and nearly empty.

main ramifications of the pulmonary artery are distended with blood, the capillaries are bloodless, the lungs are usually pale, always light in weight, and in consequence of the anæmia of the capillaries the lungs collapse to an extreme degree when the chest is opened. The blood has evidently been arrested in the terminal branches of the pulmonary artery, just before it has reached the capillaries. These terminal branches, we know, are the muscular-walled arterioles, and their contraction affords

an adequate explanation of the arrest of the pulmonary circulation.

Engorgement of bronchial veins

Here it may be well to mention that in some cases the mucous membrane of the bronchi and the surface of the lungs are more or less cyanosed. This dark colour is due to a backward engorgement of the *bronchial* veins and capillaries, which share in the congestion of the whole systemic venous system. In some cases the congestion is so great that ecchymoses appear beneath the pleura.

It will be seen from the results of this experiment that the two sides of the heart during the process of apnœa are not fully distended at the same time. On the contrary, as soon as the distension of the right cavities commences, that of the left begins to subside; and by the time that the distension of the right side is complete, that of the left has entirely passed away.

The true theory

We are now in a position to give a rational physiological explanation of the facts revealed by Dr. Rutherford's most instructive experiment. Artificial respiration being suspended, unoxygenised black blood at first passes freely to the left side of the heart and the systemic arteries and capillaries. Arrived there, either by its direct stimulation of the muscular arterioles or, more probably, by a reflex influence through the vaso-motor nerves and centre, the arterioles are excited to contract, and by this action of the arterial stopcocks the blood pressure in the arterial trunks is increased and the left cavities of the heart become distended and dilated, as seen in the exposed heart of the living dog (fig. 1, p. 24).[1] The force which

[1] If respiration is suspended when the lungs are collapsed and airless, the pressure in the systemic arteries begins to rise immediately;

distends the left cavities in the first stage of apnœa is derived from the contraction of the right ventricle, and is thence transmitted through the pulmonary vessels. The circulation through the systemic arterioles is impeded, but not arrested; some black blood passes through the capillaries, and this black blood becoming more and more entirely deoxygenised, arrives through the veins at the right side of the heart and the pulmonary vessels. Reaching the pulmonary arterioles and capillaries, it excites there the same arterial contraction and resistance as had before occurred in the systemic vessels. The resistance offered by the contracting pulmonary arterioles, while on the one hand it tends to empty the left side of the heart and so to lessen the blood pressure in the systemic arteries, on the other it causes that great distension and dilatation of the right cavities, more especially of the auricle, which are invariably found to exist when the chest is opened soon after death from apnœa, and which, in Dr. Rutherford's experiment, was plainly seen to occur during the lifetime of the animal (fig. 2, p. 25). It would appear that, while the systemic arterioles *immediately* resist the passage of imperfectly aërated blood, the resistance offered by the pulmonary arterioles does not commence until the deoxidation of the blood has passed beyond a certain stage, and this resistance, again, is rapidly overcome by the readmission of atmospheric air to the lungs.

If the pulmonary arterioles began to contract, and so to resist the onward movement of the blood, as early as

but if the lungs are distended with air when respiration ceases, the rise of blood pressure is somewhat delayed in consequence of the presence of some oxygen within the lungs.

do the systemic arterioles, death from suspended respiration and circulation would be much more rapid than it actually is.

Some additional facts which were observed during the progress of this experiment are worthy of remark. It was noted that the increased arterial pressure, which commenced as soon as black blood began to pass into the systemic vessels, had existed for some seconds before the left auricle and ventricle began to dilate, and continued for some time after the dilatation of those cavities had reached its height; then, while the distension of these cavities persisted, the arterial pressure began to fall, and it was just at this time that the right cavities, which had heretofore retained their normal size and form, began to be distended and dilated.

The question arises, What was the immediate cause of the diminished arterial pressure which began while the left cavities were still distended? It might possibly be due to diminished contraction of the terminal arterioles, but this is not a probable explanation. It may be a result partly of over-distension of the left cavities of the heart lessening their contractile power. It is not unlikely too that the heart's contraction may be in some degree enfeebled by the circulation of black blood through its nutrient vessels, but this obviously does not explain the dilatation first of the left cavities and subsequently of the right: a phenomenon which can be accounted for only by excessive contraction, first of the systemic and then of the pulmonary arterioles.

Another probable concurring cause of the lessened pressure in the arteries at this period is, that the contraction of the pulmonary arterioles lessens the blood

supply to the left side of the heart before the right cavities begin to dilate, as we have seen that, in the earlier stage, the systemic arterioles contract and cause increased arterial tension before the left cavities become distended and dilated.

The subsequent rapid fall of pressure in the systemic arteries, and the comparative emptiness of the left side of the heart, are fully explained by the increasing impediment to the pulmonary circulation and the consequent scanty blood supply to the left heart and the arteries.

Inhalation of nitrogen

Some physiologists have suggested that the suspension of the respiratory movements might have much influence in impeding the pulmonary circulation; but Drs. Alison and John Reid proved that when an animal is made to breathe pure nitrogen gas, although the respiratory movements continue, the flow of blood through the lungs is arrested as speedily as when the chest is motionless. Dr. Reid [1] fixed a tube with a stopcock into the trachea of an animal. When the stopcock had been closed sufficiently long to cause a decided failure of the circulation, a large bladder provided with a stopcock and full of pure nitrogen was fixed in the tube in the trachea, when both stopcocks being opened, the failure of the circulation continued to increase. Then a bladder of the same size filled with atmospheric air was substituted for that containing nitrogen, when the circulation was rapidly renewed. Dr. Reid says: 'In this experiment the same mechanical movements of the chest which failed to renew the circulation of the blood through the lungs when nitrogen gas was inspired, rapidly effected that object when atmospheric air was

[1] *Physiological, Anatomical, and Pathological Researches*, p. 24.

permitted to enter the lungs, even when tried on the same animal subsequently to the failure of the nitrogen, and consequently at a more advanced stage of the process of asphyxia. This experiment was repeated several times, and, when the requisite care was taken to procure and employ pure nitrogen, invariably with the same result.'

A similar experiment is now daily performed upon hundreds of human beings who inhale nitrous oxide gas, which rapidly produces anæsthesia with a more or less complete arrest of the pulmonary circulation, while the respiratory movements continue. We will now set forth some particulars of this nitrous oxide experiment.

Nitrous oxide anæsthesia

Nitrous Oxide Anæsthesia.—In the phenomena of apnœa with the resulting rapid arrest of the circulation which occurs when nitrous oxide gas is inhaled as an anæsthetic, we have a very interesting confirmation of the results obtained by excluding atmospheric air from the lungs of animals; and, on the other hand, physiological experiments, and, above all, Dr. Rutherford's most instructive demonstration, enable us more completely to understand and interpret the facts of nitrous oxide anæsthesia. On several occasions I have availed myself of the opportunity afforded me by the courtesy of the staff of the Dental Hospital in Leicester Square to watch the phenomena which attend the inhalation of the gas, and I will now briefly describe them.

In most cases, during the first few seconds the pulse and the breathing are quickened, as a result probably of emotional excitement. In the next stage, the breathing becomes slow and shallow and the pulse slower, full and firm. Then, after a period which varies in different

cases from forty to eighty or ninety seconds, the pulse suddenly becomes almost, or even quite, imperceptible, the features become livid, the pupils are widely dilated, there is a state of general muscular rigidity; in short, all the phenomena of the first stage of an epileptic fit are present. The mouthpiece being removed, the morbid phenomena quickly pass away, the features regain their normal colour, the pulse returns, and for a few seconds has again a full and throbbing character, but quickly regains its normal condition.

Explanation of phenomena

The explanation of the phenomena appears to be sufficiently obvious. It is admitted on all hands that, at the temperature of the body, the nitrous oxide gives up no oxygen to the blood or the tissues. The gas becoming rapidly diffused and replacing the oxygen in the lungs and in the blood, black unoxygenised blood passes into the systemic arteries, and excites, through the vasomotor nerves and centre, contraction of the muscular arterioles. The resistance thus offered to the passage of unaërated blood through the terminal arteries explains the temporary fullness and tension of the radial pulse.[1] The partially unoxygenised blood, passing through the systemic capillaries, soon becomes so entirely deoxygenised as, when reaching the lungs, to excite contraction of the pulmonary arterioles. The resistance thus offered to the passage of blood through the lungs explains, on the one side, the systemic arterial emptiness with feebleness or even complete disappearance of the

[1] Some writers have asserted that the full pulse during the early stage of nitrous oxide inhalation is evidence of the direct stimulant action of the gas upon the heart; but since the exclusion of atmospheric air has precisely the same effect upon the pulse, that explanation of the arterial fullness and tension is evidently untenable.

pulse, and, on the other, the systemic venous fullness with lividity of the skin. The epileptiform condition is explained by the sudden and extreme diminution of the blood-supply to the brain, the blood at the same time being unaërated.

Rabbits killed by gas

If the inhalation were continued, death would occur from the complete arrest of the pulmonary circulation and consequent over-distension of the right side of the heart; and this is the mode in which death occurs when an animal is killed by the continued inhalation of the gas. In March 1875, my friend and former colleague Mr. Hamilton Cartwright assisted me to kill two rabbits with the gas. In both animals convulsions preceded death; and, the chest being opened immediately after death, the right cavities of the heart and the systemic veins were greatly distended with blood, while the left cavities and the aorta were comparatively empty and flaccid; the blood on both sides of the heart being equally black. The lungs were anæmic and collapsed to an extreme degree.

It will be seen that the phenomena observed during life and the appearances after death from the inhalation of the nitrous oxide gas are similar to those which result from suspension of the respiration in the human being and in the lower animals. (See fig. 2, p. 25.) The rapid renewal of the circulation with recovery of consciousness when atmospheric air is readmitted to the lungs is also identical with the results of experiments on animals.

Enlightened as we have been by Dr. Rutherford's demonstration of the condition of the heart's cavities during the successive stages of apnœa, we may be sure that if we could look within the pericardium of a

patient who is inhaling nitrous oxide gas we should see that, with the full, tense pulse of the first stage, the *left* cavities of the heart are distended, and that in the later stage of complete anæsthesia, with a vanishing pulse and livid features, the *right* cavities are fully distended, while the left are nearly empty.

It is evident from the many thousands of cases in which the gas has been inhaled without any grave mishap that in the hands of a skilled and careful operator little risk attends the employment of this anæsthetic ;[1] but it is also obvious that to a patient with a feeble, fat heart the distension of the right cavities which accompanies the disappearance of the radial pulse and the general lividity of the features must be attended with some degree of risk, and the danger must be increased when, the muscles of the trunk and limbs being convulsed, the pressure of the contracting muscles upon the veins drives the blood forcibly towards the right cavities of the heart, and so adds to their distension.

Cause of convulsions

I have been told by a gentleman who has had a large experience in the administration of the gas that on one occasion the patient was so violently convulsed that she was thrown from the chair on to the floor. The epileptiform convulsions which result from nitrous oxide must have for their main cause anæmia of the brain, consequent on the sudden and complete arrest of the pulmonary circulation. In these cases there can be no accumulation of carbonic acid in the blood. The convulsions therefore are analogous to those which

[1] The late Mr. Clover, in a letter to the *Lancet* (vol. i. 1876), stated that he had put to sleep more than 11,000 persons with the gas, without one fatal result.

Kussmaul and Tenner produced in rabbits by ligaturing the carotids and subclavians. (See the chapter on the Pathology of Epilepsy in my 'Medical Lectures and Essays.')

Theory of cardiac paralysis

Now, having given what I believe to be a true explanation of the phenomena of apnœa, I propose to criticise the theory which assumes that the arrest of the circulation is a result of the muscular tissue of the heart being poisoned and paralysed by the circulation of venous blood, both sides of the heart being, as is affirmed, equally distended after death. This statement as to the condition of the heart's cavities is in direct opposition to the observation of many very competent physiologists and pathologists, from Dr. John Reid onwards.

Distension of right cavities

As I have already stated, the great distension of the pulmonary artery, the right cavities of the heart, and the large systemic veins is a fact generally admitted. The only probable explanation of this great distension is that it is a result of an impeded passage of the blood through the lungs, and such an impediment would seem *à priori* to imply a deficiency of blood in the parts beyond, including the left cavities of the heart.

The lowering of the sluice gates, with which Professor Huxley compares the contraction of the arterioles, does not flood, but empties the irrigation canals beyond, and an enormous accumulation of blood on the venous side of the systemic circulation of necessity involves a corresponding deficiency on the arterial side.

So great is the distension of the right cavities that the blood spirts from a wounded vein as from an artery, and, as Dr. M. Foster says, 'it may frequently be observed that the beats recommence when the over-distension of the heart's cvaities is relieved by puncture of the great

vessels.'[1] This observation applies only to the right side of the heart, and it is inconsistent with the assumption that its contractions had ceased because its muscular tissue was paralysed by venous blood. The visible contractions of the right ventricle cease when its most vigorous efforts are unable to overcome the obstruction in front. The puncture of a vein allows the blood to flow backwards; thus the over-distension is relieved and the visible beats are resumed.

Bichat's theory

We have also seen that if in a case of apnœa the air be readmitted to the lungs soon after the heart has ceased to beat, the heart's action is quickly restored. Now this fact is inconsistent with the theory which I am combating. The theory is an ancient one propounded by Bichat. If it were a true theory, when once the circulation and the heart's action had been brought to a stop the arrest would be permanent and irremediable, and the disappearance of the pulse under the influence of nitrous oxide inhalation would be a final and a fatal disappearance. For the theory assumes that in order to renew the heart's beats aërated blood must reach the coronary vessels, an event which could not be brought about without the previous renewal of the heart's action. We are often reminded that *post hoc* is not always *propter hoc*, but no one requires to be told that *post hoc* cannot possibly be the cause of something which has gone before. Aërated blood reaches the capillaries of the

[1] Dr. John Reid has an interesting paper on the *Effects of Venesection in renewing and increasing the Heart's Action under certain Circumstances*. He found by experiments on animals that when the right cavities of the heart are much distended, as a result of asphyxia or other causes, opening the jugular vein allows a reflux of blood from the heart, by which its distension is lessened and its contractions are renewed.

heart *after* its action has been resumed and the circulation restored by the readmission of air to the lungs. The true interpretation of the phenomena in question appears to be that the readmission of air to the lungs causes, through nervous agency, relaxation of the pulmonary arterioles; the aërated blood then passes on to the left side of the heart and soon reaches the cardiac capillaries, but this is a consequence and not the cause of the renewal of the heart's beat.

Sir Thomas Watson

Sir Thomas Watson, commenting on Bichat's theory, says: 'There are two well-known facts which, on this theory, would be inexplicable—the comparative emptiness of the left side of the heart and the restoration of the suspended functions by the timely performance of artificial respiration. The air could never reach and revivify or depurate the venous blood stagnating in the capillaries of the heart.' [1]

The renewal of the heart's contractions by the readmission of atmospheric air to the lungs, and their renewal by the puncture of a distended vein, have this in common, that the beats, having ceased in consequence of over-distension of the right cavities, are restored by the relief of that distension; in the former case by removing the obstacle in front, and in the latter by permitting a backward escape of the blood.

Effect of an over-dose of curara

Dr. Sanderson gives very few particulars of the method of observation and experiment which convinced him that both sides of the heart are equally distended after death from apnœa. There is one method by which probably such a result might be brought about. It has been found that an over-dose of curara paralyses not

[1] *Lectures on the Principles and Practice of Physic*, 5th edit. vol. i. p. 72.

only the voluntary and respiratory muscles, but also the vasomotor nerves.[1] Paralysis of the pulmonary arterioles would deprive them of their power of regulating and arresting the flow of blood through the lungs. After the death of an animal thus poisoned an equal or nearly equal amount of blood might be found on the two sides of the heart; a result, though brought about in a different way, similar to that which Mr. Erichsen observed when a newly born puppy had been suffocated (ante, p. 20).

Dr. Sanderson's statements reviewed

One of Dr. Sanderson's statements is not quite consistent with the generally accepted views as to the effect of contraction of the arterioles. He says, with regard to the contraction of the small arteries: 'The immediate consequence of this contraction is to fill the venous system.' But surely the immediate effect of contraction of the small *systemic* arteries is to lessen the onward current of blood into the veins and to cause a backward accumulation in the arterial trunks, with an increase of pressure there. The contraction of the arterioles is equivalent to the arrest or diminution of the running stream by 'lowering the sluice gates' which regulate the supply to a system of irrigation canals.

On the other hand, the immediate result of contraction of the *pulmonary* arterioles is to fill and distend, first the pulmonary artery, then the right side of the heart, and lastly the systemic veins.

With reference to the enlargement of the heart in the last stage of asphyxia, Dr. Sanderson says this is due to

[1] In the lecture before referred to, Dr. Rutherford says: 'The dose of curara should be just sufficient to paralyse the voluntary muscles. If the dose be excessive the vasomotor nerves are also paralysed.' And he indicates the suitable dose for animals of different kinds and sizes (*Lancet*, February 17, 1882, p. 213).

Dr. Sanderson criticised

'the lengthening of the diastolic interval and to the quantity of blood contained in the great veins, which, in fact, are so tense that if cut into they spirt like arteries.' It would, I think, be more correct to say that the lengthening of the diastole, the enlargement of the heart, and the tense fullness of the large veins are all results of the obstructed pulmonary circulation. The engorgement takes a retrograde course from the resisting pulmonary arterioles; so that the distension of the heart precedes that of the venous trunks and is not caused by it.

Again, this high tension of the veins, which also exists in the walls of the heart, is not consistent with Dr. Sanderson's statement that 'all the heart's cavities' (it would be better to say the heart's *walls*) 'are relaxed.' In fact, so tense are the walls of the right heart that the blood is forcibly ejected when they are punctured; and this high tension is an index and a result of the force with which the contraction has continued until the circulation has been finally arrested by the obstruction in the lungs.

Dr. Sanderson says: 'It will be seen that no very obvious change in the heart and great vessels will occur until the last stage (corresponding to what I have called the second stage of asphyxia) is approached.' From this statement it is evident that his experiments have not rendered visible to him the great dilatation of the left cavities, so clearly demonstrated by Dr. Rutherford as occurring during the first stage, concurrently with the high systemic arterial tension, and preceding the final dilatation of the right cavities.

If an over-dose of curara has deprived the systemic

arterioles of their vital contractility there will, of course, be no arterial resistance and no distension or dilatation of the left cavities of the heart during the first stage of apnœa. Dr. Rutherford was careful to avoid this source of error.

I have thus critically examined Dr. Sanderson's theory of asphyxia in order to show that his statements are not so consistent, either with each other or with the generally accepted doctrines of physiology, that we should be called upon to accept without question a doctrine which cannot be reconciled with the careful observation of some of the ablest and most trustworthy men who have investigated the complex phenomena of apnœa.

Impediment in lungs ignored

It is very remarkable that the conclusive evidence of impeded circulation through the lungs in the later stages of apnœa, which the earlier writers on the subject so clearly indicated, should be entirely ignored by those physiologists who believe that the circulation is arrested because the heart is paralysed by venous blood.

Summary of Conclusions

The facts and inferences relating to apnœa which appear to be unquestionably established are the following:

When the chest is opened immediately after death the right cavities of the heart are distended with blood, while the left contain comparatively little blood.

Dr. Rutherford's experiments show that, with the increase of blood pressure in the systemic arteries during the first stage of apnœa, the left cavities of the heart are

greatly distended, and this distension entirely disappears while the right cavities are becoming overfilled.

The final distension of the right cavities, with comparative emptiness of the left, is the result of an impediment to the passage of blood through the lungs.

The only probable explanation of the obstructed pulmonary circulation is that which attributes it to extreme contraction of the pulmonary arterioles, consequent on complete deoxygenation of the blood.

The phenomena of apnœa are characterised by two well-defined stages. In the first stage there is systemic arterial resistance, with resulting distension of the left side of the heart. In the second stage there is pulmonary arterial resistance, with distension of the right cavities and comparative emptiness of the left.

The statement that both sides of the heart are equally distended immediately after death is incorrect, and the theory of cardiac paralysis, which is based upon that erroneous assumption, cannot be maintained.

The following experiment, performed by a non-professional friend who takes a deep interest in physiology, would, even by itself, suffice to disprove the above statement. A guinea-pig having been chloroformed, the chest was opened while the heart was still beating; when, as a result of the apnœal condition, the right cavities became greatly distended while the left were collapsed and empty. Here, again, rigor mortis had no share in the result.

SECTION III

A COMPARISON OF VARIOUS PATHOLOGICAL PHENOMENA WITH THOSE OF APNŒA

I SHOULD not have deemed it necessary or desirable to discuss at so great length the subject of apnœa but for the fact that a true theory of the phenomena affords great assistance in the solution of some most interesting and important pathological problems.

Choleraic collapse

For instance, fig. 2, p. 25, which represents the condition of the heart's cavities in the final stage of apnœa, is also a correct representation of the appearances found after death during the collapse stage of Asiatic cholera.

The distension of the pulmonary artery and the right cavities of the heart, indicative of an impeded circulation through the lungs, during the stage of collapse was first described by Dr. Edmund Parkes, though he failed to explain the phenomena.[1]

If the following extract from Dr. Parkes (on 'Asiatic or Algide Cholera') were severed from the context it might be supposed to refer to cases of apnœa: 'The right side of the heart and the pulmonary arteries were generally filled, and in some cases distended, with blood;

[1] See the chapter on 'Epidemic Cholera' in the author's *Medical Lectures and Essays*.

the left side and the aorta were generally empty or contained only a small quantity of dark blood. The inference which was drawn from the state of the cavities in the greater number of cases was that the right side had continued to receive blood till, in some cases, it became full and even distended, while the left side received little or no blood, but had continued to contract, in some cases even violently, upon the last drop of blood which had entered it.' Again: 'The conditions of the heart and lungs seem to point out unequivocally that in cholera the blood does not pass [freely] through the lungs.'

Theory of post-mortem distension

It has been suggested that the great distension of the right side of the heart and the venous system is the result of a post-mortem movement of the blood, and this notwithstanding the evidence that such distension exists during life, and notwithstanding that, as in cases of apnœa so in cases of choleraic collapse, the sooner the inspection is made after death the greater is the distension found to be. This suggestion, for its unreasonableness, is about on a par with the theory that the left ventricle is emptied after death from apnœa by rigor mortis (see ante, p. 11).

Effect of venesection

The great distension of the right side of the heart and the systemic venous system during choleraic collapse is associated with lividity of the lips and skin and with a sense of oppression and pain in the region of the heart. In the chapter on Epidemic Cholera ('Medical Lectures and Essays,' p. 83) I have quoted several cases in which great and permanent relief from these distressing symptoms has been afforded by venesection.

We have seen (ante, p. 34) that when in cases of

apnœa the over-distended heart has ceased to contract in consequence of obstruction in the pulmonary arterioles, the beats may often be renewed by allowing blood to escape from a punctured vein. In like manner, without doubt, the surprising relief which has often been afforded by venesection during choleraic collapse is explained by its lessening the engorgement of the right heart and the veins consequent on the impeded flow of blood through the lungs.

Theory of collapse

Many years since I publicly expressed my conviction that the arrested circulation through the lungs is the result of contraction of the pulmonary arterioles, excited by the choleraic virus[1] in the blood. Most of those who have opposed this theory have done so in ignorance of the forces which are concerned in maintaining the circulation. They have been unable to appreciate the fact that the impediment caused by the simultaneous contraction of all the pulmonary arterioles is greater than the most vigorous contractions of the right ventricle are able to overcome. If such critics would first make themselves acquainted with the physiology of the circulation, and then study the phenomena of nitrous oxide anæsthesia, they would learn how speedily the flow of blood through the lungs may be arrested by the contraction of the pulmonary arterioles, and they would no longer attempt to explain the acknowledged impediment to the pulmonary circulation in choleraic collapse by the untenable hypothesis that the blood is thickened by loss of water, and is consequently arrested in the *capillaries*.

[1] The contraction of the pulmonary arterioles, excited by the choleraic poison, is analogous to the spasm of the glottis which results from the inhalation of irritating gases or vapours.

One fact alone is sufficient to refute this hypothesis—namely, that the pulmonary capillaries, instead of being choked by thickened blood, are as bloodless as they are after death from acute apnœa.[1] Although, in consequence of the anæmic condition of the capillaries, the lungs are very light in weight and collapse to an extreme degree when the chest is opened, they often present the same cyanosed appearance with congestion of the bronchial mucous membranes and ecchymoses beneath the pleura, as we have seen to occur after death from apnœa (see p. 26). These appearances, as we have before explained, are the result of retrograde engorgement of the bronchial veins and capillaries, consequent on the obstruction in the pulmonary vessels.

Retrograde engorgement of bronchial vessels

It is both interesting and instructive to compare the symptoms of choleraic collapse with those of spasmodic asthma. The general appearance of a patient during a severe paroxysm of asthma is, in many respects, very like that of one in the collapse stage of cholera.

Cholera and asthma compared

Dr. Hyde Salter, in his masterly treatise on asthma, says:[2] '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

[1] I have elsewhere explained that the blood-thickening during the collapse stage of cholera is a consequence, and not the cause, of the impeded pulmonary circulation; and I have also shown that a similar blood-thickening, from the same cause, occurs in cases of prolonged partial apnœa (*Medical Lectures and Essays*, pp. 42, 142). So great is this tendency to blood-thickening, as a result of blood stasis, that in cases of prolonged choleraic collapse or prolonged partial apnœa the pulmonary artery, the right cavities of the heart, and the large veins often become more or less obstructed by fibrinous coagula.

[2] Page 72, 2nd edit.

and blue and shrunk; I have known the body deathly cold and resist all efforts to warm it for four 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 with sweat, combined with the duskiness 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 dyspnœa; it is so feeble sometimes that it can hardly be felt.'

The resemblance between some of the most striking symptoms of the asthmatic paroxysm and the collapse of cholera is obvious. What, then, is common to these two forms of collapse?

In both asthma and cholera the flow of blood through the lungs is impeded by the contraction of the pulmonary arterioles, and this is the immediate cause of the collapse in both diseases.[1] In cholera the contraction of the arterioles is excited by the poisoned blood in the vessels; in asthma it is a result of the partial apnœa occasioned by spasm of the bronchi. In cholera there is a primary asphyxia,[2] the result of spasm of the pulmonary arterioles, and a secondary apnœa, consequent on the scanty stream of oxygen-bearing arterial blood that reaches the tissues; in asthma there is a primary apnœa, the result of bronchial spasm, and a secondary asphyxia, consequent on

[1] In the chapter on 'Cholera' in *Medical Lectures and Essays* (p. 121) I have quoted several cases in which all the symptoms of choleraic collapse, except the gastro-intestinal discharges, were caused by a fibrinous plug in the pulmonary artery.

[2] For the definition of *asphyxia* and *apnœa*, see p. 7, note 1.

the impeded flow of unaërated blood through the lungs. In both forms of disease the symptoms of collapse may speedily be removed for a time by measures which relax the primary spasm—in asthma by the inhalation of chloroform, which overcomes the bronchial spasm; in cholera by the injection of a hot liquid into the veins,[1] which quickly reaching the lungs, relaxes the arterial spasm.

Hæmoptysis from retrograde engorgement of bronchial vessels

In the apnœa of asthma we have evidence of a retrograde engorgement of the bronchial system of vessels in the occasional occurrence of bronchial hæmoptysis, and in the more constant occurrence of bronchial mucous expectoration, which usually is copious and prolonged in proportion to the intensity and duration of the previous paroxysm.

Dr. Hyde Salter, in the first edition of his book, attributed the hæmoptysis of asthma to rupture of the *pulmonary* capillaries; but in the second edition, with the truth-loving candour of a physiologist and a physician, he adopts the explanation which I had given, and attributes the hæmoptysis to a retrograde engorgement of the *bronchial* veins and capillaries, consequent on the impeded flow of blood through the pulmonary arterioles. After explaining my theory he says (p. 89, note): 'I believe he is perfectly right; I believe he has solved the difficulty, and his solution satisfactorily explains to my mind not only the source of apnœal hæmoptysis, but, what I never could well understand before, the invariable sequence of bronchial mucous exudation upon any form of protracted partial apnœa; for that which would produce bronchial hyperæmia, even though passive, would

[1] See the chapter on 'Epidemic Cholera' in *Medical Lectures and Essays*, p. 88.

necessarily produce an increase of the bronchial secretion.'

Variations of the Pulse.—Without a competent knowledge of the physiology of the circulation it is impossible to interpret correctly many modifications of the pulse. The two physical forces upon which mainly depend the volume and power of the pulse are the contractions of the left ventricle at one extremity and those of the arterioles at the other. But another important factor is the volume of blood in the systemic arteries. For instance, the small and feeble pulse in the collapse stage of cholera, and during a severe paroxysm of spasmodic asthma, is the result of the scanty stream of blood which is transmitted through the lungs to the left side of the heart.

The rapid change in the character of the pulse which occurs during the process of nitrous oxide anæsthesia I have already described and explained (see ante, p. 30).

Pulse of apnœa

When, as a result of disease of the respiratory organs, imperfectly aërated blood passes into the systemic arteries the pulse is always modified.

Some time since I was attending, with two other practitioners, a case in which a copious effusion into both pleuræ caused great distress of breathing. The pulse, which had been rapid, small, and feeble, was found at our last consultation to be less frequent and with more volume and power. One of my colleagues thought this a favourable change, but I did not; for I noticed that the finger nails and the lips were blue, and I concluded that the fuller and slower pulse was the result of contraction of the systemic arterioles, excited by the

circulation of venous blood. The patient died a few hours after our visit.

Acute Bright's disease

Most valuable information may be obtained from an intelligent study of the pulse in the various stages and forms of Bright's disease. In cases of acute nephritis, with a scanty secretion of urine and consequent uræmia, the pulse is usually full and tense, a result of contraction of the systemic arterioles, excited by the impure blood. The fullness and tension pass away when the excretory function of the kidneys has been restored.

Chronic Bright's disease

The long-continued uræmic condition which results from chronic degeneration of the kidneys, and especially from that form of disease which results in the small red granular kidney, is attended with very remarkable changes in the circulatory system. The radial pulse is very full and tense, the large arteries are thickened and tortuous. There are the physical signs of hypertrophy of the left ventricle of the heart—namely, extended area of cardiac dullness on percussion, the apex below and external to its normal position, a strong heaving impulse, with reduplication of the first sound and accentuation of the second sound over the aorta. There is evidence, then, of high arterial tension. Some years since I was led to search for the cause of this condition by the following considerations: Hypertrophy of the left ventricle without disease of the valves or of the large arteries—a fact first made known by Dr. Bright many years ago—is a result, as he suggested, of some impediment 'in the minute subdivisions of the vascular system.' Reasoning from analogy, I thought it probable that the impediment is caused by the contraction of the arterioles excited by impure blood, and further, that their long-continued over-

Hypertrophy of arterioles

action would result in hypertrophy of their muscular coat, corresponding with the cardiac hypertrophy. This led me to search for and to find the hypertrophy which I had anticipated in the arterioles of every tissue that I examined, in the kidneys, intestines, skin, muscles, and pia mater.[1]

The prolonged over-action of the arterioles has registered itself in a conspicuous hypertrophy of their muscular coat. No kymograph could afford a more certain indication of excessive contraction of the arterioles, with resulting high arterial tension.

Erroneous theories

I have the satisfaction of finding that the existence of this hypertrophy of the muscular coat of the arterioles is now very generally acknowledged, but an unphysiological interpretation of the facts has sometimes been suggested. Some pathologists, ignoring the fact that the contraction of the arterioles tends to impede the flow of blood, assert that they become hypertrophied in their active efforts to aid the heart in propelling the blood through the resisting capillaries. Another suggestion is that there is some unexplained obstruction in the *capillaries*, and that the arterioles become hypertrophied to enable them to bear the strain to which they are subjected between the obstructing capillaries in front and the hypertrophied left ventricle behind. Such theories as these may be left to die a natural death.

There is no evidence that the capillaries, unless when plugged by coagula, have any power to impede the onward movement of the blood.

Large arteries tortuous

The tortuosity and thickening of the larger arteries in these cases is a result of the excessive strain to which

[1] See *Medical Lectures and Essays*, p. 694.

they are subjected between the hypertrophied left ventricle and the resisting arterioles. It is probable, too, that the contact of the impure blood may tend to cause degeneration of the arterial walls, as a result of which they are more liable to be ruptured by the high pressure to which they are subjected.

Cerebral hæmorrhage

One of the most frequent and most disastrous results of the excessive strain upon the arteries in cases of granular kidney is the rupture of a cerebral artery. The powerful left ventricle then forces the blood through the torn vessel into the brain tissue, the result being a rapidly fatal sanguineous apoplexy.

Relation between albuminuria and arterial tension

Attempts are sometimes made to explain cases of albuminuria by increased vascular pressure. It is highly probable that excessive pressure on the Malpighian *capillaries* might cause a transudation of albumen through their walls, but it should be borne in mind that the tension and pressure within the *arteries* is no measure of the pressure within the capillaries in front. The arterial tension is the result of contraction of the arterioles, and this contraction lessens the pressure within the capillaries, so that, other conditions being the same, there should be an inverse relation between arterial tension and intracapillary pressure. The albuminuria which occurs as a temporary condition soon after assuming the erect posture in the morning and before food has been taken is probably due to defective contraction of the renal arterioles, in consequence of which there is increased pressure upon the walls of the Malpighian capillaries, with a resulting transudation of albumen. That high arterial tension is often associated with albuminuria is unquestionable, but it is also indisput-

able that in the class of cases in which arterial tension is developed in the highest degree—cases of contracted granular kidney—the amount of albumen is, as a rule, very much less than in cases of large white kidney, which, during the greater part of their progress, are unassociated with increased arterial tension.

Venous engorgement

There is reason to believe that a retrograde venous engorgement, the result of obstructive cardiac or pulmonary disease, may so distend the Malpighian capillaries as to cause albuminuria, a result similar to that obtained by a ligature on the renal vein.

Raynaud's Disease

Raynaud's Disease.—A remarkable form of deranged circulation, which was originally described by Dr. Maurice Raynaud, under the name of Local Asphyxia and Symmetrical Gangrene of the Extremities, is, by the general consent of all who have written on the subject, attributed to spasm of the systemic arterioles. Raynaud's original treatise,[1] and his later researches, which were published in the 'Archives Générales de Médecine,' vol. i., 1874, have recently been translated by Dr. Thomas Barlow. This translation, together with a valuable appendix by Dr. Barlow, forms part of a volume of 'Selected Monographs' which was published last year by the New Sydenham Society. Referring to that volume for the detailed history of this remarkable affection, I propose here to do no more than indicate very briefly its general characters.

Essential features

The essential features of Raynaud's Disease consist in a local arrest of the circulation, with consequent coldness, blueness, and often dry gangrene of extreme parts, such as the fingers, the toes, the tip of the nose, and the

[1] *De l'Asphyxie locale et de la Gangrène symétrique des Extrémités.* Paris, 1862.

ears. There is also a remarkable tendency to symmetry in the parts affected; so that the disease usually, though not quite constantly, implicates corresponding parts on the two sides. Thus the upper or lower limbs, or all four together, may be bilaterally affected.

Recurring hæmaturia

In some cases the local gangrene is associated with that form of recurring hæmaturia which has been designated Intermittent Hæmoglobinuria. This feature of Raynaud's Disease has been discussed in much detail by Dr. Dickinson.[1] In most instances of this form of hæmaturia, although all the blood constituents are present in the urine, the corpuscles are usually disintegrated. Professor Murri[2] of Bologna believes that the blood corpuscles are disintegrated in the superficial vessels in which stagnation has occurred, and that arterial spasm is an essential factor in the disease. He holds that the corpuscles are broken up by the combined action of cold and carbonic acid in the stagnating blood; but the whole subject requires, and will repay, a more thorough investigation than it has hitherto been possible for it to receive.

Misuse of some terms

I venture to criticise Dr. Raynaud's use of the terms *local syncope* and *local asphyxia*. The arrested circulation, he says, 'can be compared to nothing better than syncope, in which the action of the heart is momentarily suspended.' But surely to compare the failure of the circulation resulting from suspended heart-action with an arrest which is acknowledged to be caused by arterial contraction is to confound phenomena which are essentially distinct. The proper term by which to designate

[1] *Miscellaneous Affections of the Kidneys and Urine.*
[2] *Dell' Emiglobinuria da freddo.* Bologna, 1880.

the arrested circulation in Raynaud's Disease is *local asphyxia*, a result of arterial contraction.

Raynaud, however, referring to the presence of unoxygenated venous blood in the implicated tissues, calls *this* 'local asphyxia.' The correct designation is *local apnœa*.[1]

Raynaud refers to the demonstration of the muscularity and the regulating function of the arterioles as 'one of the most beautiful discoveries of the century;' yet, with a confusing inaccuracy of language, he in several passages speaks of contraction of the *capillaries*, when, from the context, it is evident that he is referring to the arterioles. It is very desirable to avoid such a misapplication of terms as may tend to perpetuate erroneous ideas as to the function of the various parts of the vascular system.

The examples and illustrations which I have given of the derangements of the circulation which are associated with various forms of disease should suffice to show that a knowledge of the physiology of the circulation—a correct appreciation of the force which propels the blood onwards and of the force which impedes its passage and regulates its distribution—is essential for everyone who undertakes either to construct or to criticise a pathological theory.

[1] See the definition of the terms *Asphyxia* and *Apnœa* at p. 7, note 1. The function of respiration is not complete unless the blood and the tissues receive their full supply of oxygen. Apnœa, therefore, may result not merely from an interruption of the mechanical act of breathing, but also from any condition which impedes the due oxidation of the blood and the tissues.

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