REMARKS ON THE SITE AND MECHANISM OF THE CARDIAC MURMURS IN DEBILITY AND ANÆMIA.

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IN approaching this subject, it seems almost unnecessary to contend for the general proposition, that in debility and anæmia the heart shares in the morbid condition; that the inevitable result of this is relaxation of its muscular structure, and consequent dilatation of its cavities. Hope, Walshe, and Stokes all recognise this more or less fully. Parrot, writing in 1866, says: "In anæmia and chlorosis, Beau, who admits a dilatation with hypertrophy of the left heart, explains it by the impoverishment of the blood, which entails a relaxation (*relâchement*) of the tissues, and especially of the muscular substance of the heart. Bamberger, Friedreich, Wunderlich, Vogel, and Shark think that in chlorosis the fibres of the ventricular walls undergo relaxation, brought about by disordered nutrition, from diminution of the coloured corpuscles in the blood" (*Archives Générales de Médecine*, Août 1866, page 153). More recently, Schroetter says: "After certain febrile affections, there ensues, on account of the breaking-down of the muscular fibres of the heart into a molecular detritus, and the consequent relaxation of the muscular tissue, an acute dilatation of the heart. Doubtless, the changes which occur in chlorosis may be explained in this manner" (*Ziemssen's Cyclopædia of the Practice of Medicine*, vol. vi, p. 201). Hayden places anæmia at the head of the list of causes producing "primary dilatation" (*Diseases of the Heart*, Dublin, 1875, p. 558 *et seq.*). Dr. Balfour has so recently and so strongly advocated this, that it must be fresh in the memory of everyone. Dr. Goodhart evidently holds similar views. The further proposition, that dilatation may lead to regurgitation at the auriculo-ventricular orifices, occupies an equally positive place in the literature of cardiac disease.

Although the foregoing propositions led to the inevitable conclusion that murmur might result from the dilatation of debility, the principle was but falteringly applied. Hope (1839) seems to have been the first, in this country at least, to teach that murmur was, in certain pathological states, due to altered physical properties of the blood, to unfilled vessels and modified velocity of current, and that the cardiac murmur of anæmia was to be thus explained. From his time this doctrine has, with various modifications, been tenderly handed down from authority to authority. Skoda, in 1853, expressed some dubiety that watery blood should necessarily create murmur; but he was overridden and buried under the weight of other authorities. Parrot, in 1866, attacked the position from a clinical standpoint; but a belief, which had nearly completed the third decade of its existence, seems to have been but insignificantly affected by the effort. More recently, and in our own country, Dr. Balfour has assailed the position, offering an explanation which would place the murmurs partly in the domain of dilatation murmurs, and rob them, at least in the first place, of their classic site and mechanism.

It would occupy too much space to follow closely the history of the supposed site of the blood-murmur. Hope (1839) placed it at the aortic orifice; Hughes (1854) advocated the claims of the pulmonary artery; Parrot (1866) argued that it was a regurgitant murmur due to tricuspid insufficiency from dilatation of the ventricle; Walshe, with whom Hayden agrees, placed it in the aortic and pulmonary areas, and over the body of the heart, nearly as far to the left as the nipple-line; Balfour places it, in the first place, over the left auricular appendix, which, he holds, reaches the chest-wall to the left of the pulmonary artery, thereafter at the tricuspid orifice, and, finally, in the aortic and pulmonary arteries. In the more advanced cases, there are, according to this view, four murmurs; in fact, we have here a concretion of all former opinions, with the addition of the left appendix.

Dr. Macalister of Cambridge (*BRITISH MEDICAL JOURNAL*, October 28th, 1882) has lucidly and ably brought before the profession generally the work done by Ludwig and Hesse, demon-

strating the manner in which regurgitation may take place in a debilitated heart.

The aim of this paper is to try to remove the cardiac murmurs from their classic position of hæmic murmurs, and to explain them wholly by the facts of dilatation and regurgitation.

The cardiac phenomena in debility may be illustrated by a series of cases culled from those which have lately been under my care. Occasionally we have the opportunity of following the various stages in an individual case; such has not been my privilege recently.

CASE I. Pulsation in the Second Left Space; no Murmur.—A. H., aged 15, had been confined to bed for weeks suffering from Bright's disease, with obstinate hæmaturia. She was pale but plump. There was slight precordial tremor, becoming pulsation in the second left space, where it extended for over an inch from the sternal edge. There was marked accentuation of the pulmonary second sound, but no murmur anywhere. There was no venous pulsation, and no *bruit de diable*.

CASE II. Pulsation in the Second Left Space, and Systolic Impurity.—J. R., aged 18, had evidence of commencing phthisis at the right apex, and was subject to recurring and slight hæmoptysis, with rise of temperature. After one of these attacks there was precordial pulsation, visible, when lying down, from the fifth to the second left space. In the second space the pulsation was distinctly divided into two parts; the inner extended for a quarter of an inch to the left of the sternum, and conveyed the idea of continuity with the pulsation in the other spaces, while beyond this there was a feebler, and seemingly separate, pulsation extending for one inch. The external jugular was visible, but did not oscillate. The pulmonary second sound was loudly accentuated, and the first sound in the pulmonary area was prolonged, and occasionally murmuring. In some days this passed off, only leaving a slightly accentuated pulmonary second sound.

CASE III. Pulsation and Murmur in the Second Left Space, only present after Exertion.—Thomas L., aged 11, was suffering from a mild attack of articular rheumatism. On the third day of treatment, the pulse was 80 and the temperature 99.2°. There was pulsation in the external jugular, and at the root of the neck. There was a slight and diffused heaving visible over the precordium. The apex could be felt in the fourth space, an inch outside the nipple. There was no murmur, but the pulmonary second sound was loudly accentuated and slapping in character. On slight exertion, either by rising up in bed and lying down again several times, or by taking several deep inspirations, followed by very complete expiration, the precordial heaving became more marked, and, especially during expiration, there was a marked pulsation in the second left space, about an inch from the sternum, and over this a loud but localised murmur was heard.

CASE IV. Bruit de Diable: Murmur in Second Left Space, and no Pulsation.—Miss G. H., aged 17, was anæmic, but plump; there was undulation at the root of the neck, and for some distance along the course of the internal jugular. There was a *bruit de diable*. There was no precordial pulsation; but a rough systolic murmur was audible in the second left space, followed by a loudly accentuated pulmonary second sound. The murmur entirely vanished even on incomplete inspiration.

CASE V. Murmur in Second Left Space: No Pulsation: Faint Apex-Murmur.—Miss F., aged 23, was very pale and chlorotic. The pulse was 100 to 120 (excited), empty, and jerking. The external jugular vein pulsated synchronously with the radial pulse, and the vein collapsed and disappeared between each systole. There was a *bruit de diable*. There was no precordial pulsation. There was a faint and localised systolic murmur at the apex, and a murmur of the same rhythm in the second left space, completely lost on partial inspiration. The pulmonary second sound was accentuated. When she was standing up, there was no pulse in the external jugular veins, and no murmur over the heart.

CASE VI. Pulsation in Second, Third, and Fourth Spaces: Systolic Murmur in Second Space.—James H., aged 13, was suffering from a mild attack of typhoid fever. He had a thin-walled chest, with marked interspaces. The pulse was 80, regular, and of fair force and volume. There was precordial pulsation in the second, third, and fourth spaces; in the second, it extended outwards for one inch and a half; in the fourth, it reached one inch beyond the nipple. There was a systolic murmur confined to the second space, and loudest over the outer part of the pulsation. The pulmonary second sound was accentuated. There was oscillation in the external jugular veins, and no *bruit de diable*. The leading changes which occurred during the progress of the case, were, that the apex-pulsation de-

scended to the fifth space, one inch outside the nipple-line; that the murmur disappeared, in a day or two, for one day, and then re-appeared; that it again vanished when the boy was at his worst, with a pulse of 60, very small and weak, and when the precordial pulsation was much less. As he improved, the murmur again developed, and finally vanished when the rehabilitation of the heart was evidenced by a stronger and more frequent pulse, by the venous pulsation being reduced to a mere flicker, by the apex retreating under the fifth rib, and by the area of pulsation in the second left space lessening in extent.

CASE VII. Slight Precordial Pulsation: Distension of Veins: Systolic Murmur over Body of Heart, and in Second and Third Right and Second Left Spaces.—Mrs. H., aged 33, was very pale and slightly chlorotic. She was examined lying down, the external jugular vein was not only full, but so distended that it quite stood out, and did not collapse during any part of the circulatory cycle; it, of course, pulsated. There was faint precordial pulsation visible in the fourth left space inside the nipple, and in the third space, one inch from the sternum. There was no apex-beat; but, over its normal site, there was a systolic murmur, lost one inch outside the nipple-line; it was louder over the site of pulsation in the fourth space; less loud in the third and second spaces; also audible over the sternum, from the level of the second space downwards; heard also in the second, right and in the third right space, for nearly two inches from the sternal edge, and not present over the manubrium sterni, or in the carotids. It was louder in the second right than in the second left space; and, in the third right space it gradually lessened in intensity as the stethoscope was carried outwards, until it could only be detected with the greatest attention as a very distant faintly blowing sound. The pulmonary and aortic second sounds were about equal in intensity, and neither of them loud. There was a venous murmur between the origins of the sterno-mastoid muscle, presenting two distinct periods of loudness during a cardiac revolution. The veins running down in front of the trachea pulsated visibly. In the erect posture, the external jugular was only marked by a thin blue line during ventricular systole, and no murmur could be heard anywhere over the precordia. This patient rapidly improved under appropriate treatment, the veins becoming much less engorged; the murmur disappearing first from the third, then from the second right space, and from over the sternum, until it was only present in the second, third, and fourth left spaces.

The sequence of phenomena revealed by the stethoscope is thus (1) accentuation of the pulmonary second sound, with perhaps systolic impurity in the second left space; (2) murmur in the second left space; (3) murmur in the third and fourth left spaces, in addition to the second; (4) murmur from about the nipple-line inwards over a triangular area, the left boundary of which is formed by a line drawn from below the nipple to a point in the second left space, one to two inches from the edge of the sternum, and often heard at the same time over the sternum adjoining this area; (5) murmur in the second right space ("aortic area"); (6) murmur in the third right space.

There are, of course, modifications of the first sound audible in the various regions, before the supervention of murmur, which herald its approach, and are quite appreciable; and the degree to which these modifications go on to murmur, and the appearance of murmur in the various positions referred to, often afford an accurate, ready, and speedy indication of the effect that treatment is producing. I say nothing here of precordial pulsation, which is too much affected by the physical qualities of the chest-wall in individual cases to be of much value. The evidence given by the various pulsations in the neck of the condition of the right side of the heart provides a most interesting and valuable study, but one which cannot at this time be entered upon. The effect of the recumbent position in allowing the development of a tricuspid murmur in cases of extreme debility, and occasionally in cases of organic disease at the left auriculo-ventricular orifice, has important bearings upon cardiac disease in general, which seem to have been largely overlooked.

I shall now endeavour to explain the clinical phenomena. The accentuation of the pulmonary second sound, if no lung affection be present, must be taken as indicating an abnormal accumulation of blood behind the mitral orifice. Whether this be due to the incomplete emptying of the left ventricle during its weakened systole, and consequent imperfect relief to the full auricle; or, from the first, due to a certain amount of regurgitation; or to both these causes, it is unnecessary to discuss here. The fact of accumulation of blood in the pulmonary circuit, including the left auricle, is sufficient for our present purpose, and is warranted by the evidence given by pulsation at the root of the neck, by the course of the external

jugulars becoming visible, and perhaps by pulsation appearing over the right ventricle, that a like accumulation is taking place in the right chambers of the heart and the large vessels leading to it.

With reference to the murmur in the second left space, it is necessary to consider, in the first place, where it is produced; and this investigation may, with all respect to the great ones who have given their authority to other views, be confined to a consideration of the claims of the pulmonary artery, and the left auricular appendix. The latter may be taken first. Hitherto, although the claims of the appendix have been somewhat warmly advocated, no *post mortem* observations seem to have been recorded in its support in this country, and only this would justify the confidence reposed in the future of the theory. So far, it has been based on the fact in normal anatomy that the appendix is seen to the left of the pulmonary artery, and on a belief that it has been seen overlying the base of the ventricles in cases of mitral stenosis; and, as the murmur attains its maximum intensity about one inch to the left of the sternum, it is contended that this point is to the outside of the pulmonary artery, and therefore over the appendix.

The fact of cardiac dilatation is accepted by the advocates of this theory; we therefore have only to learn, from *post mortem* observations, made with the heart *in situ*, the changes which occur in the relations of the various parts of the heart to the thoracic wall in this condition. It will, I submit, be found that in less degrees of dilatation the conus arteriosus occupies part of the second left space, that the same space is occupied by the pulmonary artery, and that the left limit of the vessel is from one to one and a half or two inches from the edge of the sternum. In greater degrees of dilatation, the conus arteriosus occupies the inner part of the second space for about two inches, and the origin of the pulmonary artery is under the second rib. In both degrees of dilatation the left appendix is invisible from the front, and is deeply buried behind the root of the pulmonary artery and adjoining ventricle. The following six *post mortem* observations are offered in support of this; five have been made lately; the other was somewhat accidentally preserved from a time when I was investigating this subject simply for my own satisfaction.

OBSERVATION I.—Mr. A., aged 65, died of general paralysis. He was a patient of Dr. Lockie, and the necropsy was made by me in his presence. The sternum and its attached cartilages were removed with care, so as to avoid disturbing the parts beneath; the pericardium was freely incised, and along with the overlapping lung drawn aside. After removing the intercostal muscles, the sternum and its cartilages were replaced, when it was seen that the second left space was occupied by the conus arteriosus and the pulmonary artery for one inch and a half, the junction of the two running diagonally across the space from above outwards and downwards. The left auricular appendix was invisible. The second right space was occupied by the appendix of the right auricle, which also lay under the sternum opposite this space. The aorta did not become uncovered by the appendix until it reached the cover of the second rib. In the third right space, the outer wall of the right auricle was fully one inch and a quarter from the edge of the sternum. The apex of the heart was opposite the fourth space and fifth rib. Again raising the sternum, the left wall of the pericardium was drawn outwards, so as to permit a view of the heart in partial profile, when part of the appendix, about the size of the tip of the little finger, was revealed nestling against the left postero-lateral aspect of the pulmonary artery; a line drawn from the second rib, where the cartilage had been severed from it, traversed a course of two inches before reaching this point. Both auricles contained fluid blood. All the chambers of the heart were relaxed and somewhat dilated. The valves were healthy. The muscular fibres showed various degrees of fatty degeneration when examined microscopically.

OBSERVATION II.—J. F., aged 35, a male patient under Dr. Lediard, in the Cumberland Infirmary, died of "surgical kidneys." The body was plump, and there was considerable subcutaneous fat, which, as well as the skin, had a peculiarly yellow hue. The second left space was occupied for about one inch and a half by the conus arteriosus and pulmonary artery; the junction of the two traversing this space. The left appendix was invisible; but its tip could be seen by getting a profile view, as in the former case. The second right space was occupied by the appendix of the right auricle, which entirely covered the aorta here. The right auricle extended for about one inch outwards in the third right space.

OBSERVATION III.—G. N., aged 46, a male inmate of Garland's Asylum, died of pernicious anæmia. The conus arteriosus occupied the second left space, and the origin of the pulmonary artery was under the second rib. The left appendix was invisible, and its tip

was only revealed by drawing aside the conus arteriosus and adjoining artery. The heart was very fatty, with a mottled endocardium, and its cavities were relaxed and dilated. It weighed twelve ounces and a half.

OBSERVATION IV.—J. S., aged 35, a female patient under Dr. Lockie, in the Cumberland Infirmary, died of pernicious anæmia. The conus arteriosus occupied the second left space for about one inch and a half, and extended upwards under the second rib, under which rib also lay the origin of the pulmonary artery. The left appendix was invisible, and could only be seen by turning the heart round, and removing about an ounce of fluid from the pericardium. The heart was fatty and dilated.

OBSERVATION V.*—A man, aged 30, died in the Wolverhampton General Hospital of pernicious anæmia. The origin of the pulmonary artery was under the second rib. The conus arteriosus occupied the second left space for fully two inches. The left appendix was not visible, and the heart had to be turned upwards to bring it into view.

OBSERVATION VI.—J. B., aged 47, a female patient in the Garland's Asylum, died of what is clinically known as general paralysis. This case, although not bearing directly on the question at issue, has some interesting relations to it, and is therefore given here. The patient was greatly emaciated. The origin of the pulmonary artery was at the level of the lower border of the second rib. The second left space was occupied, for two inches, by the conus arteriosus and a narrow margin of the left ventricle. There was a strip of left ventricle from this point to the apex, occupying the anterior surface of the heart as it lay in position. At the apex, it occupied fully one inch of this aspect, and presented a large milk-spot. The apex was under the fifth rib. The second right space was occupied by the right auricular appendix. The tip of the left appendix could only be seen by getting a profile view in the manner already mentioned. The left ventricle was in a state of concentric hypertrophy, its wall measuring one inch and a quarter in thickness. The mitral orifice barely admitted two fingers, and, when viewed from the auricular side, it showed a thick hard ring round its orifice. The left auricle, considering the size of the heart, was probably enlarged; its appendix was not lengthened, but was, perhaps, somewhat increased in width. The wall of the right ventricle was not much thicker than the wall of an empty urinary bladder; and when opened, by carrying the knife from within the pulmonary artery downwards to the apex, keeping close to the septum, it gave the idea of being simply a flap stretched over the greatly hypertrophied left ventricle, which bulged far into its cavity. All the valves were healthy. The coronary arteries were very atheromatous, the anterior one being a calcareous tube. The arteries throughout the body were in a similar condition. The heart weighed twelve ounces and a half.

The above cases are typical, and, as such, they materially interfere with the acceptance of the auricular appendix theory, which is, perhaps, to be regretted, as it is a theory of intrinsic beauty and considerable charm. We are thus reduced to accept the popular belief that the pulmonary artery is the seat of the murmur. In

clinical proof of this, I give the following tracings, which bear an essential resemblance to some of those taken by my friend Dr.

* This case I have referred to in the *Edinburgh Medical Journal*, August 1882.

Gibson, and on which Dr. Balfour partly founds his theory, although I am not aware of his having publicly ventured on an analysis of them. Tracing A was taken from Case VI; B, C, and D were taken from Case III. Here I may say that, in chlorosis, the chest-wall is too thick, and the patients, as a rule, too plump, to give tracings of any value whatever. A and B were taken from the site of pulsation and murmur in the second left space; C and D, from the third left space, with different pressures. All four present the common feature that the space *a*, between the dotted lines, is occupied by a gradual rise immediately preceding the systolic shock; this

represents the time of the filling of the ventricle, and of course the auricular systole. This is succeeded, as already indicated, by the abrupt rise caused by ventricular systole. The summit of A and B

(from the second space) is sharp, descent beginning at once, and interrupted, as is well seen in B, by two slight halts in its course, only faintly produced in the tracing, when the button is raised from the vessel during inspiration, and which resemble the tidal and dicrotic waves of an ordinary arterial tracing. A does not show this so well, as it was taken with the button of the sphygmograph resting lightly on the chest. On the other hand, the summit of C and D (from the third space) is broad, with a slightly downward inclination; and following this is a sudden fall, at the end of which the cycle begins again. The tracings C and D, from the third space, are, of course, from the right ventricle; and its sustained summit is the result of a heart well in contact with the chest-wall. The tracings A and B, from the second space, may be explained thus: the space *a* represents, as has been said, the filling of the ventricle, and must, therefore, have been obtained from the conus arteriosus; but, with the contraction of this part of the heart downwards, the pulmonary artery comes under the button of the sphygmograph, and gives the arterial character to the down line. This is consistent with our *post mortem* observations, and with our knowledge of the change occurring on the right side of the heart during ventricular systole, and may, therefore, be claimed as a clinical demonstration of my contention.

Having thus come to the conclusion that the murmur can only have its site in the pulmonary artery, the question next arises, How is it produced there? Lack of space forbids an examination of the various answers given to this question; suffice it to say, they all present insuperable difficulties to their general acceptance. Some months ago I had the honour of making a communication on this subject to the Medico-Chirurgical Society of Edinburgh,* when I advanced the explanation, based on anatomical facts, and a consideration of the condition of the heart and of the pulmonary circulation, that, owing to the dilatation of the left auricle and the instantaneousness of its diastolic fullness, due to its imperfect emptying into the ventricle on the one hand, and, on the other hand, from the volume of blood pressing into it from the surcharged pulmonary veins, the pulmonary artery was, during ventricular systole, compressed by it; and that this was the cause of the murmur heard in the vessel. This might more accurately be regarded as an interference with the calibre of the vessel, from partial flattening, as it is stretched, during systole, over the excessively full base of the left heart. It will be understood that this fullness increases the distance the vessel has to cover to reach the root of the lungs. I further contended that, in more advanced debility, the murmur which appeared in the third and fourth spaces was a tricuspid regurg-

* See *Edinburgh Medical Journal*, August 1882, and *Transactions of the Medico-Chirurgical Society of Edinburgh*, vol. i, 1882, Oliver and Boyd. Dr. Balfour's criticism of that communication appears in the same journal for September, and in the same volume of *Transactions*. My reply to Dr. Balfour appears in the same journal for November.

gitant one; and that, at a certain stage, the murmur in the second left space was also tricuspid, for the conus arteriosus occupied that space; and that the murmur disappeared from the pulmonary artery by its being carried upwards and outwards, and thus away from the line of pressure of the auricle. This change allows the vessel to reach the root of the lungs with greater ease, and relieves it from the traction it had, up to this point, to endure as its root descended with the contracting ventricle.

On that occasion, I did not go beyond what has been here called the third stage. In the fourth stage, the murmur is, as a rule, heard all over the cardiac dulness to the left of the sternum; it may not extend quite as far as this in the space below the nipple, but it is heard exceedingly rarely beyond it. At the same time, it is often heard over the greater part of the sternum, and may attain its maximum loudness in the fourth space; but this is not invariably the case, although, as a rule, it no longer attains its maximum in the second space. The fifth stage is characterised by the extension of the murmur to the second right space, and has often added to it part of the preceding stage. In the sixth stage, the murmur is heard in the third right space. In the communication referred to, it was submitted that the murmur in the third and fourth left spaces was heard over the part of the right ventricle which, when the ventricle was enlarged, was the first to approach the chest-wall, and might be seen or felt pulsating there; that this fact, combined with other clinical facts, and clenched by the undenied presence of tricuspid regurgitation, led to the conclusion that the murmur must be attributed to this cause. The further extension of the area of audition of the murmur, which continues to be a tricuspid one, is explicable by the continuance and progress of the cardiac changes; for it will be found that, with the enlargement of the right chambers, what is practically a rotatory movement of the organ takes place to the left round its longitudinal axis, that this has the effect of bringing more and more of the right side into closer contact with the chest-wall, and that this change proceeds from left to right until the right ventricle approaches the sternum sufficiently for the murmur to be heard over this bone. But the murmur also appears in the classic "aortic area" (the second right space), and has, from Hope's time to Dr. Balfour's latest utterances, been regarded as produced at the origin of the aorta.

Case VII, and one or two other cases which I have seen recently, had a murmur here, and it was louder here than in the second left space, but it was quite lost over the sternum above this space, and not a trace of it was conducted into the carotids. The murmur was so loud that, had it been produced at the orifice of the aorta, it would have been a physical impossibility for it not to have been carried onwards with the advancing stream; on the other hand, our *post mortem* observations show that the second right space is occupied by the appendix of the right auricle, and that here the aorta is under its cover. The inference that the murmur is a tricuspid regurgitant one carried into the appendix, and not aortic in origin, is unavoidable; and when we get the sixth stage, with the murmur appearing in the third right space, which space we have seen to be occupied by the right auricle, and the murmur gradually fades on going outwards, until it is only audible as the softest of breath-sounds, the clinical evidence is surely incontrovertible, and amounts to demonstration.

I do not seek now to go beyond this stage, as I have had no illustrative cases lately; but it is to be remembered that the right side of the heart may be still further dilated than the stage to which it has been followed here.

The foregoing was mainly attained before I knew aught of Parrot's work; but to him undoubtedly belongs the credit of being the first to recognise that, in the more advanced stages of debility, the cardiac murmur was due to tricuspid regurgitation; how he overlooked, or did not attempt to explain, the primary murmur produced in the pulmonary artery, I know not. It is for me heartily to accept, while honestly trying to extend, his work, believing the site of this murmur at the tricuspid orifice, and its regurgitant mechanism, to be in accordance with the facts of pathological anatomy, in harmony with clinical observation, consistent with reason, and bearing in its simplicity evidence of its truth.

I have to acknowledge the kindness of Dr. Lockie and Dr. Lediard, honorary members of the Infirmary staff, and of Dr. Campbell, Superintendent of Garland's Asylum, in allowing me to utilise the above cases. At the same time, I have to acknowledge the skill and readiness with which Dr. Waters, House-Surgeon to the Infirmary, and Dr. Macphail, Assistant Medical Officer at the Asylum, carried out my suggestions in the *post mortem* room, and their courtesy in permitting me to take part in the examinations.

ABSTRACT OF LECTURES ON PHYSIOLOGICAL DISCOVERY.

Delivered at the Royal Institution of Great Britain.

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LECTURE IX.—THE NERVOUS SYSTEM: REFLEX ACTION.

ALL mental operations were referred to the action of the soul or mind, residing in the material frame, and it was long before such operations were in any way associated with physiological changes. Even many animal movements were supposed to be caused by the direct operation of the spirit. At last, movements were divided into voluntary, involuntary, and mixed. These are clearly defined by Robert Whytt (1714-1766) in his celebrated essay published in 1751. He taught that a certain power or influence lodged in the brain, spinal marrow, and nerves, is either the immediate cause of the contraction of the muscles of animals, or is at least necessary to it. He also studied involuntary motions, such as the contraction of the pupil by light; but he explained many such phenomena by the action of a "sentient principle," residing in the part and in the central nervous organs. He recognised that the stimulus applied to a sensory nerve excited something which was transmitted to the central nervous organs, but he called the direct effect of the stimulus a sensation occurring in the part affected.

In his work on "Nervous, Hypochondriac, and Hysteric Disorders," published in 1764, he shows that so-called sympathetic actions of one part of the body with another, could not be explained by anastomosis of nervous filaments, but by the transmission of impressions to nerve-centres, and the retransmission outwards to the parts affected.

For many years the term *sensorium commune* was used by physiological writers, and may be met with in the works of Lancisi (1654-1720), Willis (1622-1675), Boerhaave (1668-1738), and Camper (1722-1789). It usually meant the portion of the nervous system affected by all kinds of nervous impressions. Its function related more to what we would now term sensation, as all other kinds of mental activity were referred to the action of the immaterial principle or soul. In the *sensorium commune* also, as expressed by Descartes (1596-1650), the mind came into relationship with the body.

Little advance was made until the publication by Unzer of his *Principles*, in 1771. In this work he discriminates between movements with "conceptions," and those without "conceptions." He had the idea of "a transformation of the external impression into the internal without any conceptions being experienced." Thus, he describes the movements produced on irritating the foot of a decapitated frog, and he clearly refers the conversion of sensory into motor impressions to changes occurring in ganglia.

These views of Unzer were expressed more fully by his intellectual successor, Prochaska, in his *Dissertation*, published in 1784. Both of these writers, however, supposed that, when a nerve is irritated at one point, the influence will travel to each end, and the effect produced may be either sensation or motion. Whilst we know that an irritation of a nerve-filament does produce something which travels in both directions, we also now understand that the physiological effect observable to the senses depends on the arrangement at one end of the nerve. This view, however, influenced both Unzer and Prochaska in their theories of reflex action. They both saw that external impressions reached the *sensorium commune*, and were, by it, converted into motor impressions. Neither of them recognised clearly the fact that the sensory impression was conveyed inwards by one set of nervous filaments, and that the motor impression was conveyed outwards by another set. Nor did they know the parts of the *sensorium commune* involved in specific nervous actions, such as we would now term reflex.

The lecturer then described the experiment of Gilbert Blane, in 1788, in which he divided the spinal marrow of a kitten, by cutting it across the neck. The hind paws being then irritated, by pricking them, the muscles belonging to the posterior extremities were thrown into contraction, so as to produce the effect of shrinking from the injury. In repeating the experiment, he found that when