

ART. VIII.—*On the Diagnosis and Treatment of certain Affections of the Heart.* By ROBERT J. GRAVES, M. D., &c. &c.

No disease requires more attention than pericarditis, whether we consider the importance of the organ engaged, the frequency of its occurrence, or its often insidious and latent progress. In studying this affection we can derive little or no assistance from ancient or even modern authors, except of the most recent date, for inflammatory affections of the heart and its investing membranes were either completely overlooked or grievously misunderstood, until long after the investigations of Laennec had disclosed the advantages of physical signs. It was then discovered that, contrary to the received opinion, pericarditis and endocarditis must be ranked amongst common affections, and that they are accompanied, during their origin and progress, by physical signs highly characteristic, and of such importance, that they enable the practitioner not merely to distinguish the first stages of the attack, but to anticipate its origin and extinguish it at its very commencement. The truth of this assertion is proved by every day's experience; and we have now the satisfaction of knowing that inflammation of the heart and its membranes is not necessarily either fatal or intractable. Still, however, we must not suppose that recent investigations have satisfactorily established the value or the meaning of all the physical signs that can be detected from the commencement to the termination of inflammatory affections of the heart, for the nature and position of the organ engaged, whose motions can be seen, felt, and heard, occasion changes in the physical signs, which alter and vary from stage to stage, from day to day, nay, sometimes from hour to hour. The study of variations so numerous and yet so important, will require the co-operation and well-weighed testimony of many observers; with a view of promoting the cultivation of this fertile field, the following remarks have been collected, and will, I trust, con-

tribute to enlarge and render more accurate our views, in certain points connected with this department of pathology :

CASE I.—Sounds produced by Pericarditic Friction, closely resembling those derived from Valvular Disease ; method of Diagnosis.

A man named Mulcahy, æt. 23, was admitted December 1st, 1841. He stated that he had led a very intemperate life, his usual allowance being from six to eight glasses of whiskey daily. He earned a livelihood by playing on a wind instrument, and after a few hours' performance, used to suffer from distressing palpitations and pain about the heart. At times he was affected with a sense of fainting, which usually terminated in vomiting. He followed his avocation till about two months before admission, when he was attacked with rheumatism, and shortly after with great dyspnœa, anasarca, &c. &c. On admission, his surface was cold, lips and hands livid, feet swollen, and belly distended. He suffered from dyspnœa ; cough, with bloody expectoration ; his eyes were staring and protruded ; face tumid ; jugulars turgid, but not pulsating ; pulse 70, regular, but small and weak ; respiration 28 ; urine scanty and highly albuminous ; extreme debility. The left lobe of the liver occupied the epigastric region, in which situation alone pressure caused pain. He had slight pain in the right shoulder. There was no dulness except over the lower and back part of both lungs, where the respiration was weak and accompanied by a moist crepitus ; the cardiac region sounded duller than natural. The motions of the heart were evident, strong, diffused, and were accompanied, not by the two natural sounds, whose duration and tone are so different from each other, but by two loud, prolonged sounds, of equal duration but of different tones ; the first was a *bruit de scie*, the second was a musical sound, closely resembling the noise made by rubbing the moistened finger on glass. These phenomena were only heard at the base, and were quite inaudible at the apex of the heart ; but they extended from the base along

the aorta, and were very distinct under both clavicles, particularly the left; they were not heard either in the carotids or in the cervical portions of the subclavians. In no situation was there the least frémissement; no thrill in any of the arteries of the neck or upper extremities; no abnormal sound over the abdominal aorta. The next day, his condition was much the same, except that instead of the musical sound we had a well-marked and loud *leather creak*, very much prolonged and masking the normal second sound, and a strong frémissement was felt over the base of the heart; there was no increase of dulness. The pulse continued regular, 72; the respiration only 20; but he was evidently sinking, and on the following morning he died.

Post Mortem.—General anasarca; both pleural cavities were occupied by a large quantity of fluid, upon which the lungs floated; on the left side the heart was bedded in the lung, and both were carried into close apposition with the anterior parietes of the chest, so as to bring the heart into extensive contact with the sternum and costal cartilages. There was no fluid in the pericardium, but its surfaces were coated with lymph, shreds of which extended from one surface to the other at the base of the heart. In this situation, the lymph appeared to have been very recently effused; it was easily removed, and presented an irregular honeycomb appearance. At the apex of the heart, the opposed membranes were firmly united. The heart itself was hypertrophied and both its ventricles dilated. All the valves, the endocardium, the aorta and pulmonary artery were quite free from the least trace of disease.

There were many particulars connected with this case that might have led a practitioner to consider it as one of valvular disease. From the man's own account it appeared, that he had for a long time suffered from palpitations, faintings, dyspnœa, anasarca, &c.; and his mode of life and occupation frequently produce that affection; but the physical signs were more likely to mislead than either the history of the case or the general symptoms. He had an enlarged heart, detected by increased

dulness, two prolonged sounds masking the natural sounds of the organ, not audible at the apex, but exceedingly distinct over the origin and course of the aorta, one of these sounds having a perfectly musical tone, a character which hitherto has not been noticed in pericarditis. At our first visit, these circumstances, taken in connexion with the absence of *frémissement* over the heart, or of pain or uneasiness about that region, together with the state of the pulse, might have easily led to a wrong diagnosis. But, on the other hand, the phenomena differed in many points from those *supposed* to be indicative of disease of the aortic valves. The sounds, though heard to a great distance, did not follow exclusively the course of the aorta or its trunks; they were not heard either in the carotids or subclavians in the neck, nor was there any thrill or visible pulsation in these vessels; and in addition, the sounds appeared, when examined by the stethoscope, to be derived from a superficial source, and were almost equally intense over a large space. These were the circumstances that induced me to look upon the case as pericarditis.

The next day the matter was put beyond doubt, for the musical sound had disappeared, and was replaced by a leather-creak, attended by a strong *frémissement* over the base of the heart. During all this time the pulse remained at 72, was perfectly regular, though weak, but the action of the heart was much stronger than natural, a circumstance frequently observed in this disease.

The manner in which the heart was pushed against the bony parietes of the chest, satisfactorily explains the fact of the sounds being heard to so great a distance, the organ itself at the time acting with more than usual vigour. But what were the conditions that gave rise to the musical sound? Let us reflect for a moment upon the actual state of the heart and pericardium in this case. If we examine the parts when removed from the subject, seeing the heart collapsed, and the pericardium loosely surrounding it, we cannot then understand how such sounds

could be produced, by the motion of the one within the other. But such is not the condition of these parts in the living body : the pericardium is there firmly fixed at its apex and base, it is tense and stretched like the parchment of a drum ; and if in this bag we have an enlarged heart moving slowly backwards and forwards, the heart itself being turgid with blood, and rigid from the contraction of its muscles, we have the conditions that most probably gave rise to the sounds described, the true intensity and loudness of which would, of course, be altered and vary according to the varying condition of the two surfaces rubbed together ; and it is well known, that membranes, similar in structure to that covering the heart, and lining the pericardium, have their surfaces altered by inflammation remarkably and rapidly, being at one time smooth and dry, and then becoming quickly smooth and moist, and afterwards covered either with puriform matter, or denser lymph, which latter may coagulate, forming on the rubbing surfaces either a punctiform roughness, or ridge-like pseudo-membranous projections ; and it is evident that each of the conditions now enumerated must alter the tone of the sounds produced by the friction of the opposing surfaces, or affect their loudness and duration. Pericarditic sounds may, therefore, be as loud, *and as prolonged as valvular sounds*—a fact hitherto scarcely sufficiently dwelt upon by pathologists—pericarditic sounds, like valvular, may likewise be accompanied by *frémissement* ; and, consequently, in endeavouring to make the diagnosis between the two sets of sounds, we must seek for means of distinguishing them, not in their loudness, their tone, or their duration—not in the presence or absence of *frémissement*, but in the fact, that pericarditic sounds appear to the attentive ear to issue from a more superficial source, are much more extensively diffused, and are almost equally audible in regions of the chest very distant from each other, as, for instance, under both clavicles. Pericarditic sounds, too, undergo much quicker alteration in character than valvular, which, when once formed, are almost always permanent ; and to conclude,

pericarditic sounds seem to be conducted by the solid parietes of the chest, while valvular sounds are chiefly propagated by the contents and parietes of the great vessels. The following case, published by Dr. Watson, in the Medical Gazette for 1835, illustrates, in a strong manner, the peculiarities of pericarditic sounds which I have just alluded to. He says, that in his case the murmur “represented very exactly the upward and downward action of a saw on rough wood, was by far the loudest sound of the kind that he ever heard. It was distinctly over the *whole of the chest*, both before and behind, only somewhat fainter as the distance from the heart became greater : with your ear upon either scapula, you might have supposed that you were listening to the deep buzzing vibrations of the larger string of a bass viol.” At the *post mortem* examination it was found, that the pericardium was every where, except at its posterior part, covered with a “thin coat of *firm* gray lymph, quite rough with minute papillæ, projecting from almost every point of its surface, of an almost *horny consistence*, harsh and resisting to the touch.”

The following case is, in many particulars, extremely worthy of notice, and is unique, so far as my experience goes, in this, that the rheumatic inflammation seized the pericardium *before* the joints. This fact proves, that physicians have been hitherto too prone to attribute pericarditis, carditis, or endocarditis, to metastasis, a doctrine applicable to some cases, but by no means to all, for, as in the present instance, the first symptom of a rheumatic inflammation may occur in the pericardium before any of the joints are affected ; and in the case of Reddy, to be next related, the pericarditis began at the very time that articular inflammation had reached its maximum intensity. But if the heart and its investments may be thus attacked at the very beginning, or during the acme of rheumatic fever, it is easy to believe, likewise, that inflammations of the heart and membranes may commence for the first time towards the termination of rheumatic fever, when the articular inflammation has almost

disappeared, and under such circumstances, a superficial view of the phenomena discovers the easiest explanation in metastasis ; neither is it unimportant to observe, that the fever usually accompanied by inflammation of the joints, and termed rheumatic fever, is a fever *sui generis*, and as readily distinguishable from the fever caused by inflammations, as is the fever of typhus, small-pox, or measles. In truth, in rheumatic fever, the quickness of the pulse, heat of the skin, tendency to profuse sweating, debility, restlessness, and thirst, may all exist, without any inflammation of the joints, and may be solved without such inflammation ever occurring, as I have witnessed in several well-marked cases of individuals liable to rheumatic fever, and who had previously suffered from attacks of fever with arthritis in the usual form, and subsequently, on exposure to cold, were seized with symptoms of pyrexia, which, in intensity, duration, and every other particular, were identical with their former fevers, save and except, that from beginning to end not a single joint was inflamed. But it may be asked, am I correct in calling such a fever *rheumatic* ? My answer is, that in the instances referred to, the urine was exactly the same as in former attacks, and the sweats, whose abundance by no means alleviated the fever, had a peculiar odour which never occurs except in rheumatic fever ; another characteristic mark was likewise observable, viz., that though the fever was intense, and thirst considerable, and tongue furred, yet the appetite was not necessarily impaired, at least at the commencement of the fever. These considerations are of practical interest, and prove that in the treatment of acute rheumatism we cannot hope to cure the fever directly by means which merely tend to get rid of the articular inflammation. *As arthritis may exist without rheumatic fever, so rheumatic fever may exist without arthritis ; when combined they each aggravate the other, but the cure or disappearance of one does not necessarily determine the removal of the other.*

CASE II.—A woman, æt. 19, named Fitzgerald, was admitted September 1st, 1841, into the hospital, labouring under

febrile symptoms of a trifling character. She complained principally of headach, with loss of sleep. Her pulse was quick and her tongue foul. For these symptoms she was treated, and every thing seemed going on favourably, till September 5th, when the following observation was made :

Face pallid and anxious; breathing hurried, 40; alæ nasi dilated at each inspiration; pulse has fallen from 90 to 50, very *weak, irregular and intermittent*; no cough or pain in the chest; no palpitation; physical examination did not detect disease any where except over the cardiac region, in which there was a distinct rubbing sound, accompanying both sounds of the heart. It was most intense at the apex of the organ, and appeared to accompany the first sound more particularly. It was attended with a very perceptible frémissement; in no situation had it the character of a "*soufflet*." The impulse of the heart was exceedingly strong, and its sounds very loud. She was cupped over the heart and put on the use of calomel and opium, five grains of the former with one of the latter, every fourth hour.

September 6th. Countenance much improved; pulse 72, full and soft, but still irregular and intermittent; respiration 28; alæ nasi not dilated; no pain in any part. The *friction* sound is still very evident, though less intense, particularly at apex of heart; no dulness, impulse stronger than on yesterday, sounds of heart very distinct. Blister over the heart, and continue the pills of calomel and opium.

September 7th. Mouth sore; pulse 76, small, soft, regular, *without any intermission*; respiration 28, countenance good; the impulse and sounds of heart are both good; the friction is barely audible, being most intense over the right side of heart. Cont. pills.

September 8th. No trace of frottement; the sounds and impulse of heart natural; pulse 80, regular and soft.

10th. Was last night attacked with pains in the loins, knees,

shoulders, wrists, and ankles. These joints are now exceedingly painful, red and swollen. Pulse 80, small and soft.

It is unnecessary to go through the details of the case. Suffice it to say, it ran the usual course of severe articular rheumatism, and lasted for about ten or twelve days. The heart was daily examined, and exhibited no sign of disease throughout. The treatment consisted of opium in large doses, *one grain every third hour*; it succeeded admirably, and seemed to expend itself solely on the disease, for during the whole time she was taking it, it never produced contraction of the pupil, headach, hot skin, furred tongue or constipation.

M. Chomel has long since shown, that when the pulse becomes suddenly feeble, faltering, intermittent, or unequal, without any apparent and adequate cause, this sign, especially if attended with the usual concomitant symptoms of an obstructed circulation, affords the strongest evidence of pericarditis; and Dr. Hope asserts, that on this sign alone he has seen M. Chomel found a successful diagnosis in the last stage of a typhous fever, where the symptoms were extremely complex. Chomel's observation is, I think, correct, and leads me to discuss the motions of the heart in pericarditis and carditis more at large. *In some cases of pericarditis the heart's action becomes increased in strength for many hours before any physical sign of pericarditis can be detected*, and before any pain is felt in the region of the heart. In such cases, when the usually acknowledged symptoms of pericarditis are added to this already existing augmented action of the heart, the latter goes on increasing, and finally becomes excessively violent, and does not begin to decrease notably for several days after the peculiar symptoms of pericarditis have disappeared. This course may be, perhaps, explained by supposing that the muscular substance of the heart became inflamed before the pericarditis came on, and continued to be so after the pericarditis had subsided, for it is a general principle, that super-added inflammations generally yield to remedies, before the fundamental and primary disease exhibits a manifest improvement.

In rheumatism, the action of the heart should be carefully watched, and when it becomes increased without any apparent cause, that occurrence alone is sufficient to warn us of the approaching danger.

This point has not, I believe, hitherto attracted the attention it deserves, and its importance is enhanced by the fact, that an increase in the heart's action may not only precede the physical, but the constitutional symptoms of inflammation of that organ or its membranes, and consequently may be the only beacon to forewarn us of a danger still beyond the visible horizon, and undiscoverable by any other means. Connected with the motions of the heart is the remarkable disparity that exists between the energy of the heart's action and the strength of the pulse, for it often happens, that the pulsations in the cardiac region are violent while the pulse is weak and thready at the wrist. This disparity consequently prevents us from deciding on the propriety of antiphlogistic measures by examining the pulse, a circumstance which shews as how erroneous *a priori* conclusions are in medicine, for surely it is in inflammation of the heart and its membrane that we should have expected the pulse to be our most certain guide. In pericarditis, it was formerly supposed that the pulse was invariably accelerated, except towards the close of the disease when the vital powers of the heart became exhausted, or its motions impeded by effused fluid. But this is so far from being correct, that in several of my cases it will be found that the pulse was not quicker than natural from the beginning to the end of the disease, and in the boy Reilly, whose post mortem examination will be just now detailed, several German physicians of eminence who honoured my clinic with their presence, could not be persuaded, by the evidence of the most indubitable physical signs, of the existence of pericarditis, because they said, in no such case could the pulse be natural in its frequency, softness, and rhythm, and they were only convinced when the pericardium was opened. A perfectly natural pulse is not an unfrequent occurrence in pericarditis, but the case before us exhibits

a very remarkable peculiarity, viz., *a sudden decrease in the frequency of the pulse at the very origin of the disease.* Of this I have witnessed but one other example where, in the commencement of pericarditis, the pulse fell to 36, and was extremely weak, faltering, irregular, and occasionally intermittent.

The gentleman whose pulse thus fell in a way similar to that of Fitzpatrick, likewise recovered. The causes that produce quickness of the pulse in one case and its slowness in another, in every other respect apparently similar, will for ever remain undiscovered; and the same observation probably applies to the causes upon which depend irregularity of the heart's action. It is well known, that certain forms of dyspepsia, hysteria, and nervous diseases, occasion palpitations of the heart, and every variety of irregularity and intermission in the pulse, and that without any inflammatory or organic complication. When, therefore, inflammation attacks the heart or its membranes, palpitation with irregularity, weakness, and intermission of the pulse, may be its indirect effects acting on the nervous energy of the heart. This explanation seems the most satisfactory that can be advanced; but still we cannot help thinking, that the rhythm of motions of heart are sometimes directly interfered with by the inflammation, nor is it difficult to conceive, that where perhaps one auricle and ventricle are inflamed, while the other auricle and ventricle are free from disease, the simultaneous action of these parts may be deranged. Be this as it may, and whichever hypothesis we adopt, it is of paramount practical importance to recollect, *that a weak, irregular, and intermitting pulse may exist in the very commencement of pericarditis*, that it may not exceed the natural frequency, or as in the two cases detailed may fall much below that standard, and yet antiphlogistic treatment be required. The result of my experience is, that in carditis and pericarditis, when the pulse is weak, irregular, and intermitting, when it is soft, natural in its frequency, or else morbidly slow, that general venesection should never be employed; leeches over the region of the heart, cupping, blisters,

calomel with opium are best suited to this emergency, if it occurs during the acme of the disease, but when towards the close, our chief reliance must be placed on powerfully blistering the region of the heart, dressing the vesicated surface with mercurial ointment, and exhibiting internally small doses of calomel with large doses of opium, and, if necessary, wine. *Digitalis* exerts little or no control over inflammation of the heart; and, like colchicum, if given in doses at all proportioned to the danger, it often suddenly produces dangerous or even fatal prostration of the nervous system. In protracted forms of cardiac or pericarditic inflammation I have found colchicum combined with mercury and opium a useful adjunct; and where the disease is decidedly chronic, refusing to yield to treatment, much benefit is sometimes derived from hydriodate of potash. Should a case, where either the internal or external surfaces of the heart are engaged, resist all these remedies, the analogy of rheumatic iritis leads Surgeon M'Donnell, my clinical clerk, to propose the internal administration of spirits of turpentine, a proposal that meets my approbation. A similar analogy would, under certain circumstances, point out large doses of guaicum, or its preparations, as likely to afford relief in very obstinate and intractable pericarditis. Part of Reddy's case was published in the January Number of this Journal, and I now bring forward the remaining portion of its history to prove that pericarditis may arise when the articular inflammation is at its height.

CASE III.—*Pericarditis arising at the Moment that the Inflammation of the Joints was most intense.*

On the day of admission his pulse was only 72, and the action and sounds of the heart natural. The next day, June 18th, eight o'clock A. M., the pulse had risen to 96, was full, strong, and regular; the heart's impulse was greatly increased, and with the first sound was heard a distinct "*bruit de soufflet*." There was no increase of cardiac dulness; no pain or uneasiness on pressure over this region or in the epigastrium; no palpitations; in short, so far as the patient's sensations were concerned,

the cardiac affection was completely latent. He was again examined at twelve o'clock the same day, and we now heard, in place of the bruit de soufflet, a well marked frottement accompanying *both* sounds, extending all over the cardiac region, and attended with a very evident frémissement. The cardiac region was not duller than usual. The pulse had risen to 108; it was strong and hard, but not irregular. He was cupped over the heart, and calomel and opium largely exhibited; five grains of cal. and one of opium four times a day. The next day the *friction* sound had disappeared, but the soufflet had returned, and again accompanied the first sound. Two days after there was no soufflet whatever.

The pericarditis in this case did not follow the subsidence of the inflammation in any other part, but occurred at the very acme of the articular disease. The day on which the sound of friction was first heard, was that on which he suffered most acutely from the articular affection.

CASE IV.—*Pericarditis.*—*Pulse regular.*—*Peculiar Eruption over the Body, &c.*

A boy, named Reilly, æt. 14, was admitted September 15th, 1841. He stated, that a fortnight before admission he was attacked with shivering, headach, pains in the loins extending along the margin of the ribs, and with severe pain in the pericardial region and violent palpitations. Two days after, an eruption appeared. When admitted, he presented the appearance of an advanced stage of typhus. He was quite collapsed, feet cold, and hands blue. The surface of the body was covered with an eruption of a small size and elevated, giving to the hand the sensation of being covered with particles of sand. It was of a *miliary* form, and filled with a sanguinolent fluid. It seemed to have appeared in successive crops, for in some parts it was quite fresh, and the little vesicles were full and prominent; in others they were broken and levelled. Pulse 72, scarcely perceptible. He got wine and hot jelly, and warm stupes to his legs.

September 16th. I saw him for the first time. His pulse was then 72, *weak, but regular*; his respiration 40, and laboured; lips livid; great anxiety of countenance. He complained of extreme pain in the cardiac region, increased by pressing the ribs towards the heart, or by making deep pressure in the epigastrium, so as to push upwards against the diaphragm. The stethoscope detected a remarkably loud frottement all over the præcordial region, accompanied with a strong frémissement. The frottement was heard with both sounds; and in some situations, particularly towards the right nipple, it had the character of the bruit de cuir neuf; there was no bruit de soufflet; impulse violent, and sounds of heart loud; no dullness; *the morbid sounds did not extend beyond the cardiac region.*

There was scarcely any change till two days after, when, in addition to the pericarditis, he was attacked with acute pain in the right hypochondrium, with excessive tenderness on pressure. The next day the legs and belly began to swell, and new phenomena were observed in the neighbourhood of the heart. The friction sounds, which two days before were very loud, corresponding to the apex of the heart, were now perfectly absent, and though the cardiac region sounded clear, yet immediately above the nipple, and for two inches and a half upwards, *there was complete dullness*, and all over this dull region we heard the *friction* as loud as ever, and that modification of it called *the leather creak*, which was still confined to the right side of the heart. It was found, that these sounds were quite independent of the respiratory movements, for they went on uninterruptedly during the cessation of breathing. Early next morning he died, and the post mortem examination was exceedingly illustrative. The lungs and pleura were quite healthy. The heart occupied a situation higher in the thorax than usual; *its base corresponded to the space between the first and second ribs*, and was evidently pushed up by the left lobe of the liver, and the fluid so suddenly effused in the abdomen. On sitting up the

pericardium it was found thickened ; the external layer was very vascular, and both it and the layer covering the heart were thickly coated with lymph. At the apex of the heart the two surfaces were closely united, but at the base there was no attempt at union. In this situation, but more particularly towards its sternal aspect, the lymph was thrown out in greater abundance, and presented a rough and nobby appearance. The substance of the heart as well as the valves and endocardium were free from disease. The peritoneum was quite healthy, but its sac was distended with a large quantity of straw-coloured serum, without any lymph. The liver was greatly enlarged and engorged with blood, which exuded freely from any incision made into it. The intestines and stomach were quite normal. The kidneys exhibited the second stage of the albuminous nephritis (so called), and the urine in the bladder was albuminous.

It is right here to draw the reader's attention strongly to the fact, that although the impulse of the heart was violent, yet the sound produced by the roughened pericardiac surfaces against each other was very limited in extent, being only audible over the region immediately covering the heart ; whereas in Mulcahy's case, and that detailed by Dr. Watson, the pericardiac friction gave rise to a sound audible over even the most distant parts, and in them nearly as loud as in the cardiac region. What can be the cause of a difference so striking ? It cannot be accounted for by any corresponding difference in the nature of the lymph effused, and a consequent difference in the physical constitution of the rubbing surfaces, for no such difference could be perceived between the pericardiac pseudo-membranes in the case of Mulcahy and that of Reilly. In Dr. Watson's patient they are represented to have been somewhat of a horny nature, a fact which may be thought to explain the loudness and extensive diffusion of the sound ; but as such an explanation does not account for the great difference observed as to the extent and diffusion of the pericardiac sounds in the two other patients, it becomes

a matter of great interest to ascertain its real cause, and, after much consideration of the subject, and duly weighing all the phenomena exhibited during life and revealed by dissection, I have little or no hesitation in affirming, that, in Mulcahy, the sounds were louder and more extensively audible; because, first, his heart was greatly hypertrophied and enlarged, and, consequently, the rubbing surfaces were actually greater in extent; secondly, because, as happens in all cases of considerable enlargement of the heart, the position of that organ within the chest is altered, and a much greater proportion of its body comes in contact with the chest; and thirdly, (but upon this I shall not insist so much as upon the two preceding) because in Mulcahy the water effused into the pleural cavity pressed the heart still more closely against the sternum and ribs, which thus acted as conductors of the sound.

These cases are then peculiarly instructive, *as indicating a great difference between the diffusion of the rubbing sounds heard in pericarditis attacking a heart previously healthy and of natural dimensions, and pericarditis supervening where the heart is enlarged and hypertrophied.*

Having spoken of rheumatic inflammation as affecting the substance of the heart itself, it is necessary to observe, that the existence of rheumatic inflammation of the heart is rather inferred than proved. A little reflection will, at all events, convince us, that rheumatism, properly so called, affects certain systems of muscles much more frequently than others. The locomotive muscles are those most usually the seat of rheumatism; and even among them, an inexplicable difference may be detected upon close examination. Those employed in the motions of the head and neck, and those which perform the flexion of the lumbar spine, being by far more frequently affected than any others; on the other hand, all that class of muscles which is connected with organic life, is comparatively exempt from muscular rheumatism; thus the extensive system of the intestinal muscles are seldom, if ever, so affected. The vesical

muscles are similarly circumstanced, and it may be doubted whether the muscles of the heart do not enjoy the same immunity. The muscles of the heart, it is true, are often excited into inordinate action by rheumatic inflammation of their lining or covering membranes; but this very increase of action would be either impossible, or attended with excessive pain, if the muscular structure was attacked by rheumatism at all resembling that which we observe in lumbago or crick in the neck. Such an affection would render the heart's motions, particularly when increased, extremely painful; indeed it would most probably arrest them altogether.

UPON "BRUIT DE SOUFFLET" AND "FREMISEMENT," AS MEANS OF
DIAGNOSIS IN DISEASES OF THE HEART.

Recent authors have contributed much to cardiac pathology, and, if we credit all they assert in their books and essays, have left but a scanty harvest to be reaped by their successors. My own experience, however, has been very unsatisfactory, inasmuch as it has not unfrequently appeared at variance with the rules laid down by authors, and I have, consequently, been led to believe, that the means of distinguishing diseases of the heart from each other, have not been yet brought to the alleged degree of perfection, and indeed many reasons induce me to conclude that such perfection is unattainable, for we can localize disease of the heart only by the following means: first by the sort of derangement each induces in the circulation and its associated functions; secondly, by the changesuch disease produces in the motions of the heart as felt by the patient, or as perceived by the eye or hand of the observer; and thirdly, by the morbid sounds developed during the heart's action.

The numerous observations and dissections I have made, have convinced me, that the functional derangements produced by disease of any particular part of the heart, are seldom sufficiently characteristic to enable us to make out whether the disease be situated in the auriculo-ventricular or semilunar

valves, nay, it has frequently occurred to me, that all the symptoms supposed to be indicative of disease of the right side of the heart, have been occasioned by diseases of the left side, and *vice versa*. So far, indeed, from symptoms being always precise enough to point out the seat of the disease, they are often insufficient to indicate its very existence, an assertion proved by numerous specimens exhibited at the Pathological Society.

The chief means of distinguishing which of the valves of the heart is diseased, is derived from the supposed direction of the sound. This is by far the most useful diagnostic mark we possess, and by it we may often, but not always distinguish disease of the right from disease of the left side of the heart, and we may, even occasionally, though not often distinguish diseases of the auriculo-ventricular from those of the semilunar valves. Another means of diagnosis much relied on, is taken from the morbid sound accompanying, and therefore being a perversion of the first or of the second sound of the heart, but is at each motion of the heart, valves are opened and valves are closed, a morbid sound may be produced by any change of structure which permanently prevents the complete opening or shutting of the valves, and consequently the same sound may arise, either from changes of structure obstructing the advancing blood, or from changes permitting regurgitation; in other words, it is impossible to judge at the moment a sound occurs, which of these is its cause.

As to the motions of the heart, their derangement scarcely ever indicates the seat of disease with any precision.

CASE V.—Bruit de Soufflet all over the Cardiac Region, but particularly loud to the Left of the Nipple, and not extending along the Aorta; Post Mortem—no Valvular Disease; extensive Disease of the ascending Aorta.

A man named Connell, æt. 50, was admitted August 10th, 1841. He stated, that for eight years before admission, he had

suffered from palpitations and dyspnœa, which had increased greatly in severity of late; he had always led an intemperate life, and for many years was in the habit of drinking from ten to twenty glasses of whiskey in the day. When admitted he was much emaciated, his belly was distended and his legs œdematous. He had cough with purulent expectoration, no dyspnœa when at rest, and his pulse was 74, soft and regular; decubitus on the right side; no pain in any part of the chest or abdomen; no enlargement of jugulars, but the tops of the ears and lips are blue; no visible pulsation, thrill, or bruit de soufflet in any of the arteries of the neck or upper extremity, and when at rest, no suffering from palpitation. *Physical signs.*—Chest sounded dull all over right side, both before and behind; in the upper part, the respiratory murmur was weak and mixed with crepitus, below it was scarcely audible. The left side sounded clear, and the respiration was loud, puerile, and free from rale; there was slight increase of cardiac dulness, particularly towards the sternum; the impulse of the heart was strong and rather diffused, its sounds loud; the first was accompanied by a bruit de soufflet, audible all over the cardiac region, but remarkably intense to the left of the nipple. *This did not ascend along the course of the aorta, nor was it accompanied by any frémissement.*

From his admission into hospital till his death, which took place five weeks after, there was not the least change in the cardiac signs. The physical phenomena did not undergo the slightest alteration; the pulse was always natural in frequency and free from any intermission or irregularity, and unless disturbed, his breathing appeared easy and tranquil. The anasarca increased, and the cough became more distressing; the crepitus heard on admission gradually passed into gurgling, and on the 20th of September he died.

Post Mortem.—The abdomen was greatly distended with fluid; the intestines were healthy; the liver was somewhat enlarged, and its edges rounded off, but otherwise natural; the gall bladder contained a few calculi; the *lungs* were connected

to the parietes by old adhesions; the left was exceedingly healthy; the right was studded with tubercle, and its apex was occupied by small cavities; the *heart* was hypertrophied, and the pericardium universally adherent, the union being effected by a dense cellular membrane. There was not the least trace of vascularity or of recently deposited lymph, but the pericardium was much thickened. *All the valves of the heart, the semilunar, tricuspid, and mitral were perfectly healthy; the aorta was dilated at its ascending portion (not at its arch), its lining membrane completely removed, and its inner surface rough and scabrous from an abundant deposition of earthy matter in its middle coat.* The arch of the aorta and its descending portion were extremely healthy; and the normal condition of the aortic valves was put beyond question, by pouring water down the aorta, not a drop of which escaped into the ventricle.

In this case, the permanency of the bruit de soufflet during many weeks, and its being constantly confined to the same place, left no doubt of its being owing to an organic fixed cause. This bruit, though heard over the right side of the heart, was more audible over the left, and, therefore, we looked for that cause in the left cavities, and we assumed that the mitral valves were the seat of disease, or altered in their structure. This diagnosis, however, I considered more tentative than certain, and explained to the class, that I had not much confidence in it, for, though the bruit was loudest immediately over the situation of the mitral valve, yet, in the majority of cases, regurgitation, through the left auriculo-ventricular opening, is accompanied by a marked derangement of the pulse. Against its depending on disease of the aortic valves or of the inner surface of the aorta itself, I urged the fact, that the bruit could not be heard along the course of the aorta, as recent writers say it invariably is in either of these cases. The absence of visible arterial pulsation and thrill was opposed to the supposition of permanent patency of the aortic valves. Dissection proved that

the bruit was occasioned by a roughness of the internal aortic surface, embracing the whole of its ascending portion. Here, then, is a fact totally at variance with received notions, and, in my opinion, quite subversive of the rules laid down by those pathologists who think they can always discover the causes of cardiac bruits by a close examination of the intensity and diffusion of the sound. I leave it to others to explain the fact, as certain as it is anomalous, that a loud bruit de soufflet, caused by extensive aortic roughness, had its maximum intensity over the region of the mitral valves, and could not be traced along the ascending aorta. How are we to distinguish such a case as this from disease of the mitral valves? The following, from Dr. Budd, in his *Clinical Remarks*, at King's College Hospital, published in the *Medical Gazette* for January the 7th, 1842, exhibits symptoms, functional and physical, almost so perfectly identical with those detailed in the instance of Connell, that a candid observer, reading the history of both, must conclude that they necessarily depend upon exactly the same structural alterations. "A girl, named Maria Pepler, was admitted into King's College Hospital on the 18th November, 1840. She was 25 years of age, and had been living in service. She stated that her health was very good until five years previously, when she became affected with dropsy of the legs, which went off at the end of six weeks. Since that time she had been subject to palpitation and shortness of breath, with occasional cough; and the dropsy had recurred whenever she had taken cold. On admission, she complained of palpitation, much increased by any exertion, and of occasional faintness. There was difficulty of breathing to such a degree, that she was unable to lie back; and a troublesome cough, attended with expectoration of a frothy mucilaginous fluid, and sometimes so prolonged as to bring on vomiting. The lips and cheeks were of a purplish hue, and there was great distention of the jugulars. Much dropsical swelling of the lower extremities, but no cedema of the hands and face. A systolic bruit was heard over the

præcordia, loudest at the point of the heart, *and to the left of the mamma*. At the point of the heart no diastolic sound could be heard. Towards the sternum and base of the heart, the systolic bruit diminished very much in intensity, and the natural diastolic sound became inaudible. There was no morbid sound in the course of the aorta or carotids. Auscultation of the lungs indicated increased secretion from the bronchial tubes. On the 14th of December she died suddenly.

“*Post Mortem*.—The heart was of enormous size, placed transversely and quite uncovered by lung. The right ventricle is enormously dilated, and its parietes are thicker and firmer than those of the left ventricle. The apex of the heart is formed by the right ventricle, and descended lower than the left. The left ventricle is not dilated or hypertrophied. Both auricles are greatly dilated, and were gorged with blood. The mitral valves are joined together, and perfectly rigid, forming a permanent aperture which scarcely admits the tip of the little finger. A good deal of bony matter is deposited under the investing membrane of the valves; but there are no vegetations on their surface. One or two extremely minute warty growths on the tricuspid valves. The aortic valves are, perhaps, a little thickened; but, in other respects, they are perfectly natural, as are also pulmonary valves and the aorta.”

Notwithstanding the boasted perfection of the means pointed out by recent writers, and which they aver, always indicate with certainty the nature and locality of valvular diseases of the heart, it must be allowed that these means were totally inapplicable, as leading to a diagnosis between the cases of Connell and Pepler. We are led, therefore, to the humiliating confession, that excessive disease of the mitral valves cannot be always distinguished from aortic roughness in the present state of science.

In Connell's case we did not even suspect the existence of aortic roughness, because some of the physical symptoms believed to be most strongly indicative of that roughness were wanting, viz., vibration felt along the right edge of the sternum,

and loudness and roughness of the systolic bruit heard there and over the arteries of the neck. The absence of these two so vaunted diagnostic symptoms, in a case where there were ossific plates on the inner surface of the ascending aorta, is scarcely more destructive of the presumptions of modern cardiac signs, than is the presence of the very same two symptoms in the following case, also related by Dr. Budd (*Medical Gazette*, December 24, 1841), and in which they originated from diseased aortic valves. After detailing the sufferings and post mortem of the patient, whose name was Coyne, Dr. Budd sums up his remarks by saying, "When Coyne was admitted into the hospital, it was evident, from the great extent of the dulness at the præcordia, that the heart was much enlarged, and from the powerful and heaving impulse, that there was hypertrophy of the left ventricle. We inferred also, from the visible pulsation of the arteries, and from the diastolic bellows-sound, heard about the base of the heart, that the aortic valves were diseased, and admitted regurgitation. The loud systolic bruit heard at the apex, might also arise from such disease of the aortic valves. The strong vibration felt by the hand showed that there was some ossification.

"So far our predictions were realized. But we were led to imagine, from the strong vibration felt along the right edge of the sternum, from the third rib of the clavicle, and from the loudness and roughness of the systolic bruit heard there and over the arteries of the neck, that there were ossific plates on the inner surface of the ascending aorta. In this, however, we were mistaken; this portion of the artery was quite healthy.

"This case of Coyne shows us how perfectly a vibration, originating at the aortic valves, and causing a systolic bruit, may be propagated along the arteries."

In the same lecture Dr. Budd ascribes to the late Dr. Hope, the discovery of those symptoms and signs, which indicate "permanent patency of the aortic valves," whereas it is well known that Dr. Corrigan, in his paper on this subject, was the

first to lay down rules for the diagnosis of that affection, and, I believe, that even at the present his communication contains all that we know on the subject.

CASE VI.—*Bruit de Soufflet and Fremissement all over the Chest, both before and behind, and in the Arteries of the Neck, &c.; without Evidence of Pericarditis or Valvular Disease.*

A remarkably fine girl, about ten years old, named Mary Robinson, was admitted, November 1st, for symptoms supposed to depend on hydrocephalus. For this disease she was treated in the usual way, and appeared to improve gradually. Four days after admission the following note was taken: lies half asleep; occasionally crying out from pain in the head; her face is pale; lips puffed and pale; head drawn back; muscles of the neck rigid; there is no appearance of abscess or tumour in any part of the neck, or œdema. The head is hot, but the pupils are quite natural; there is a very remarkable pulsation in both carotids, attended with loud bruit de soufflet and thrill; the action of the heart is violent, its sounds loud, and with the first is heard a very loud bruit de soufflet, which is not confined to the cardiac region, but is heard all over the chest, *both before and behind*, and in every situation there is a strong fremissement. There is no bruit in the abdominal aorta; she has no dyspnœa, palpitation or cough; no pain on pressing over the heart, or pushing up the diaphragm against the apex of the organ. Pulse 100, pretty strong and full; digestive functions natural; skin hot.

She remained in the Hospital for ten or twelve days after the above note was taken, the bruit and thrill gradually became less distinct, but at the time of her departure they had not entirely disappeared.

In the case just detailed, a most remarkable feature was the intense fremissement or *thrilling vibratory motion*, preceptible by the hand on whatever part of the chest it was placed. This

thrilling motion appeared nearly equable throughout all the pectoral regions, and was synchronous with the systolic motions of the heart and a loud bruit de soufflet, which, likewise, was equally audible all over the chest. The phenomena in this case were, in my opinion, totally unconnected with pericarditis or valvular disease, and the result showed that opinion to be correct, for the physical phenomena disappeared under the use of nervous medicines and nutritious diet. It becomes interesting to determine, first, how we are to distinguish such a case from pericarditis or valvular disease; and secondly, how are we to account for the physical signs which this girl exhibited. With respect to the first question, it may be thought, that a thrilling vibratory motion so intense, and a bruit de soufflet so loud, and both nearly equable all over chest, could not be produced by pericarditis, but this is not correct, for I saw along with Dr. Parkinson, a case in North Great Charles-street, where a bruit de soufflet as loud, and a vibration as intense were established all over the chest in the interval between our morning and evening visit, in a gentleman labouring under pericarditis. I regret that I took no note of this case at the time, and consequently cannot say whether the bruit de soufflet and thrill extended to the carotids. I regret this the more, because if they did not so extend the diagnosis between such a case and that of our patient Robinson would be obvious. The absence of any dyspnœa or other irregularity of the respiratory function, made it evident, that in Mary Robinson the thrill and bruit were unconnected with pericarditis, for pericarditis could not give rise to such phenomena except when most intense, and when thus intense it always produces functional derangements easily to be recognized.

With respect to the diagnosis between the phenomena observed in our patient and those which occur in valvular disease, it is sufficient to remark, that in the latter the thrill and bruit are never equally diffused over the whole back and front of the chest. Next, with regard to the cause of these phenomena, it

is to be held in mind, that similar physical signs are produced by vibrations arising from the blood flowing through roughened arteries or diseased valves; a result sufficiently explicable by ordinary principles of acoustics; and secondly, that they may be caused by pericardial friction in pericarditis. Physiologists have applied themselves to the explanation of the thrill and bruit so often heard in hysterical, nervous, and exhausted patients; but I am not aware that these phenomena have, in such persons, been observed to extend beyond the vascular system, or have been imparted in all their intensity to the whole parietes of the chest.

I do not feel myself at present enabled to offer any solid reasons for either supporting or opposing the opinion generally advanced concerning the cause of frémissement or bruit in the arteries of the nervous or debilitated; and I am equally at a loss to account for these phenomena, as observed in the thoracic parietes and arterial system of Mary Robinson, and my consciousness of the difficulty of offering any adequate explanation is increased by the fact, that they were entirely absent in the abdominal aorta and arteries of the lower extremities.

ART. IX.—*Researches on the Pathology and Diagnosis of Cancers of the Lung and Mediastinum.* By WILLIAM STOKES, M. D., M. R. I. A., Regius Professor of Physic in the University of Dublin. Physician to the Meath Hospital, Secretary to the Pathological Society of Dublin.

I PROPOSE, in the present paper, to examine into the actual state of our knowledge of the history of thoracic cancer, and to examine how far its diagnosis, direct or indirect, can be considered to be established.

Although the disease seems not unfrequent in these countries, yet we have but few recorded examples of the affection, and still fewer instances in which any accurate observations of symptoms have been made. It is, in fact, only within the last