witnessed the execution of a woman who had destroyed her infant by the same means.

A case related in Paris and Foublanque's "*Medical Jurisprudence*", is so curiously illustrative of the above, and of the necessity of attending to such peculiarities of structure, that I shall subjoin a brief account of it.

At the Devon assizes, in March, 1800, Thomas Bowerman was indicted for the murder of Mary Gollop when fourteen years of age; more than a month before the trial, his daughter, Elizabeth, twelve years of age, stated, that she saw him kill Mary Gollop by pushing an awl into her head; and pointed out the spot, near the ear, where the perforation had been made. In consequence of this statement, in February, 1800, two years and a half after her death, the body of Mary Gollop was disinterred, and an inquest held. The skull was examined, and a small hole, of the size of an awl, was found near the ear, just where Elizabeth Bowerman had pointed out; the coroner's jury, in consequence, returned a verdict of wilful murder against the prisoner. The case attracted the attention of Mr. Sheldon, who, after examining the skull, declared his opinion that the hole, supposed to have been made by an awl, was a natural perforation for the passage of a vein; and pointed out the fact, that there was a sort of enamel round it which could not have been there if it had been made by force. He, moreover, produced a dozen or more human skulls, having in them similar perforations, variously situated, and presenting a similar appearance of polish round their edge. The consequence was that the grand jury ignored the bill.

ART. VIII.—*Observations on Pericarditis*. By ROBERT LAW, M. D., Professor of the Institutes of Medicine in the School of Physic in Ireland.

PERICARDITIS ever has been, and ever must be, a subject of deep interest. The pathological conditions of a structure so intimately connected with so important an organ as the heart must ever commend themselves to our most anxious attention, and especially when the subject is one of extreme difficulty. For, although the diagnosis of pericarditis is less obscure now than it was formerly,—although it cannot now be said that there is not a single characteristic sign to denote its presence—and, thanks to auscultation and percussion, we are not left to vague

conjecture, or to infer *par voie d'exclusion*, that, because the disease is nothing else, it is, therefore, likely to be pericarditis,—with all that these modern helps have done towards rescuing this important disease from the difficulty and obscurity that enveloped it from its origin to its termination, of which it might be truly said, that it rose in obscurity and set in obscurity:—we have still much to learn about it before our knowledge is as complete as the importance of the subject demands. It is a singular fact, connected with diseases more or less intimately related to the central organ of the circulation, that they are amongst the most obscure and difficult of detection of all diseases; and that, while we have such confidence in the heart that it will not allow anything to be seriously amiss in the system without apprizing us of it—while we almost constitute it a *custos salutis*, a kind of detective—although it may fulfil these functions in reference to the affections of other organs and systems, and discover on them—still we find it exercises a most remarkable reserve on its own affections.

How often will unexpected death reveal an aneurism of the aorta whose existence was never even suspected during life! And how often are we indebted to the derangement the aneurismal tumour produces in the functions of other organs, for its discovery! How often has dyspnœa or dysphagia been the earliest announcement of this formidable disease, while the heart has maintained the most unruffled composure! Pericarditis also, not long since, shared in this obscurity. But we may be told that auscultation and percussion have produced for this disease a certainty of diagnosis that leaves little to be desired. I willingly admit that much has been achieved by these valuable diagnostic helps; still, much remains yet to be done before the requirements of practical medicine are fully satisfied. I contend for the frequency of the disease, and for its obscurity, on the grounds of the frequency with which we meet with appearances of inflammation of the pericardium, even to the extent of producing complete obliteration of its cavity, when no symptom during life bespoke its existence. And if such an amount of unequivocal inflammation consist with absolute latency of the disease, are we justified in questioning the inflammatory character of the *tâche laitue*, because we meet with this appearance so much more frequently than palpable pericarditis presents itself to the physician? If this white spot were met with elsewhere, its inflammatory nature would be readily conceded, and especially if there existed at the same time other inflammatory appearances which are most commonly present with the *tâche laitue*, such as cellular bands

of various lengths connecting the opposite pericardial surfaces. Besides, so far from there being anything either in the function or structure of the pericardium to exempt it from inflammation, both are calculated to produce a peculiar susceptibility of it.

I would canvass the actual state of our knowledge of the diagnosis of this disease with the view of seeing what auscultation and percussion have achieved for us. I would also notice some of the difficulties that present themselves to us in the use of those diagnostic helps; and I shall conclude my remarks with what I fondly trust will prove to be a contribution of some value in the diagnosis of this interesting disease, at a stage of it when diagnosis almost allows that it is utterly at fault.

We know that when the opposite pericardial surfaces, roughened by lymph, are allowed to rub against each other, they produce a sound fitly designated the attrition murmur. It is the proper sign of pericarditis, and its discovery has been the grand achievement of auscultation in redeeming this disease from its diagnostic reproach. But, I would ask, is this sign constantly present when the pericardial surfaces are in the physical condition to produce it? No, it is not. The surfaces may have the necessary conditions for its production, but may be, and very frequently are, kept asunder by interposing fluid, and it is not until this be more or less removed that the phenomenon will be developed. Something of the same kind occurs not only in pleuritis, but in pleuro-pneumonia, where, in case of hepatization of the lung—although the pleural surfaces are in a condition to produce the attrition murmur if they rubbed against each other—yet the state of the lung being such as to refuse admission to the air, the motion required for the production of the phenomenon is wanting, and it is not until resolution of the hepatization has made some progress, as indicated by the crepitus redux, that it appears. In many cases the pleural element of the pleuro-pneumonia would have altogether escaped detection if the stethoscope had not discovered the unlooked for friction sound. I have met with cases of pericarditis where the extent of the effusion never allowed the pericardial surfaces to come into contact, so that the false membrane in which the heart was enveloped exhibited a uniformly rough, flocculent appearance. In other cases, although the effusion had been so considerable as completely to submerge the heart, the point of it could still rise above the fluid and strike against the opposite pericardial surface, and so produce a very circumscribed friction murmur. And in the necroscopic examination of such cases we could measure the exact

limits of where we heard this murmur, by the smoothness of the point of the heart and of the opposite surface, strongly contrasting with the rest of the rough flocculent false membrane. We here see an instance of function modifying organization, and nature endeavouring, as far as possible, to assimilate the pathological product to its parent structure. In some cases of pericarditis, with very copious effusion in certain positions of the body, the friction murmur will be present, and be absent in others. Dr. Corrigan and Dr. Stokes have both noticed this fact, the sound being very marked when the patient was examined in the horizontal posture, while it was no longer to be heard when he sat up.

As many cases of pericarditis are attended with extensive effusion, when the interposition of the fluid will interfere with the production of the friction sound, auscultation now only intimates to us the feeble, and, as it were, the deeper and more remote action of the organ. It is now on percussion we have chiefly to rely, which helps us, by marking out the extent of the effusion. This occasions a dullness, which can be due only to pleuritic effusion, or to dilatation of the heart, or pulmonary solidification, or pericardial effusion. We shall have but little difficulty, in general, in distinguishing between effusion into the cavity of the left pleura and effusion into the cavity of the pericardium. In case of pleuritic effusion, the heart will be displaced unless it be retained in its position by an adhesion contracted between its exopericardial surface and the internal surface of the lung. Can we as readily distinguish between the dullness caused by a dilated heart and by pericardial effusion? I believe we can, generally; the history of the case will materially aid us; any considerable mount of dullness from effusion will be sure to be attended with a sense of oppression, which comes on suddenly, often preceded by pain in the pericardial region. I say, often preceded by pain,—but I would ask (*en parenthese*), why is not the pain more constant, when it is so frequent a symptom of the inflammation of this structure elsewhere? This has often perplexed me. Before experiencing the sense of oppression, the patient was not aware of any præcordial distress; there was no premonitory announcement of it; the distress, if any, from dilated heart is ever gradual. I believe, further, that the application of the rule, laid down by Laennec, will generally avail in establishing the distinction, namely, that in case of dullness from effusion, the sounds of the heart are strictly confined within the limits of the dull sound, while in dilatation they are heard beyond these limits; so that the extent to which

they are heard beyond the limits of dulness serves as a measure of the dilatation of the organ. A case may occur, however, in which this rule may fail us, when pericarditis with effusion is complicated with aneurism of the ascending and arch of the aorta, so that the more obscure signs of the pericarditis are lost in the more striking and palpable signs of the aneurism. I have met with this complication in a woman, aged 28, who was under my care in hospital, exhibiting the unmistakable signs of aneurism of the ascending and arch of the aorta. Her breathing was greatly oppressed, and attended with stridor; she had also some difficulty in swallowing; her face was deeply congested; her neck swollen; there was considerable dulness to percussion at the upper part of the sternum, and towards the right side; corresponding to the dull sound there was a double pulsation, which could be followed down to the heart, whose action was weak. The patient was constantly in the sitting posture, with the body bent forwards; there was no question of the existence of an intro-thoracic tumour, and almost as little of that tumour being an aneurism; the absence of an abnormal sound satisfied us that the aortic valves were free. The patient was almost *in extremis* when admitted into hospital. Examination of the body revealed a large aneurism of the ascending aorta (in which the valves were not engaged), and of the part from which the anterior innominate proceeds; there was, besides, a pericardium filled with turbid serum, in which the heart, covered with flocculent lymph, seemed suspended; the extent of the effusion prevented the immediate contact of the opposite pericardial surfaces; and the double pulsation of the aneurism, mingling, as it were, with the double sound of the heart, nullified the two signs by which the complication could have been detected, viz., the attrition murmur, and the confinement of the heart's sounds within the limits of the dulness produced by the effusion. Sir Philip Crampton communicated a precisely similar case to the Dublin Pathological Society, in which the more prominent signs of aneurism so completely obscured the pericardial complication that its existence was not suspected during life.

The dulness caused by a solidified left lung can hardly be confounded with the dulness of pericardial effusion, although I confess I have met with cases of pericardial effusion so considerable as to push aside the left lung, and so to compress it posteriorly that it no longer admitted the air, and thus there did exist a difficulty in determining if the pericardial affection was complicated with pneumonia. Such a case I saw in Sir Patrick Dun's Hospital, under the care of Dr. Smith; and

such another I had under my own care; but in this latter case my difficulty was removed by making the patient assume the prone position, when the sound posteriorly became clear, and the respiration audible. This manœuvre removed the compression of the lung. But I may be asked, might not the compressing material—and which changed its place with the position of the body—might it not have been effusion into the pleura. I answer in the negative: for, if it had been, it would have caused dextrocardia or displacement of the heart, which it did not do. An adhesion between the exopericardium and pleura lining the internal surface of the lung could alone prevent this. In case of a solid lung, the heart's action appears stronger and more audible than under its normal circumstances, from the pulmonary structure being now a better conductor of sound; while, when the organ is surrounded with fluid, and compressed by it, it beats more feebly, and is heard through a medium of inferior conducting powers.

If we have had the opportunity of observing from its commencement a case where effusion into the pericardium has taken place, as occurred to me very recently, we find the area of dulness gradually diminishing, and, sooner or later, is heard a very limited, circumscribed, friction murmur corresponding to the apex of the heart. This murmur is then heard to an increased extent, both in length and in breadth, from the apex to the base of the organ, and from side to side. It is at once curious and interesting to observe the different characters of the sound in different parts, varying in smoothness and softness, according to the relative portions of the surfaces coming into contact with each other; the sound corresponding to the parts that had been longest rubbing against each other being comparatively smooth, while those brought into more recent contact emit a much rougher sound. I had an opportunity of remarking this in a case which I had under my care a short time since; the smooth friction sound corresponding to the apex contrasted strongly with the coarser sound generated nearer to the base of the organ. I would wish to direct especial attention to the direction of the friction murmur as serving to distinguish between it and a valvular murmur, which, as far as regards the sounds themselves, is sometimes no very easy matter. While the different friction murmurs are produced in the direction of the motions of the heart in the pericardium, the valvular murmurs follow the current of the blood, whether in an outward or in a retrograde direction. The heart, as it acts normally in the pericardium, has a triple motion:—1. A tilting forward motion, by which its point

strikes against the side of the chest; 2. A motion from its apex to its base; 3. A motion of rotation on its axis. If the heart be free to act in the pericardium, unrestrained by adhesive bands, the result of a former inflammation, or by intervening fluid, the result of the existing inflammation, its rough surfaces will rub against each other and produce the friction sound in this triple direction. I have seen the double murmur of aortic valve disease sometimes resemble the attrition murmur of pericarditis so closely, that if I had but the sound alone to distinguish them, I should have found it extremely difficult; but happily we had the directions of the respective murmurs to aid in our diagnosis.

A previous inflammation will produce a partial adhesion of the pericardium, and this will modify the extent and direction of the friction murmur. I had a remarkable instance of this in the case of a man, about forty years of age, in which the *bruit de cuir neuf* was more marked than I had ever before heard it. He had long been the subject of a cardiac affection; the pericardium was found to be greatly increased in thickness, and to have acquired the consistency of fibro-cartilage. Its cavity was divided into different chambers, formed by membranous dissepiments of various lengths and depths proceeding from its opposite surfaces. Some of these chambers contained fluid more or less turbid, and many were lined with recent lymph. I had no doubt that it was a case of long standing, and had been the subject of various inflammations occurring at different times, the lymph of the original inflammation being the matrix or the blastema of the subsequent pathological processes which had issued in its increased thickness and induration.

It is impossible to fix the duration of the friction murmur. It may pass away in very few days, leaving not a trace behind. Dr. William Beatty brought me to see a case of pericarditis with friction murmur; when we came to listen to it he admitted that what he had heard distinctly the day before had now ceased. An unusually strong action of the heart was alone to be heard, from which I predicted an adhesion of the pericardium, which post-mortem examination proved to be the case. At other times it may continue for weeks, or even months, when it disappears. What then happens? Either a complete removal of the material on which the murmur depended, or a partial removal of it, the remainder connecting the opposite surfaces, and destined to become cellular bands of various lengths, according to the position they may occupy, —longer the nearer they are placed to the apex of the heart,

in consequence of its greater motion, and shorter as they may happen to be nearer to the base, on account of the more limited motion of these parts; or it may remain so as to effect a complete agglutination of the opposite surfaces, and thus entirely obliterate the cavity of the pericardium. Have we now done with the disease? Has it now undergone a radical cure? Or, before we canvass this question, I would ask, have we any sign, or signs, upon which we can rely as indicating adhesion of the pericardium? These are deeply interesting questions; I shall consider the latter first, and shall refer to what are regarded as the highest authorities on such a subject.

Dr. Hope remarks: "I certainly consider this diagnosis to be one of the very few connected with the heart which cannot be made with absolute certainty, and I never, therefore, venture to assert respecting it."

He further remarks, that by a combination of signs he has succeeded in detecting it:—First, by the heart's beating as high up as natural in the chest, and causing a prominence of the cartilages of the left præcordial ribs. Second, and which he regards as the most characteristic of all, an abrupt jogging or tumbling motion of the heart, very perceptible in the præcordial region with the cylinder. Third, a history of previous pericarditis, especially if connected with acute rheumatism, affords strong presumptive evidence corroborating the preceding signs, and the absence of such history should make the auscultator pause before he ventures on a diagnosis in stronger terms than that it is probable or possible. With regard to the first sign stated by Dr. Hope, I would say, that I have never observed this high action of the heart and prominence of the cartilages of the left præcordial ribs in adhesion of the pericardium, but I have observed them in another morbid condition of the pericardium, in case of effusion into its cavity, when the fluid, gravitating towards the most dependent part, pushed up the organ towards its base, and produced a prominence corresponding to the base, the appearance which Louis designates "*voussure*." It is the pushed-up heart that forms the prominence, not the fluid, as is generally believed.

As to the second sign proposed by Dr. Hope, I can only say, that his description of an abrupt, jogging, tumbling motion, does not describe the irregular action that I have sometimes observed in cases of adherent pericardium. Dr. Walshe's description of it as a tumultuous, confined action goes nearer to conveying an idea of what I have heard. As I have observed it, it has been a kind of pulling, dragging motion, as if one contraction of the ventricle was resolved into a series of short,

abrupt contractions, sometimes so feebly and faintly expressed as if no impulse were communicated to the blood by the heart as it passed through it. The jogging, tumbling motion that I have observed has been in a quite different affection, viz., in the weak, gouty heart. M. Bouillaud remarks : " Je ne connois encore aucun signe qui puisse faire reconnaître les adherences du pericarde." Dr. Sanders thought he had discovered a positive sign of an adherent pericardium in the retraction or dimple taking place during the systole of the ventricle in the epigastrium immediately below the false ribs, and which he ascribed to the diaphragm being drawn in by the ascending motion of the heart. Dr. Hope observes on Dr. Sanders' sign :—" I have searched for this attentively in several cases of adhesion, but have not been able to detect it in any degree that could constitute a sign. Dr. Heim, of Berlin, also proposed this sign. Laennec, speaking of it, says :—" J'ai cherché inutilement depuis deux ans verifier cette observation chez tous les malades qui presentaient quelque signe de trouble de la circulation, et je n'ai jamais pu apercevoir le creux dont il s'agit, et dans le nombre de ces sujets il s'en est trouvé plusieurs dont le cœur adherait au pericarde." Dr. Stokes is the last authority to which I shall refer, as being the latest, and who had, therefore, the opportunity of testing the value of the signs of those who preceded him. He remarks :—" I more than doubt that there is any certain physical sign of adhesion of the pericardium, and have never been able to verify the sign of Dr. Hope of the double jogging impulse." I am bound to say, that none of the signs hitherto proposed has done more than to enable us to assert the likelihood of an adherent pericardium.

The sign which I propose, and with confidence, has this advantage, that we need not have followed the disease in its progress, nor do we require to have had any previous knowledge of the case. I have again and again tested its truth by post-mortem examination, as have others to whom I have communicated it, and have found that it may be relied upon. The sign to which I allude is, "*the persistence of the same extent of dulness to percussion in the præcordial region, no matter what position the individual may assume.*" The area of dulness on percussion in the præcordial region will be the same under every varying position of the body. The heart becomes so braced up that it cannot move as it does in its normal state, when, if examination be made, the patient either lying, or sitting, or standing, the results of percussion will vary accordingly, the dulness being greater in the first position, and less in the two latter. The individual himself, also, is quite con-

scious of the existence of some solid resisting body within his chest, which does not move in the changes of posture of his body, but impedes its motions.

I have proved this sign in cases where I have seen the patients all through their attack of pericarditis, and also in cases where the adhesion had been already formed, and have never found it to disappoint me. I, therefore, claim for it that it is *the physical sign* that may be relied on in proof of an adherent pericardium.

I would now return to the questions whose consideration I postponed, viz., when adhesion of the pericardium has taken place, have we now done with the disease? Has it now undergone a radical cure? Can we give the patient a *billet de santé*? I shall pursue the same course in the consideration of these questions that I did in the former instance, and shall adduce the opinions of those whom I regard as the highest authorities on the point. Our improved pathological knowledge has simplified the solution of these questions in some degree, inasmuch as we now know that much that had been charged upon pericarditis did not really belong to it—we allude particularly to the hypertrophy—which, from its having been so often found in cases of adherent pericardium, was thought to be an almost necessary result of it. We now know that this hypertrophy is most commonly the effect of the endocarditis that is so often coincident with pericarditis.

It is remarkable how various are the opinions of different high authorities on the pathological importance of adherent pericardium. All the older pathologists, viz., Lancisi, Vieussens, Meckel, Senac, Corvisart, and Morgagni, thought that with a complete and intimate adhesion of the pericardium the patient could not live in a state of health. Laennec, in stating his own opinion on this point, speaks first of Corvisart's in the following terms:—"Il (Corvisart) ne pense pas au reste qu' on puisse vivre et vivre sain avec une adhérence complète et immédiate du cœur au péricarde." Then, in asserting his own opinion, he remarks:—"Je puis assurer que j'ai ouvert un grand nombre des sujets qui ne s'étaient jamais plaints d' aucun trouble dans la respiration ou dans la circulation, et qui n'en avaient présenté aucun signe dans leur maladie mortelle quoiqu' il y eut adhérence intime des poumons ou du cœur, et je suis très porté à croire d' après le nombre de cas de ce genre qui j'ai rencontrés que l'adhérence du cœur au péricarde ne trouble *souvent* en rien l' exercice de ses fonctions." M. Bouillaud observes:—"Les adhérences ne me paraissent pas troubler nécessairement le jeu du cœur, ou

en rencontre du moins chez des personnes qui jouissaient de la plus florissante santé." It is remarkable that English physicians regard adherent pericardium with much more alarm and apprehension. Dr. Hope, from having always found enlargement of the heart in cases of adherent pericardium, concluded that the former is the constant and necessary result of the latter. He remarks:—"I have observed that cases of adhesion terminating in enlargement often hurry to their fatal conclusion with more rapidity than almost any other organic affection of the heart; and, on the other hand, I have seen patients repeatedly die from the consequences of adhesion, the history of which I could trace eight, ten, or more years, yet some individuals would represent their health to have been perfect during the greater part of that time. Hence I infer that, though close adhesion may not for a time create much inconvenience, its effects are ultimately fatal." Again he remarks:—"Unless the effused lymph as well as the serum be absorbed, it causes an adhesion of the pericardium, and thus constitutes a destructive disease." Dr. Watson designates adherent pericardium an apparent but unreal recovery. Dr. Stokes has not expressed himself distinctly or directly on this point. My own experience on the subject is, that while I have met with several cases of adherent pericardium where the patients suffered no inconvenience either in the circulation or respiration, and when death from other causes proved the adhesion;—still I have met with so many more cases in which the patients, after having had one attack of pericarditis, and affording every reason to believe that the pericardium had become adherent, were afterwards the subjects of an intercurrent inflammation in the cardiac region, which at length terminated fatally; and where post-mortem examination revealed an adherent pericardium, the adhesion being effected through the medium of a false membrane, whose depth and density were in proportion to the age and duration of the disease, and which besides exhibited various pathological modifications, such as being the seat of hemorrhage, suppuration, tubercle, and cancer:—with such evidences as these, and comparing the one set of cases with the other, I have no hesitation in saying, that I should ever consider an individual with an adherent pericardium to be in an insecure state, inasmuch as the disease has left behind it a sickly product, not alone more susceptible of being affected than the parent structure was, but which has, as a principle of its constitution, on the occasion of every future pathological movement, a tendency to become more and more degenerate, and thus to drift more and more into unhealthy action.