It is, I would say, equally necessary to the infant and the child; whereas, in reference to the middle periods of life, the diminution or increase of supply must to a great extent rest upon the judgment of the practitioner. By the copious administration of stimulants the flagging powers of life are upheld; by the free exhibition of sedatives, pain and irritation and spasm are subdued. To how great an extent it may become necessary to press these measures is amply illustrated in the case which I have given in full detail. Here it was necessary absolutely to flood the patient with wine and opium, and in every way abundantly to supply nutritive aliment.

In the foregoing pages I have endeavoured to give an unbiassed and candid exposition of all the facts bearing upon the important question of resection of the knee-joint,—a question of vital importance to the profession and to society at large. In doing so, I trust I have dispassionately considered the subject, and every contingency likely to arise. The important information afforded by numerous hospital surgeons, whose names are herein recorded, makes the mass of evidence brought to bear upon the subject all-convincing and conclusive. Not only are the ordinary details of the cases given, but likewise a continuous account of them afforded up to the present day, thereby creating a record sufficiently comprehensive towards refuting every objection, and establishing the operation of excision of the knee-joint upon the soundest principles of our art, upon the solid basis of truth.

In conclusion, I have only to add how deeply I feel indebted to those able men who so readily answered all my inquiries, and to thank them in the name of that noble profession under whose banners we have taken soldiership, and whose cause we have pledged ourselves to support and exalt.

ART. II.—*Pathological Affections and Relations of False Membranes*[a]. By ROBERT LAW, M.D., Professor of the Institutes of Medicine in the School of Physic in Ireland, &c.

SEROUS membranes are pre-eminently susceptible of inflammation. They exhibit this susceptibility more strikingly than any other structure or tissue in the animal body, with, perhaps, the single exception of their kindred structure—areolar tissue. This fact is attested both by living and by posthumous

[a] Read at a Meeting of the Association of the Fellows and Licentiates of the King and Queen's College of Physicians in Ireland.

proof, exhibited in the frequency with which the practical physician meets with cases of peritonitis, pleuritis, pericarditis, and arachnitis; and in the still greater frequency with which, in his post-mortem examinations, he finds unlooked-for traces of inflammation of this tissue, whose existence, in many instances, had never been suspected during life. Although pleuritis be a common disease, how infinitely more frequent are the pleural adhesions and other marks of inflammation of this membrane discovered after death! And how small is the proportion of cases of pericarditis with which the practitioner meets, however numerous they may be, when compared with the frequency with which he finds in his necroscopic examinations Baillie's white spot (*tâche laitue*) on the surface of the heart, a want of proportion so great as to cause the originally inflammatory character of this phenomenon to be questioned, but of which I have no doubt for the following reasons:—1st. Because, whenever it is present there are generally also to be found other evidences of a former inflammation having existed, such as cellular bands, of various lengths, connecting the opposite pericardial surfaces near the base of the heart, and which owe their continuance to the little motion to which this portion of the organ is subject. 2nd. The thickened, opaque appearance of this spot is exactly the same as that presented by other serous membranes which have been inflamed. 3rd. Although pericarditis does very often exhibit itself under characters so palpable and obvious as to bring it under the notice of the physician, experience warrants us in asserting that this is far from being always the case. I have found in my examinations of patients who have died of various diseases, complete obliteration of the cavity of the pericardium, the result of an old inflammation of whose existence they seemed not to have been aware, as, in the enumeration of their existing symptoms and former illnesses, they made no allusion to any cardiac affection. I have met with instances of pericarditis developing itself in the course of other diseases, some of which owed their detection, not to any symptoms that arrested the attention of the patient, but to the accidental application of the stethoscope, which discovered the friction-sound; while others were entirely overlooked, being obscured by the more prominent symptoms of the disease to which the pericarditis was superadded, and whose unsuspected existence examination after death alone revealed; and in not a few of these cases there was complete cohesion of the opposite pericardial surfaces through their entire extent. If, then, pericarditis, to such an extent as to produce a perfect obliteration of all the cavity of the peri-

cardium, can consist with such a degree of latency, it is not too much to suppose that so much of it as would be necessary to produce the white spot should be present, and still elude the notice both of the patient and physician, I would add, as a further reason for my belief of the inflammatory origin of this appearance, that the function of the organ entailing upon it incessant motion, and its organization, are both calculated to engender an inflammatory susceptibility at least equal to that of the pleura.

I assert, therefore, for serous membranes a peculiar proneness to inflammation, which is probably due to the low degree of vitality with which they are physiologically endued, exhibited in their comparative insensibility and deficient vascularity in their normal state. Serous membranes, when inflamed, pour out a fluid composed of serum and lymph or fibrine. The ordinary destination of the former is to be removed by absorption. This may be the case with the latter also, but its removal is never accomplished so speedily as that of the former. The friction-sound of pleuritis and pericarditis is heard long after the serum has been absorbed, and in many cases has to wait for its absorption for this phenomenon to be developed. The rough surfaces have hitherto been kept asunder by the interposed serous fluid. In some cases, after a much longer time, the disappearance of the friction-sound tells us that the fibrine has been removed, and that the surfaces have regained their normal smoothness. In other cases, however, the fibrine has a different destination; it is retained in the system, and becomes the seat of a process which has for its definite end to make it attain to, by a series of changes, a state in which it resembles either the parent structure or that which is most allied to it in organization, viz., cellular structure. I would here remark, that a pathological product never arrives at the perfection of the structure which produced it, but always takes an inferior or subordinate place in reference to its organization, and approaches more or less to the original structure according as it may be more or less required to fulfil the functions of this structure. We thus see, that while this product generally resembles cellular structure, if it should so happen that any two portions of it rub against each other, they lose their rough, flocculent, cellular character, and become smooth. I had an opportunity of observing this in a specimen taken from the body of a man who died of pericarditis, in which there was such an amount of effusion into the cavity of the pericardium that the opposite surfaces were kept asunder, and the point of the heart alone tilted against a small portion of the opposing surface, and,

therefore, the parts that were thus allowed to come into contact alone became smooth, resembling the parent structure, while the rest was rough and flocculent. It is not easy to assign any determinate time within which a false membrane becomes organized. The period differs in different cases. There is a case recorded by Sir Everard Home, in which the false membrane is said to have been formed, and to have admitted of injection, or to have exhibited vessels in it, within nine-and-twenty hours. It was a case of strangulated hernia which had been operated upon. The portion of intestine that was strangulated presented no appearance of lymph when it was returned. The patient only survived the operation nine-and-twenty hours, and for the last five hours of his life there was no pulse to be felt at the wrist. When the body was examined, the portion of intestine that before presented no appearance of lymph was now coated with it, and this lymph received injection. I would here observe, in reference to organization, that to refuse to a structure its pretensions to be considered an organised structure until it presents vessels capable of receiving injection, would be to ignore the organic pretensions of many animal structures in which the blood is not contained in vessels, but is loosely diffused through their parenchyma, and which, notwithstanding, retain an undisputed title to organization; while, on the other hand, if we admit the blood in the lymph to be evidence enough of its organization, we recognise the ordinary law of disease which degrades or lowers the condition of an organ or structure to what it is permanently in an inferior animal, or to what it had been for a time in the same animal in the progress of structural development. The vessels, according to this law, form subsequently in the false membrane, and it now assimilates to a higher organization in the animal series.

I would here introduce an observation of Andral's, which appears to be peculiarly applicable. He remarks: "It is admitted that the phenomena designated vital only manifest themselves under certain conditions in the arrangement of the molecules of a body—arrangement that is called organization —but the meaning of this term is far from being precise or determinate. We are not to believe that the manifestation of life only occurs when this organization exists, such as we observe it in the higher animals, and such as we are accustomed to represent it to ourselves. Follow the series of living beings, and you will observe the instruments of vital agency to diminish more and more in number and complication; you will even find them to disappear, and still there will be life. Life is not less in the vegetable seed, the liquid drop, the first ru-

diment of the animal embryo—even in these parts we find less than in the blood of the rudiments of what we call organization. In the absence of the forms to which common opinion attaches the idea of life, vital acts may still be performed. So far, then, from imposing on the manifestation of life certain conditions of molecular arrangement, observation exhibits to us a thousand different instances of life, not by forms, but by acts."

When this pathological product is duly organized, it assumes all the physical, physiological, and pathological characters of a normal structure. It has ever been an object of interest to the pathologist, both from the changes that take place in itself, and from the effects that it produces in the organs with which it is connected. The subject is too extensive a one to admit of my entering into anything like a full detail of it. I must content myself with a general view of it, and reserve for a future communication some of its most interesting and important relations, and its connexion with some of the most important organs.

This morbid product is susceptible of all the pathological conditions of which an original structure is susceptible: it is subject both to active and passive hyperemia; and often have we seen exhibited, at our pathological meetings, as specimens of hemorrhagic pleurisy and pericarditis, what were really this product of a former inflammation, now the seat of a recent sanguineous effusion. The great depth of it was to me proof enough that it was no new product, but that it had been of old date. In fact, we can always determine the age of this morbid product by its depth. I shall, on the present occasion, more particularly consider its development in its relation to the heart and lungs. I have had for years under my care and observation cases which I originally met as cases of acute pleuritis and pericarditis, and in which I was satisfied, from their peculiar signs, that this morbid product survived the acute disease and remained behind, and had become the seat of subsequent disease; and whenever I had an opportunity of examining them after death I ever found that the depth of the false membrane was proportionate to its age, and to the number of times that the patient's complaint during life had led me to suspect that it had been the seat of morbid action.

Senac records a case in which he says the false membrane of the heart attained to a depth of four inches; and although he admits that this is an unusual depth for this morbid product to attain to, yet that it is no uncommon thing to meet with it an inch thick. Pinel describes in the following terms the post-mortem examination of the famous Mirabeau, who died of pericarditis:—" Ou trouva des traces d'inflammation dans l'esto-

mac, dans le duodenum, dans une partie de la surface du foie, dans le rein droit, et dans le diaphragme, mais la partie la plus fortement affectée fut le péricarde, qui contenait une quantité considérable d'un fluide epais, jaunâtre, et opaque; des concrétions albumineuses recouvraient toute la membrane séreuse qui enveloppa le cœur excepté la pointe; le péricarde lui-même avait contracté une épaisseur des quatre lignes." The depth of the false membrane here described is a proof to me that it was not of recent formation, but that it was the result of former disease, and that it was it, and not the pericardium, that was the seat of the fatal inflammation. We can easily understand why this individual should be the subject of such a disease. The part that he played in the political drama of the day would have been cause enough; but, in addition to this, there was no kind of sensuality in which he did not indulge: as Pinel remarks:—"Mirabeau plein de confiance dans l'energie de sa constitution et entrainé par l'habitude du plaisir sentait de se jouer des ses forces physiques."

I have asserted that false membrane taking its place amongst the other normal structures of the body is at least as susceptible of disease as other structures are. It is not alone the seat of hyperemia, as I have already observed; it is the matrix of suppuration either in the form of circumscribed abscess or diffuse inflammation; it is also the seat of non-analogous products, such as scrofulous and cancerous tubercle, exhibiting this tendency more strongly than an original structure, as we have seen illustrated by this striking fact, that one and the same inflammation will cause effusion of lymph in a normal structure, while it will issue in the deposit of tubercle in this adventitious product, the result of a former inflammation. I have in my Pathological Museum a specimen of a false membrane of pericarditis, containing tubercles, which were a simultaneous result of the same inflammation that gave rise to the effusion of lymph on the lung.

I have already remarked that this false membrane will be the seat of abscess, in proof of which I would adduce the following case:—James Shepherd, aged 13, came under my care in hospital after having been ill four days, his illness beginning with sharp lancinating pain in the right side. He had considerable heat of skin and quickness of pulse, with extreme hurriedness of respiration. He could only lie on the right side, although he still felt pain in this side. There was no apparent enlargement of the side. Percussion yielded a dull sound through all the right side, both anteriorly and posteriorly. There was no trace of respiration to be heard in any part of this side, ex-

cept between the spine and the posterior margin of the scapula, where it was bronchial. No change of position made any alteration in the stethoscopic signs. The general dulness of the side and absence of respiration continued *in statu quo*. Treatment made no impression on the disease. He sank under it. Examination of the body exhibited the following interesting appearances. The pleura pulmonalis and costalis on the right side cohered through their entire extent; and the hand, in attempting to separate them, sank into what appeared to be softened substance of the lung, from which a large quantity of purulent matter immediately escaped. On removing the lung its surface was coated with flocculent lymph in the form of a shreddy membrane, and its substance appeared unusually dense and heavy. We now directed our attention to the point where the hand sank into the lung, and from which the matter issued; it was about half way between the apex and base, and between the anterior and posterior margins of the organ, we found here a well-defined circumscribed large abscess, and apparently in the substance of the lung. However, on closer examination, we discovered that the substance of the lung was not the nidus or seat of the abscess, but that in the situation of the division or sulcus of the lobes, which had been obliterated by a former inflammation, the cellular membrane that constituted the connecting medium of the opposite surfaces was the real seat of it, and the pressure of the abscess on the substance of the lung in its immediate vicinity had given it the condensed appearance of a carnefied lung. The rest of the organ was either hepatized or in a state of engorgement. I have seen what has been exhibited as a specimen of abscess of the base of the lung to be in fact nothing more than abscess in the cellular membrane, the product of a former inflammation which had caused the adhesion of the portion of the pleura lining the base of the lung, and that which lined the corresponding surface of the diaphragm.

I believe that many cases which have been supposed to be abscess of the heart were nothing more than suppuration of the false membrane, the result of an ancient pericarditis, as we know how little exposed the heart is to the formation of abscess, in consequence of the very small proportion of areolar membrane that enters into its structure,—a wise economy of Nature to secure so important an organ against disease, which is ever in the direct ratio of the amount of areolar membrane that enters into its composition. I have heard of ossification of the heart, which I knew to be no more than the transformation of the false membrane of pericarditis into bone. I also

have cases in proof of the fact of the false membrane of pleuritis being the seat of diffuse inflammation. I had one remarkable case in a man who had gangrenous erysipelas, and in whom the false membrane connecting the pleura was infiltrated with purulent matter. I have met with a case in which a false membrane investing the lungs was the seat of an extensive deposit of cancerous tubercles, at the same time that there existed a large amount of this disease in the substance of the lung; and in the same subject a false membrane coating the heart also contained cancerous tubercles in it. The case was that of a woman whose breast had been removed for cancer six months previously. The pleural false membrane was in parts fully an inch thick.

There are two remarkable circumstances connected with this morbid product that are especially deserving of attention, and with which are connected almost the most interesting points involved in its consideration. One is the remarkable contraction that it undergoes, and the influence that this contraction exercises upon the organs with which it is connected. The other is the peculiar phases of transformation through which it passes, from its appearance as lymph, then as cellular and fibro-cellular structure, and often as bone and cartilage, of which there are in my museum many remarkable specimens.

I shall first notice the contraction of the false membrane, as I conceive it will assist us, in some measure, in yielding some explanation of the transformations that this product undergoes. This contraction affects different organs differently. I shall notice its effect on the lung. It compresses it so as to lessen it in all its diameters both longitudinally and transversely; it also almost entirely annihilates its vesicular structure. I shall not farther allude to the ulterior changes it produces in the organ, as I shall have occasion to return to the subject when I come to explain more fully the pathological effects of it. I need, for the present, only remark on the obliteration of the vesicular structure, as I believe we may find in it some clue to the physiological explanation of the peculiar transformation that the false membrane undergoes. We know that in the normal state the pressure of the external air on the sides of the chest is exactly counterbalanced by the pressure of the air received into the lungs. When these are in precise normal equilibrium, the chest retains its natural shape and form. But this balance may be disturbed either by the pressure from within being greater than that from without, or by the pressure from without being greater than that from within. In both cases the shape of the chest is altered: in the former case, which occurs

in emphysema of the lungs, the chest acquires the well-known projecting form, yielding, as it does, to the pressure of the air retained in the lung. In the latter case the chest, as it were, falls in, yielding to the external pressure, which is not counterpoised by the air within, the lung being no longer capable of admitting it in sufficient quantity for such an effect, in consequence of its condensed structure. Nature calls to her aid other resources to meet this emergency. The ribs and their cartilages acquire increased strength; they undergo a process of hypertrophy. I have a specimen which presents an instance of hypertrophy of the cartilages of the ribs. If the sections on the two sides be compared, the difference is very striking. In this case the lung was compressed by a dense false membrane, and from complete obliteration of its vesicular structure admitted but little air; this side of the chest was much less than the opposite one. I believe that it is not alone the normal structures that are called upon to sustain the increased pressure, and which, to answer this call, acquire additional strength, but also the morbid products that have occasioned this change in the organ, and to which it owes its being no longer able to meet the pressure from without. These products themselves, in their ulterior organization, acquire a strength to enable them to help to relieve that condition that they have produced. We here see some of those provisional resources of Nature with which she abounds to meet the emergencies of disease, and which disease itself calls out. All the changes that the false membrane undergoes acquire for it additional strength. These consist in its conversion into dense fibrous structure, cartilaginous structure, and bony structure. This appears to me to be the explanation of those remarkable changes that we find to take place especially in the pleural false membranes. We recognise a physiological design, a purpose in them, which is at least a more satisfactory explanation of their formation than to say that when nutrition gets out of its proper orbit or track, there is no calculating where she may go—she is drifting at random. Analogy appears to lend a support to this explanation in changes which we see taking place elsewhere to answer a physiological purpose. Thus, if the dislocated bone remain out of its place for a certain time, a false joint is formed, which is effected by the areolar membrane, in the midst of which the head of the displaced bone is thrown, undergoing changes representing the different structures which enter into the composition of a true joint. Physiological necessity explains the changes in the areolar membrane. In the same way it has been observed, that the posterior cervical ligament, which is so strong and firm in the

horse and ox, and is less firm and elastic in sheep, and exists only in a rudimentary state in the cat, while in the human subject it is nothing more than areolar tissue,—in the case of man, acquires unusual firmness and strength, should he have much occasion to exercise the muscles of the posterior part of the neck, or should the position of his head be bent habitually downwards and forwards by heavy loads, and thus require for this accidental condition a strength for this ligament which the permanent natural condition of other animals demands.

I would now notice the remarkable contractile property exercised by this morbid product or false membrane—a property which exercises itself variously on different organs according to the arrangement of their structure. The influences and effects of this product were first noticed in the liver, and were seen to consist in a remarkable diminution of the normal dimensions of this organ, and in such an effect upon its blood-vessels that those of the porta distributed through it more or less refused passage to the blood through them, and from this followed congestion of this vessel and of all its tributary streams, whether of the serous or mucous capillaries, that either directly or indirectly poured their blood into it. The congested capillaries of the peritoneum allowed the serum of the blood to escape into this cavity, giving rise to ascites, while the more yielding capillaries of the mucous membrane permitted all the elements of the blood to be poured out, and thus give rise to hemorrhage, often fatal, in the form of hematemesis. The inflammatory character of this product has been questioned. Indeed, the nature of the pathological change was described by no less an authority than Laennec, in such a way as plainly proved that he quite mistook its nature. I should here remark, that I have had opportunity of pursuing cases that have presented themselves first as cases of peritonitis, terminating in effusion; the peritonitis and effusion had yielded to treatment; and, after a long interval, these same cases returned with ascites, not connected with or dependent on peritonitis[a], and which I referred to cirrhosis of the liver, a diagnosis which a fatal hematemesis, in not a few cases, afforded me an opportunity of verifying. This also served as a proof that the affection was not only inflammatory, but that it proceeded from the invest-

[a] I say, "not connected with, or dependent on, peritonitis," for I believe that in most cases of effusion into the abdomen resulting from peritonitis, percussion yields a dull sound, in consequence of the intestines being prevented by adhesions floating on the surface of the fluid, as they do in cases of effusion from other causes, when the sound is clear and tympanitic. This difference to percussion has never disappointed me as a diagnostic guide.

ing membrane of the organ to its substance, and was not centrifrugal or propagated from an inflamed duodenum to the substance of the liver, and from thence to the surface,—an opinion, I must confess, which would seem to derive countenance from the peculiar habit in which this disease usually occurs, viz., in persons affected with gastritis from the use of ardent spirits, or in whom gastro-duodenitis might be expected to occur. Dr. Corrigan was the first to give what I believe to be a correct explanation of this peculiar pathological phenomenon occurring in the lungs, and which had been designated dilated bronchial tubes. He attributed it to the same cause that operated in the production of cirrhosis of the liver, and substituted the designation of cirrhosis of the lung for that of dilated bronchial tubes. He showed how this product of disease, while it acted on the lung generally as on the liver, and contracted it in all its axes, extending into the substance of the lung at the same time that it obliterated the vesicular structure of it, dilated the terminations of the bronchial tubes, and thus, in a measure, compensated for the injury it had done.

Doubts have been entertained whether cirrhosis of the liver is really the result of inflammation. If cirrhosis of the liver and lung be allowed to be similar affections, there is no question that cirrhosis of the lung has an inflammatory origin, and, therefore, that of the liver may be inferred to have the same.

The inflammatory origin of this affection of the lungs is more within the reach of direct proof than cirrhosis of the liver. We can more frequently connect it with pleuritis or pleuro-pneumonia than we can connect the other with peritonitis. It is not easy, however, to determine why cirrhosis of the lung is so rare in comparison with the frequency of pleuritis or pleuro-pneumonia, or what is the constitutional peculiarity that causes this special product to exhibit this remarkable contractile property to prevail in any case. For it has been observed, that cirrhosis of the lung, liver, and a corresponding affection of the kidneys, with valvular disease of the heart, the result of the effused lymph exercising its contractile effects on these valves, will not unfrequently be found to coexist in the same subject, a fact of which I have had frequent experience.

Histological observations have recognised an identity of development in fibrous tissue, in false membranes, in the matter of cicatrix, and in granulations, and this identity of mode of development, and also peculiar property of contraction, would argue a positive identity of nature and constitution.

I shall now advert to some of the pathological effects of this

product on the different elements or ingredients of the different organs. It compresses all the structures of an organ: its parenchyma, blood-vessels, and nerves, especially when it penetrates into the structure of the organ. If it engage much of the structure of one lung, it will compress all its vessels, and give rise to a proportionate congestion of the vessels of the opposite lung, so that the hemoptysis, which is a very common phenomenon in this lesion, and which contributes much to its being confounded with phthisis, generally comes from the lung that is not otherwise affected; and so it occurs here as in hematemesis, dependent on cirrhosis of the liver, the blood comes from a source at a considerable distance from the seat of the disease.

I would direct attention to the complaints made by patients who suffer from affections of this false membrane, whether of the lungs or the heart. I have had opportunity of observing both for years. Some I have met with whom I had seen when first attacked with the pleuritis and pericarditis, and therefore I knew what had taken place. Other cases I met with and owed the recognition of them to the similarity of their symptoms, with those with which I was familiar. In cases of the lungs I have met with some that had been confounded with phthisis, and I knew one gentleman who was sent to Madeira for phthisis. The want of symmetry of the two sides will in general lead to a correct diagnosis. The dulness on percussion and feeble respiration, which always continue, still further discover it. The patient ever retains a sense of stiffness and inability to take as full an inspiration on this side as the other. The pain complained of is not the pain either of inflamed serous membrane, nor is it of pneumonia, but of a rheumatic character, and I believe the reason is because that the false membrane has been transformed into a fibrous membrane, and conforms to it in its pathological susceptibilities. This I believe to be the explanation of many of those pains which have been designated pleurodyniæ, or muscular pains. I have watched the false membrane of pericarditis and its affections for years. I have had the case as one of acute pericarditis, and have been satisfied of an adhesion having been contracted between the opposite pericardial surfaces; and afterwards have had the patient under my care for ailments in the cardiac region, and which did not present the phenomena of an original pericarditis, but did present those which I had no difficulty in referring to an affection of the membrane connecting the laminæ of the pericardium, and post-mortem examination has confirmed the diagnosis. I would here remark on the change which the adherent pericardium produces in the heart. It was long supposed that it produced permanent enlargement and hy-

pertrophy of the organ. Two causes contributed to this opinion: one was that it was thought probable such a result should follow from the increased action with which the heart ever tries to release itself from this impediment to its free motion,—an increased action that is always observable for some time after the adhesion has taken place, but which is not continued long. The organ appears to yield to a necessity that it cannot control, and soon resumes its wonted normal rhythm. The other cause, that so commonly led to this mistake, was the great frequency with which the heart was actually found enlarged in cases of adherent pericardium, and, therefore, the adherent pericardium, naturally enough, was regarded as the cause of the enlargement. But examination ever proved that whenever the heart was enlarged, in cases of adherent pericardium, there was also endocarditis present; and further investigations established the fact, that if adherent pericardium alone were present, there was no hypertrophy of the heart, but an actual diminution of its size, and often a conversion of its substance into fat. If we were to pursue our inquiry to its natural limits, and follow this morbid product into its effects on the endocardium, it would lead us into the extended field of valvular disease of the heart. For it is this same morbid product which, deposited originally as lymph, whether interstitially or superficially on the valves, exerts on them its contractile influence, and thus unfits them for the due performance of their function. This is too large a subject for me to do more than merely allude to at present.

The case of an intelligent and zealous pupil afforded an example of the effect of this product on a nerve. He was affected with pleuro-pneumonia of the left side, engaging the base and internal surface of the left lung. He had not been in good health at the time he was attacked. He had a very severe illness, the issue of which was for a considerable time very uncertain. When he did recover I was satisfied that the base of the lung had contracted an adhesion with the diaphragm, while the internal surface had formed an adhesion to the side of the pericardium. He afterwards became the subject of an occasional pain in the left side, which exactly followed the course of the phrenic nerve, and which seemed to begin at the situation of the primitive inflammation of the internal surface of the lung and the side of the pericardium.

I shall here close these observations, which are but preliminary to what I purpose to enlarge upon on a future occasion, deeming the subject one of the deepest importance both to pathology and to practical medicine.