If the movement of certain muscles which could not affect the pleura, produces the pain, especially if there are other evidences of rheumatism, the pain is not pleuritic. Only yesterday we had two cases in which the diagnosis turned upon this point; contraction of the *latissimus dorsi*, that could not possibly have been appreciated by the pleura, gave rise to the pain in both cases. The verdict was rheumatism.

With regard to the influence that the evidence of rheumatism has upon the diagnosis, it must be admitted that it cuts both ways. For while rheumatism is one of the commonest causes of lateral pain simulating pleurisy, it is also, in its acute and febrile form, a very common cause of pleurisy itself; so that while the evidence of its presence might suggest the non-pleuritic nature of the pain, on the other hand it would afford an explanation of, and in some cases almost constitute a presumptive proof of, its true pleuritic character.

Doubtless, the case the most difficult of solution, is the alternative between pleurisy and rheumatism of the intercostals. I have more than once been puzzled with it myself, and I have seen other and able men puzzled with it. There is in both cases the same superficial breathing, the same "stab" on attempting to take a full inspiration, the same lateral *decubitus* on the unaffected side, the same tenderness in the intercostal spaces. The rules of diagnosis that I have just mentioned to you will generally solve the mystery, but the most careful scrutiny may leave the question undecided.

The nature of this pain is no doubt the same as that of all other inflammatory hyperæsthesias, and, like them, the principal thing that aggravates it, is mechanical disturbance; hence the intolerance of pressure and of stretching, hence the superficial breathing and the *decubitus* on the sound side. I do not believe that the friction of the roughened surfaces has anything to do with the pain, for two reasons; first, because you may have, as shown in the case of Wingall, pleuritic rubbing continuing after the pain has ceased, and when nothing is felt by the patient beyond a sense of the friction; and secondly, because the pain may be severe where the existence of effusion, in considerable quantity, prevents the contact of the two pleural surfaces. It has been said that as soon as effusion takes place the pain ceases. This is not true. In the case of Franklin, the pain was of the most severe kind that I think I have ever witnessed in any case of pleurisy, while the pleural cavity was full of fluid—so full, as to preclude all possibility of friction.

Does the seat of the pain coincide with the seat of the inflammation? As far as always occurring on the same side goes, I think it does. But I doubt if it does further. Certainly the seat of pain does not coincide with the seat of the greatest friction-sound. Thus, in the case of the lad Wingall, the maximum friction was about the cartilage of the third rib, where there was no pain; while the chief pain was at the inferior angle of the scapula, where there was no rubbing. Moreover, there is a suspicious constancy about the seat of pleuritic pain, which cannot be explained by a similar constancy in the seat of the inflammation. We know, from post mortem evidence, that all parts of the pleura are liable to inflammation, while the seat of pleuritic pain is not liable to equal variety. For the seat of pleurisy there is no rule; for the seat of pleuritic pain, there is a tolerably well marked rule:—beneath the nipple, at the inferior angle of the scapula, on the acromion, and beneath the false ribs, are its characteristic situations. Moreover, some of these situations are beyond the limits of the pleura, as on the acromion, and in the interval between the last rib and the crest of the ilium, where the chief pain in Franklin's case was felt. Moreover, the seat of pain may be covered with the finger when *post mortem* appearances show that the implication of the pleura has been almost universal. From all these considerations, I am inclined to think that pleuritic pain, as far as its distribution is concerned, is chiefly reflex; and that the constancy of its situation represents some law of reflex distribution, analogous to that which makes bronchial pain sternal, and colic pain umbilical, whatever may be the exact seat of the source of irritation.

I have often asked myself the question, in cases of pleurisy, whether both costal and pulmonary pleuræ were affected, or only one, and which; and if there were any means of diagnosing this point. Is it possible to answer this question? I think, to a certain extent, it is. I think one surface may be affected without the other; and certainly both may be affected at once. I think, if there is pleuritic effusion, and pressure between the ribs at the seat of pain increases the pain, that the costal pleura is affected. I think, on the other hand, that if, under such circumstances, there is no pain on pressure, the costal pleura is not affected. I think that if pneumonia coexists with the signs of pleurisy, the implication of the pulmonary pleura is certain. I think that in all cases of pleuritic rubbing, both surfaces are certainly affected; perhaps one primarily, but *both* ultimately. In cases of pleuro-pneumonia in which there is friction-sound, the pulmonary pleura is probably affected first; and when that has been roughened, a similar state on the opposite surface of the costal pleura is set up by the chafing produced by the already roughened lung surface. In pleuritic rubbing, produced by traumatic injury of the thoracic parietes, the same events probably take place in a reversed order.

Observe the important part which the nature of the anatomical result of the inflammation plays, in these cases. In the cases of Russell and Wingall, when the febrile stage of the cold passed off, nothing remained but the inconvenience and annoyance of the stitch in the side. But Franklin's catarrhal attack left him not only with the pain, but with one lung instead of two, with the incubus of a pleura full of [illegible], upon his mediastinum and heart, and with all the circulatory and respiratory derangement and distress, that must result from such a state of things. We see from this, how, when hydrothorax is developed, it comes to constitute the substantive disease;—the pathology is lost, the morbid anatomy is everything.

What is the nature of the pleurisy in these cases? Some, no doubt would say, they are idiopathic: But I think, without adopting Sergeant Shee's definition of the word *idiopathic*,* I may show you that it would not be fair to so call them. It is quite clear that in all the cases the pleurisy was due to cold. Is this fact inconsistent with the general proposition with which I commenced my lecture,—*that serous inflammations preeminently point to states of blood-poisoning*? I think not. I think,

* In the trial of Palmer, for the murder of Cook, Sergeant Shee, in reply to a question from the bench, as to what was the meaning of the constantly recurring word "idiopathic," said that it was a word employed by doctors, to signify "that which was not understood."