

products is thickened; the epithelial cells become swollen and detached over a more or less extensive surface. The pleura denuded is rough, uneven—very soon it becomes shaggy, owing to papilliform granulations due to proliferation of the connected tissue. Later on these will undergo organisation, forming neo-membranes which will establish adhesions between its two layers, new blood-vessels will form into them, and they will then be endowed with all the properties of ordinary living tissues.

This hyperplasia solely constitutes the initial period of pleurisy. The interstitial exudation has not yet appeared, and to this fibro-plastic formation alone in the parenchyma is limited what has been called dry pleurisy, a bastard and exceptional form of pleural inflammation.

At that period, the patient complains of the rigor and fever which accompany all commencing inflammation.

The fever is, with regard to inflammation, what smoke is to fire. As long as it persists in pleurisy, the phlegmasic process has not ended its evolution. When it has disappeared, we may conclude that inflammation has almost, if not altogether, undergone all its periods.

Pleuritic fever is never very considerable, and it very seldom reaches  $104^{\circ}$  Fah. Ordinarily it oscillates between  $102^{\circ}$  and  $103^{\circ}$  in the evening, and its thermometric curve presents no uniform character as in pneumonia.

To the chill and fever is soon added an intense pain, generally situated under the nipple, and not produced by the sufferings of the pleura, but by radiation of the irritation and phlegmasic process of the serous membrane towards the intercostal nerves.

The pleura is not sensitive—it has no nerves, any more than the peritoneum, or the meninges. Intercostal nerves are in immediate relation with the pleura in the posterior third of their course; owing to this vicinity they are inflamed in pleurisy, as has been demonstrated by necroscopic observation. As all irritations of a nervous trunk resound at its terminal expansions, the stitch will be felt on the lateral or anterior part of the chest.

This pain is exaggerated by pressure. This accounts for the patient avoiding, at that period, lying on the affected side, contrary to what he will do later on.

The lung being still able to fulfil its functions, dyspnoea is slight, and caused solely by the fear of increasing the pain by a total amputation of the thorax, and also by the fever which increases combustion, and overloads the blood with carbonic acid.

As the inflammation of the parietal pleura transmitted to the intercostal nerves produces pain, so the inflammation of the visceral pleura, propagated to the adjacent cortical layers of the lung, and also to the bronchial ramifications, excites the terminal expansions of the pneumogastric, which carries the irritation to the bulb and produces by reflex action the contraction of respiratory muscles; the patient coughs.

The pleuritic cough is dry, not followed by expectoration, as in bronchitis and pneumonia, where a foreign body, exciting the expansion of the vagus, similarly provokes a tutelary reflex spasm, which expels the cause of irritation.

If you were called early enough, on applying the ear to the affected side, you shall no longer hear the soft vesicular murmur audible in the sound side, but a characteristic friction-sound, produced by the rubbing of the pleuræ thickened and covered with granulations. This friction fremitus, is of a dull, grating character, and consists of a quick succession of detached sensations, like the creaking noise of the bending of new leather. It is isochronous with the respiratory movements, and unaltered by coughing. Its intensity is sometimes so great that it becomes perceptible to the touch, and communicates to the hand, applied upon the thorax, a peculiar thrilling sensation.

Very soon the disease will enter a new phase. To the parenchymatous exudation will succeed the interstitial exudation produced by vascular exosmosis. The transuded liquid oozing through the serous membrane, deprived of its epithelium, infiltrates between the layers of the pleura, and gradually increasing, will soon fill up the pleural cavity. The effusion will modify the already existing symptoms, and give rise to new phenomena, manifested especially by physical signs, all of which result from the interposition of a non-conductor between the lung and the chest walls. This wholesome shower will, at first, relieve the clouds accumulated by the inflammatory storm in the parenchyma of the serous membrane. The intensity of the