remote lesions in various parts of the human body tends to develop pyæmia, with its peculiar metastatic deposits, or amyloid disease of the viscera—not, I believe, tuberculous disease. I regard this fact as strongly opposed to the inoculation doctrine. The very condition, chronic suppuration, as seen in necrosis, or caries for example, and which resembles so closely the action of a seton, is the very condition which is recognized as *the* cause of amyloid disease of the liver, spleen and other organs.

(5.) It is asserted that pleurisy and especially chronic pleurisy frequently causes consumption.

Now Dr. Blakiston watched for some years the course of 79 cases of chronic pleurisy with the following results — 10 were lost sight of — of 16 it was only known that they were living — and of the remaining 53 *not one* had become phthisical. Dr. Payne-Cotton's experience and Dr. Flint's is opposed to the view in question and coincide with Blakiston's. M. Aran and M. Siredey both contend that the autopsies of empyemic subjects show that tubercle is more frequently absent than one might expect.

Dr. A. Attimont's researches give 80 definite cures out of 130 cases of empyema, many of which moreover had been watched for a long time. And in 29 autopsies of empyema tubercles were absent 20 times and present only 9 times. If so many persons recovered perfectly from empyema, and if tubercle was found but 9 times in 29 cases, it certainly does not appear probable that empyema can be a very frequent cause of pulmonary tubercle.

When tubercle appears to have followed an empyema or a pleurisy, several explanations may reasonably be offered of the relationship: Tubercles may have existed in the lungs or pleura at the time of the invasion of the inflammation — or, as was admitted by Trousseau, a predisposition may have existed, which the local inflammation developed into actual disease.

(6.) If there are not sufficient grounds for asserting that the absorption of the products of pleurisy causes the