

These attacks occurred three or four times daily during the fever, gradually became less frequent and severe, but did not entirely disappear until the post-febrile depression had passed off and the temperature had risen again to normal. In this patient the attack of influenza was otherwise mild." Dr. West continuing says, "That these cases of collapse occurred so frequently with him that he found himself unconsciously speaking of the collapsing form of influenza."

In the *British Medical Journal*, 1892, Dr. St. Clair Thompson, M.R.C.P., London, reports three cases of what he calls cardiac neuralgia, one of which proved fatal. I quote particulars of this case: "Stout gentleman, æt. 39 years, took little exercise, and smoked on an average ten cigars a day, had apparently recovered completely from an attack of influenza. Eight days afterwards he had a business meeting at his house at which he smoked, drank and had a good dinner. Next morning he suffered from an attack of flatulence and vomiting, with a constant wearing pain midway between the nipple and the shoulder. Pulse 90, soft, regular and compressible. During the day the heart pain diminished without ceasing entirely, and the patient was well enough to be out of bed. At midnight the pulse was 88 and regular; an hour later, on raising his head to swallow a pill, he suddenly fell back dead. There was no autopsy."

Dr. Thompson refers to two other cases very similar in character, but not fatal. These cases differed from true angina in the absence of evidence of arterial or cardiac disease. His opinion was that the symptoms were due to the effect of the influenzal poison on the cardiac nerves.

In the *International Medical Magazine* for January, 1893, the following case is reported by Dr. J. H. Platt, of Lakewood, New Jersey: "A young woman, æt. 25 years, had been under the Dr.'s care for several years for heart symptoms, associated with a mitral regurgitant murmur. In December, 1891, she had an attack of influenza, after which lobular pneumonia developed. Her convalescence was slow, associated with great prostration and with attacks of collapse. On the evening of January 11th, at 7.45 p.m., the pulse was 102, weak and small. Fifteen minutes later, the pulse was 58, larger but weak and soft. Collapse occurred an hour after. The pulse remained

at 52 for eighteen hours when it suddenly changed to 104. Later in the day it was 96, and at 5 p.m., suddenly changed to 48. This interchangeable pulse continued for eleven days, and on several occasions the Dr. with his finger on the pulse observed the change taking place. It would occur in this wise: the pulse being 100 to the minute and regular, a beat would drop out, then after a few beats another, and after a shorter interval a third and so on, at lessening intervals until under the finger a regular pulse was felt of 50, larger and softer than the former. After a time the process would be reversed and the frequency doubled. The heart sounds corresponded with the pulse beats. Finally complete recovery took place."

I believe that Drs. McMahon and Graham of this city, saw a case in which the same phenomena occurred.

A study of these four cases, so widely different from each other in symptoms, gives us the best idea of the complex conditions produced in the heart by the influenzal poison.

How can we explain this diversity? What is the pathology of these cases? I regret to say, that of absolute knowledge we have none. We can argue from our general medical knowledge that such a complexity of symptoms can be produced only by septic causes. Under these circumstances we may obtain ideas as to causation and its mode of action by analogical reasoning from a disease of a similar nature, such as diphtheria. Of course such a method of reasoning holds good only on the assumption that influenza is a disease of bacterial origin, which there is good reason to believe.

You are aware that from the specific germ of diphtheria a tox-albumin has been separated. When this is injected into susceptible animals, it produces paralysis, nephritis and albuminuria. In other words the sequelæ of the diseases are produced. And, in addition, the same pathological changes are found on *post-mortem* examination. Every change described by Oertel, in human diphtheria, is paralleled in the disease produced in animals by the injection of this tox-albumin. The arteries may be altered in character, the capillaries may show extensive hyaline degeneration and necrotic areas in deep-seated organs may be produced.