fever poison itself. Could the nervous system be so excited by this as to produce a convulsion? In answering this question you will naturally think that the nervous symptoms are among the most prominent in typical cases of typhoid fever; you will recall the irregular chills at the onset of the disease, the pains in the loins and limbs, the ringing noises in the ears, the giddiness, the severe headache, and the profound prostration; you will reflect on the restlessness, the subsultus, vigilance, and delirium which belong to the course of the complaint almost as surely as do the diarrhœa and fever; you will think of all these evidences of great derangement of the nervous system, and be prone to conclude that convulsions are likely to be among them. But they are not. Convulsions in typhoid fever are rare, so rare as to be regarded almost as a clinical curiosity, and to be set down as something outside of the regular history of the disease. In nearly three thousand cases of enteric fever admitted into the Fever Hospital in eight years convulsions occurred in only six, Murchison tells us in his classical treatise. In typhus they happen much more frequently.

When I make these statements I allude to general convulsions. Spasmodic movements, such as jerking of the tendons, hiccough, twitchings of the muscles of the face, are, we know, not uncommon in typhoid fever; and choreic movements, rigid contraction of the muscles of the extremities and even cateleptic states, though far from common, have been often described. But convulsions from any cause in typhoid fever are extremely infrequent, and the most unusual kind is from the direct influence of the poison on the nerve centres, At least this is true of adults; a different statement must be made of children. But whether in children or in adults, convulsions due to the fever poison alone, happen at the onset, or in the first week of the malady.

Gentlemen, you know well that the vagaries of hysteria baffle any description. Among these vagaries is the occurrence of hysterical seizures in the early part of typhoid fever. I saw such a case a few years since with an eminent physician in this city. The hysterical outbreaks and convulsions in the first week of the fever were extraordinary and misleading; violent neuralgic pains in the head also existed, and the case looked like anything else rather than like one of typhoid fever. Nothing but the decided fever temperature and a certain irritability of the bowels excited even suspicion. On the eighth day appeared the characteristic eruption. I am sorry to add that the case became a very grave one, and died in the second week.

Epilepsy is among the causes of convulsions during an attack of typhoid fever. The wonder is that it does not show itself oftener. The reason I suppose to be in the strange law by which the affection is temporarily obliterated when an epileptic is stricken with an acute disease. We see whooping-cough suspending it, fevers lulling it. But the malady is not dead, it is slumbering; and it may start up vigorously during the last stages of the fever or during convalescence. I attended a case some time ago, in which three epileptic seizures happened in the course of twenty-four hours in the third week of the enteric fever. There had been no kidney complication, and no cause other than epilepsy could be discovered for the attack. The patient was very drowsy after it, yet the recovery was not much retarded by the accident. A positive diagnosis in such cases can only be made by the aid of inquiry concering the previous history. But the time of the occurrence of the convulsions should always arouse our suspicions.

Typhoid fever may become complicated in its course with apoplexy, and the effused blood lead to such damages as to give rise to long-continued palsy. A case of this kind, with right-sided hemiplegia of two years' standing, came under my notice recently at the clinic of the Jefferson Medical College, in the person of a sturdy young farmer. Where the clot is effused on the surface of the brain, convulsions may readily be associated with the apoplectic seizure. . . .

I told you, at the beginning of the lecture, that I believed uræmia to have determined, in the case we have been investigating, the fatal convulsion. Irrespective of the diseased appearance of the kidneys which I have submitted to your inspection, you may judge, from the description I have given you of other exciting causes and how they act, that they do not here apply. Let us now study a little more closely how the uræmia with its dire results is brought about.

The noxious urinary ingredients may be absorbed into the blood in consequence of the distension of the bladder. It is well known what a paralyzing effect low fever exerts on the muscular coat of the bladder. The viscus may become greatly distended, as we can recognize by percussion, although the patient seems to be voiding natural amounts of urine. The result of the accumulation may be the poisioning of the system with urea or the products of its decomposition. I shall not readily forget the impression made on my mind by a case of this kind seen a number of years ago. The young man lay for sevefal days in a state of semi-coma, associated with marked twitchings of the muscles of the face. He was more than once on the verge of a general convulsion; passing a catheter several times a day after the difficulty was recognized, removed the untoward symptoms.

But the most common cause of the uræmic poisoning, and of the convulsions that may attend it, is to be found in disorder of the kidneys. If, indeed, you will collect the recorded cases—they are, it is true, not many—you will find a condition of diseased kidney or antecedent albuminuria in the