disease. This is seen in muscular atrophy—diffuse myelitis and anterior polio-myelitis. Sometimes these causes of degeneration commence in the cord, and from it initiatory abnormal changes can be traced, but on the other hand the first evidence of disease may be in distal parts of the body. In such cases it is probable that undiscovered change had primarily taken place in special or cerebral tissues. The nerve supply simply induces pathological changes in organs and other structures. The various changes in such diseases as those of paralysis, spasm and atrophy can be accounted for in their invasion and progress by assuming the causes to exist from nutritive changes in the trophic centres, that is, when no traumatic condition is present. When centric and eccentric causes operate chronologically, the duality of malign influence is now named "deuteropathy." Our physiology thus teaches us how much proper nourishment of parts depends on nerve conditions.

It is now generally conceded that tendon—or rather muscular—reflexes are not always coexistent with Duchenne's disease. At one time, and up to a recent date, it was classed as absolutely pathognomonic of locomotor ataxia, but recent researches show that in a large number of cases this sign is absent. It seems evident the absence is due to the condition of the spinal seat of nerve supply (Prevost and Tschirew).

This location is that section of cord which supplies the third and fourth lumbar pairs. The presence or absence of this condition is no doubt largely due to the extent of nerve decay. In a medico-legal sense it is important to know with certainty that the absence of these reflexes is not negative proof of non-existence of locomotor ataxia. We are often asked in cases affecting the mental condition of a testator to diagnose this spinal disease—pure and simple—from the shuffling gait and prehensile unsteadiness of a case of paresis. The fulcrum point is, to describe a disease and its signs with initiatory mental alienation from one which, as a rule, terminates without brain disease and psychical dethronement, or, if so, only at the termination of its course. At any rate, the tendon reflex must be ruled out as having absolutely no diagnostic value in determining this disease from others of a kindred nature.

In this connection it may not be out of place to state that much diagnostic value may be found in Zoochemistry. In locomotor ataxia, for example, amyloid degeneration in the diseased portions of the cord is very characteristic. It has been held by many pathologists to be either colloid or albuminous; this is not always correct, for the well-known chemical reaction of iodine and starch takes place, with the addition of dilute sulphuric acid; and in, addition, the microscope clearly shows bluish starch granules. These starch-like bodies are easily seen in many brain affections, and often in paresis. The condition is evidently a retrograde metamorphosis of the nerve cells. When we think of our chemistry, and remember the definite changes in the alcoholic series of bodies from starch to sugar, alcohol, acetic acid and finally into carbonic acid and water, we know that all only contain three elements with different groupings and greater oxydation, and thus complexity of the analogous bodies increases.

Cerebrin is a nerve substance containing these radical elements, combined with nitrogen and phosphorus. In a degenerative and descending scale, deoxydation would bring about a more primitive grouping of this and analogous substances, and as a result a starch-like body would be generated; in other words, it might be a sugar product. The deoxydation may be caused from deprivation of properly oxygenated blood in nerve tissue, or from an undue formation of deleterious acids in wasting tissues, or from a deficiency of nerve pabulum, or all combined. There is no doubt but the amyloid condition is more frequent than is generally supposed, and is often mistaken for fat granules, or albuminous products. Fatty degeneration may be a disease peculiar to itself, or it may be simply a change of the carbon hydrates into that form, or a sort of secondary degenerative process. A *cerebraic* sugar is thus generated, which gives a starch reaction by loss of the proper equivalents of water. This want of the normal hydrate oxide is doubtless one cause of shrinkage of brain tissue. It is atrophy from a drying up process.

The importance of further investigation, through the aid of chemistry, need scarcely be stated, but differentiated chemical tests of pathological products is one of the possibilities of the future.

Along these lines our busy workers have done