lation. It is evident from recent examination that there exist these so-called "trophic centres." These spots of peculiar nerve movement and influence are rich with the multipolar ganglionic cells. Our anatomical knowledge teaches us that these regions thus endowed are in the fourth layer of the cerebral cortex, in the anterior cornua and in the posterior columns of the spinal cord. Prolongations from the minute cells also affect nutrition. This great fact is strikingly illustrated in irritation of the fifth nerve. It is followed by skin eruption, ulceration of the cornua and inflammation of the eye. In paraplegia with wasting of the muscles, we find its cause where the multipolar cells most abound, in the anterior cornea of the spinal cord. Progressive muscular atrophy has the same record, and an analogous condition, exists in posterior spinal sclerosis. We know how these states, consequent on impaired nutrition bring about abnormal conditions of the joints—such as fractures consequent on want of appropriation of animal matter to give the bones elasticity, and even dislocations from want of tone in the tissues surrounding the joints. Those who have charge of the insane need not be told how the least pressure or blow will produce ecchymosis, and a slight force will be followed by fracture of bone in those having brain diseases. Metastasis of so many ailments is, no doubt, due to changes following mal-nutrition, and the cause of these degenerations is in depreciated nerve supply from these great centres of influence. The initiatory diseased impulses are given from these centres, but must, however, be always distinguished from those produced by abnormal conditions of the periphery of the nerve apparatus, and followed by vascular changes consequent thereon. Such as the latter are brought about by vaso-motor irritation, and differ materially from the conditions of the "trophic centres" as causes of diseases from diminished nutrition. For example, such a disease as Addison's is only a result of trophic disorder of the sympathetic and ganglionic centres. The nervous condition always antedates the pathological changes in the supra-renal capsules. In other words, the difference lies in centric and eccentric causes.

Of late years the special nerve activities have been closely investigated. Many diseases which were formerly supposed to be entirely due to malign blood effects are now relegated to a class of morbid processes due to nerve depreciation. In all diseases affecting nutrition of organs, or in even physiological localities, where are found perverted functions, one or more of these nerve structures are found to be abnormally deficient in multipolar ganglionic cells, or they are curtailed in size, shape changed, or polar appendices shortened. In paraplegia, with wasting of the muscles, post-mortems show degeneration and paucity of the multipolar cells in the anterior cornua of the spinal cord. Progressive muscular atrophy has the same record and an analogous condition exists in posterior spinal schlerosis. In all forms of polyuria brain changes are found, but more particularly in the semilunar ganglia of the sympathetic. The co-existence of exophthalmic goitre in kidney complications points to morbid changes in the nerve system of organic life, with one common origin, but with different manifestations. Recent experiments show that centres of nutrition are located largely in the spine and spinal ganglia. The morbific influence of impaired nutrition consequent on diseased nerve tracts is mueh more extensive than was formerly supposed. We see it in diseased conditions, in which are sudden metastases (such as exist in joints); also in deficiency of animal matter in bone; in want of tone in the surrounding tissues of joints, and consequently facility of luxation in articulating surfaces; easily produced ecchymosis in the insane, because of low vitality, and in atrophies of organs without antecedent inflammatory processes. In simple atrophy (not the degenerative form); in chronic arthritis; in local paresis, we often find no initiatory inflammatory symptoms, but simply a wasting of certain structures from want of capacity to assimilate building up material. It is true we often find inflammation or traumatic injury in the vicinity of these depreciating processes, but in these we can trace no direct connection between the two diverse conditions. It is evident the active state in a distant part has affected the nutritive nerve centres, and indirectly the influence of normal trophic supply is found wanting.

In one class the centric disease is primary, and in the other peripheral in its origin. These conditions are often seen paralysis following apoplexy. In a few days after the attack we frequently see an invasion of bedsores consequent on low vitality.