lyzed parts, which are in part of a very characteristic type. In the legs especially, the paralytic club-foot (talipes equino-varus) is a symptom long known. It is due to the fact that from paralysis of the peronei muscles, and of the tibialis anticus, the point of the foot constantly droops, and that a contracture is gradually developed in the antagonistic muscles of the calf, whose points of insertion are permanently approximated. In paralysis of the muscles of the calf, on the other hand, there arises a moderate degree of calcaneus from the contracture of the antagonists. In the arms and in the vertebral column, in paralysis of the spinal muscles, the most manifold and sometimes very considerable contractures and deformities may also arise, the chief cause of which is always to be referred to the contracture of unparalyzed antagonists, and to external mechanical conditions like weight and pressure."

I have perhaps, gentlemen, been somewhat tedious in my quotations and in my attempts to describe the exact causes of the deformities, especially of the lower limb, in infantile paralysis; but the fact that it is these deformities, which we have to treat, and that the question of tenotomy and other operative interference may occasionally arise in connection with them must, I say, be my excuse for such prolixity.

I do not believe the diagnosis of this affection to be at all a difficult matter as a rule. We have certain definite and well-marked symptoms pointing to this lesion of the nervous system and to no other.

The motor paralysis without alteration of sensibility; the non-response of the muscles to the faradic current, together with the absence of reflex action; the non-implication of the sphincter muscles of the bladder and rectum, or of those supplied by cranial nerves; and lastly the absence of any tendency to sloughing or to sores on the parts exposed to pressure, are points which separate distinctly this paralysis from those of cerebral origin, from general myelitis, and other affections of a similar character.

Formerly, infantile paralysis was supposed to be of peripheral origin, and to have its seat in the affected muscles.

Heine, as I have said, was the first to discover that it was an affection of cerebral origin, and that its seat was in some portion or another of the spinal marrow.

It consists of an acute inflammation affecting the gray matter of the cord, generally the anterior gray cornu of one side, though the white matter is sometimes more or less implicated.

In recent cases, according to Gowers, there is abnormal redness of the anterior gray matter, the vessels running from the surface to the cornu are distended with blood.

Under the microscope the capillaries are seen distended; there are extravasations in the grey substance and swelling of the neuroglia and ganglion cells, which become granular and lose their processes; the anterior bone becomes softened and filled with granular *débris* of disintegrated nerve cells.

These are the appearances to be met with in the earlier stages. Later on the bone becomes shrunken so as to be easily seen by the naked eye; the nerve cells disappear altogether and are replaced by fine connective tissue, and well-marked sclerosis occurs.

The anterior white columns are often in a state of degeneration, the neuroglia is thickened, the nerve fibres atrophied, and the columns themselves shrunken and small.

If the paralysis affects one arm, the corresponding anterior cornu in the cervical enlargement is atrophied; if one leg alone is affected, the atrophy will be found in the corresponding lumbar enlargement. This anterior cornuitis is the primary centre of the disease; the posterior columns and the brain being always unaffected in the post-mortem.

So the lesion of infantile paralysis is an acute myelitis of the anterior gray cornu, leading to circumscribed patches of sclerosis with complete destruction of the large ganglion cells and other nerve elements.

So much briefly for the pathological changes in the spinal cord.

The changes seen microscopically in the muscular fibres in well-marked cases are about as follows:

(1) The transverse striæ are less distinct, they are frequently broken, and the longitudinal fibres become more marked.

(2) The muscular fasciæ become composed entirely of longitudinal fibres, the transverse striæ rapidly disappearing.