10. Tissue culture of glioma.

with cinematographic demonstration

Fumiaki SATO
Department of Neurosurgery, University of Tokyo

Glia cells in tissue culture of 25 cases of gliomas, 4 cases of human foetus brain and 7 cases of kitten brain are classified into 4 types. They are spongioblast, astrocyte and oligodendrocyte. Their morphological characteristics are mentioned, with special reference to differentiation between astroblast and oligodendrocyte. They are distinguished by membranous expansions of perikaryon, which are usually seen in astrocyte and astroblast but not in oligodendrocyte and clearly demonstrated by cine. record.

According to cell types seen in tissue culture, gliomas are classified into astrocytic glioma, oligodendrocytic glioma, glioma of unknown glial cells and glioma of no growth in culture. Culture of anaplastic glioma consists of various types of glial cells and multinuclear giant cells. Comparing this classification with pathological classification, it is evident that the same type of glioma in pathological classification contains various types of glial cells. Tumor cells in the glioma whose pathological diagnosis is indistinct appear clearly in culture. Therefore in some cases, tissue culture of glioma will serve for its pathological diagnosis.

The degree of outgrowth of glioma cells in vitro is parallel to clinical malignancy of the glioma. The more clinical feature of glioma is malignant, the more outgrowth of the glioma in culture is abundant. Tissue culture of glioma is useful for the judgement of clinical prognosis of the patient.

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11. Embryological studies on the development of enteric plexuses in human digestive tract.

Eizo OKAMOTO, Takashi UEDA, Akiyoshi FUKUOKA
and Hiroshi SHIRASAKA
1st Surgical Clinic, Osaka University Medical School

We have studied on the morphological changes in the so called functional disorders of the digestive tract, such as congenital megacolon, congenital hypertrophic pyloric stenosis etc., and have found several organic changes in *myenteric* plexuses of these disorders.

In order to investigate the pathogenesis and etiology of these changes more thoroughly, embryological studies on the development of enteric plexuses should be emphasized.

Only a few, however, were reported on these subjects, especially in human materials. So we investigated the development of the enteric plexuses using the human embryos and fetuses obtained at the gynecological operation or curettage.

These materials were kept in 20% neutral formalin solution for one or more months and cut in 20 μ thick celloidin or frozen sections. The sections were stained by Bielschowsky-Suzukis' silver impregnation method.

I. The Order In The Formation Of Enteric Plexuses

At first, enteric plexuses appear from the oral part of digestive tract at a few weeks' gestation. At 7 weeks' gestation, enteric plexuses are seen in the wall of esophagus, stomach, duodenum and the oral part of small intestine, while in the anal part of small intestine, colon and rectum they are not to be found.

Even at 8th week they do not appear in the colon and rectum. It takes at least 3 months to form a completely developed plexuses at the tip of the rectum.

II. The Development Of Meissner's Plexus

In early gestation, the enteric plexuses formed in the outer mesenchym of the inner muscle layer are thought to be Auerbach's plexus. Neuroblasts, originated from the Auerbach's plexus, migrate across the inner muscle layer to form the Meissner's plexuses in the submucous layer of the digestive tract at 3 to 4 months' gestation. We confirmed the fact that the chromaffine cells of intestine are not participated in the development of Meissner's plexus.

III. The Development Of Ganglion Cells

The nerve cells in myenteric plexus are as yet completely undifferentiated up to 5th month and characterized by their non-visible cytoplasm and irregular, non-vesiculated nucleus.

At approximately 6th month, nerve cells begin to show slight differentiation. At 7th month relatively mature cells of large size with more cytoplasm are increased in number amongst as yet less differentiated nerve cells, and Dogiel type I and II cells are barely noticeable.

In general, the differentiation and development of each nerve cell should not occur uniformly but appear concomitantly with a decrease in the number of undifferentiated nerve cells with the progress of the age of the fetus, so that a considerable number of immature nerve cells are usually found at even late stage gestation or after full term birth.

IV. The Development Of Terminal Reticulum

At the 4th month, the nerve cells in myenteric plexus are as yet completely undifferentiated. Only fine single nerve fibers are seen in the inner muscle layer of the digestive tract, and appear to terminate in the neighbourhood of smooth muscle nucleus.

In accordance with the maturity of nerve cells, terminal reticulums first appear as richly anastomosing, fine neurofibrillar net structure at 7th month.

V. The Etiology Of Congenital Megacolon

We reported formerly from our histological investigation that the agenesis of enteric plexus in congenital megacolon was to be one of congenital malformation occurring in the distal part of the colon and rectum. This fact was confirmed at the autopsy of a 9 month stillbirth of Hirschsprung's disease.

The congenital agenesis of enteric plexus in the distal part of colon and rectum was thought to have occurred at least before 3 months' gestation in which stage Meissner's plexus began to be formed, branching off from Auerbach's plexus.

Meissner's plexus also failed to develop, because in a ganglionic segment of this disease in which Auerbach's plexus was lacking.

In addition, it was found out in this study that enteric plexuses were formed from the oral part of the alimentary tract toward the anal part. From this fact, we can easily understand the reason why the agenesis is apt to occur in the distal part of the colon and rectum.

VI. The Pathogenesis Of Congenital Hypertrophic Pyloric Stenosis

The pathogenesis of this disease was generally accepted that the thickening of pyloric sphincter was a work hypertrophy due to foregoing pylorospasm.

The thickened muscle, however, is not hypertrophy but hyperplasia, as the individual muscle fibers are not thickened but increased in number and their arrangement is so interlacing as seen in myoma. A malformation of a mass of bile duct epithelium aberrated in the thickened muscle layer, was also found in one of our cases.

In addition, nerve cells in myenteric plexus of the pylorus were apparently more immature than those found in other parts of resected stomach. This suggests that an increased functional state as "pylorospasm" has not occurred.

From the above mentioned histological findings, we can conclude that the thickened pyloric muscle is due to congenital malformation of pyloric sphincter and not hypertrophy. Pyloric stenosis probably occurred due to both organic malformation of pyloric muscle and immaturity, in other words, hypofunction of nerve cells of this segment.

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12. Muscle work and the hypothalamo-sympathico-adrenomedullary system.

Akira TAKAHASHI, Tsutomu MATSUI, Susumu TAKAGI, Tetsuya TAKAYANAGI, Kōhei YAMAMOTO and Itsuro SOBUE

1st Department of Internal Medicine, Nagoya University School of Medicine

In order to investigate the hypothalamo-sympathico-adrenomedullary system as one of the controller of the muscle glycogenolysis and the blood sugar level following muscle work, blood sugar (Hagedorn-Jensen's method), serum lactate (Barker-Summerson's method), WBC, eosinophiles and urinary excretion of adrenaline and noradrenaline (improved method of von Euler and Floding's fluorimetry) were measured for three hours after muscle work on a bicycle-ergometer.

In healthy persons in their thirties, transient hyperlacticemia and increase of urinary excretion of adrenaline and noradrenaline for the first one hour, and