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Post-mortem Changes in Mineral Salt Distribution in Nerve Cells.*

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An analysis, by microincineration, of the distribution of inorganic salts in anterior horn cells following temporary vascular occlusion of the spinal cord (Tureen[1] brought forward the necessity of examining the post-mortem changes in these elements in similar tissues. It seemed to be especially advisable also because of recent reports on the ash distribution in human nerve cells in various pathological conditions.

Tissues were removed from etherized and bled cats at intervals ranging from 5 minutes to 27 hours. One series of animals was permitted to remain at room temperature; a second series of animals was kept in the ice box at 60°F. for similar periods. Sectioning and incineration were carried out as suggested by Scott.[2] Alternate sections of the series were stained with hematoxylin and eosin as controls. The incinerated sections were studied by dark field illumination.

The findings will be related briefly in two parts, the first of which is the appearance of incinerated anterior horn cells after immediate fixation. The results are in general in accord with those for similar types of material described by Scott[3] and by Patton.[4, 5] The first consideration is to establish a "normal" picture—a task admittedly difficult since there is considerable variation in the appearance of the anterior horn cells even under optimum conditions. In general the ash residue of the well-fixed anterior horn cell is uniformly distributed throughout the cytoplasm. This mineral is in small deposits approximately 1 to 2 microns in diameter. It is in this cytoplasmic ash that the greatest variation occurs. In some cases, for example, the remains of the Nissl substance are clearly discernible, in others not. It is as yet impossible to assign this variation to a

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1 Tureen, L. L., *Arch. Neur. and Psych.*, 1936, **35**, 798.

2 Scott, Gordon H., *Protoplasma*, 1933, **20**, 133.

3 Scott, Gordon H., *Am. J. Anat.*, 1933, **53**, 243.

4 Patton, W. E., PROC. SOC. EXP. BIOL. AND MED., 1933, **31**, 195.

5 Patton, W. E., *Am. J. Path.*, 1934, **10**, 615.

definite physiological state of the cell. Some cells show a reasonably dense mineral deposition in the background of the cytoplasm, others will be revealed as having but little of it. The reason for this condition is likewise obscure. However, it is felt that it is not likely to be associated with the technical procedure particularly with fixation. Portions of spinal cord prepared by the Altmann-Gersh frozen dehydration method show the same general characteristics as those fixed in alcohol-formalin.

The nuclear minerals are, as is the case in most other cells, distributed in the same pattern that the chromatin material assumes in stained sections. The nucleolus is represented by a massive ash deposit of size comparable to that body in the stained section. Frequently there is at the periphery of the cell a definite condensation of minerals thought to be largely due to the shrinkage of the cell during fixation.

In contrast to this picture of mineral distribution in the cell fixed immediately after death, we have a series of stages which indicate that there is a more or less progressive loss of inorganic salts with time. The demineralization is more marked and more rapid in the tissues left *in situ* at room temperature than in those that were kept chilled. The post-mortem loss of minerals starts to be noticeable at about 3 hours after death and reaches an equilibrium at 15 to 20 hours. The first noticeable loss of ash is manifested in the cytoplasm which shows progressively less mineral with time elapsed between death and fixation. This loss of mineral continues until it is difficult to distinguish where anterior horn cell leaves off and interstitial tissue begins. The Nissl substance loses its minerals early in the process, about 3 to 4 hours sufficing to make it impossible to identify with certainty. The nucleus resists post-mortem salt loss longer than other parts of the cell. Some specimens showed clearly recognizable nuclei even after 27 hours at room temperature.

The delay of post-mortem salt loss occasioned by the chilling (10°C) of the bodies in the ice box was interesting in that the changes in the cells lagged behind the room temperature tissues by 7 to 8 hours consistently. That is to say, one could expect to find the same condition in a 7-hour room temperature specimen as in a 14- to 15-hour ice box specimen. It is of some interest, too, that the interstitial cells acquired considerable mineral from the tissue fluid while the anterior horn cells were progressively losing salts. As the nerve cells lost their organic elements the tissue fluid gained them. This exchange apparently ceased at 15 to 20 hours after death at room temperature.