[redacted] AGE: 50 NP-43-39
N-43-44

AUTOPSY DATE: 1/28/43

HOURS POST MORTEM:

DATE BRAIN CUT: 2/10/43

WEIGHT OF BRAIN: 1290 grams

CLINICAL DIAGNOSIS: Hypertensive encephalopathy; possible lead poisoning.

ANATOMICAL DIAGNOSIS: Confluent lobular pneumonia.

REPORT OF THE LABORATORY OF NEUROPATHOLOGY

GROSS DESCRIPTION: The dural cap has not been preserved.

The right frontal pole has been removed for lead studies. Viewed from above the right posterior parietal region appears a little fuller than the corresponding region on the left, and the gyri are somewhat flattened. In the right parietal occipital region there is a little grayish discoloration of the sulci, suggestive of a localized meningeal reaction. The mesial portion of the lobe just anterior to the parietal occipital fissure is soft to touch. This region is broadened and the sulcal markings are less prominent.

The leptomeninges are not otherwise remarkable. The vascular pattern is normal, the distribution of veins not being remarkable.

Inspection of the median cleft shows nothing remarkable, except for a definite fullness of the mesial portion of the parietal lobe running backward to the occipital parietal junction. The occipital lobes are not remarkable. The cerebellum has a suggestion of a pressure cone. The brain stem has been cut very short, but what there is left is not remarkable in appearance. The pons is not noteworthy.

The leptomeninges at the base are slightly thickened. There is no uncal herniation.

The vessels at the base are of large calibre, the walls are moderately thickened, but there are no frank plaques. The distribution of the vessels of the circle is normal.

Vertical coronal sections were made through the brain at 1.5 cm. thicknesses. The left hemisphere throughout appears normal, and the left lateral ventricle is of about normal size, perhaps somewhat small.

Throughout all the sections of the right hemisphere, the white matter appears broader than the corresponding white matter on

the left and the white matter is soft to touch on this side. The demarcation between gray and white is sharp, except in the posterior parietal region. In Blocks P-1, 2, 3, and 4 there is superficial softening of the tissues, and in the last named block there is hemorrhagic softening. The right lateral ventricle is somewhat compressed throughout.

In Block P-1 there is a striking disappearance of the upper sphenoidal fissure and in the mesial portion of the temporal lobe in the immediate vicinity of the obliterated fissure there is a grayish tissue, which zone corresponds roughly to a similar zone on the left in Block P-2.

Multiple coronal sections were made through the disarticulated cerebellum and brain stem. These show the presence of a massive hemorrhage involving the region of the left dentate nuclei and spreading mesially across the midline. The greatest measurements are 4 x 1.

Sections Taken: Survey from the right hemisphere of P-1 and from the zone of hemorrhage in the cerebellum.

Photographic Indications: Coronal sections from the cerebellum to show the general nature of the edema of the right hemisphere and the extent of the hemorrhage in the cerebellum.

State of Fixation: Fair

Brain saved.

Gross Neuropathological Diagnosis: Massive left intracerebellar hemorrhage; edema of the right hemisphere, associated with softening in the right parietal region.

MICROSCOPIC: Survey sections were taken from the fronto-temporal region of the cortex, from the centrum semiovale, basal ganglia, pons, and cerebellum. All sections were stained with cresyl violet and H. & E.

The microscopic examination revealed the following changes:
(1) Leptomeninges: The pia arachnoid was thickened and hyperplastic in some areas. The thickening was due to distention of the meshes, infiltration with a variety of cells and hyperplasia of connective tissue. The distended meshes contained many scattered cellular elements, most of them fibroblastic in nature. In some areas there were large accumulations of macrophages. The larger blood vessels appeared thickened and moderately sclerosed. The smaller veins disclosed a marked proliferation of the endothelial and adventitial cells.
(2) Parenchyma: The changes in the parenchyma consisted of scattered areas of softening, and hemorrhages. The former were localized chiefly within the subcortex and were in different stages of organization. Some of them disclosed the characteristic feature of acute anemic infarction; others more advanced organization and glial repair.

In addition there was to be seen a diffuse ischemic degeneration of the nerve cells; some of them especially in the deeper layers were surrounded and partly invaded by glia cells (satellitosis and neuronophagia). In some areas the ganglion cells disclosed a far advanced liquefaction. The architecture of the cortex appeared changed and unrecognizable in various regions. The white substance showed a marked swelling of the oligodendroglia cells, associated with a slight rarefaction of the tissue. In some areas there were signs of progressive changes of the glia, swelling of the nuclei associated with an abundance of chromatin and cytoplasmic processes with a tendency to proliferation and accumulation in rows or clusters.
(3) Vascular system: Very striking changes were disclosed within the capillaries. They showed marked proliferative and progressive phenomena as evidenced by endothelial and adventitial proliferation and new formation of capillaries. The majority of the capillaries were made up of swollen and proliferated endothelial cells which contained chromatin-rich nuclei with an appreciable amount of cytoplasm at their poles. They were frequently filled with greenish granules and were sometimes surrounded by a few macrophages. In some areas there were to be seen newly formed capillaries with brightly stained hypertrophied endothelial cells with elongated cell bodies. Many of them disclosed numerous budding formations. In some areas the entire field appeared to be covered with an abundance of proliferated capillaries containing hyperplastic endothelial and adventitial cells. The walls of the larger vessels were appreciably thickened, the thickening being due to proliferation of the adventitial cells. The endothelial cells were seldom increased in size and did not reveal marked proliferative changes. Some of the larger blood vessels disclosed a moderate degree of arteriosclerosis. A few of the smaller veins showed a slight fibrosis and hyalinization of the vessel wall. Like the capillaries the larger vessels did not disclose perivascular infiltration with hemotogenous elements.

SUMMARY: The changes in different areas of the brain tissue are both parenchymatous and interstitial. The former, diffuse degenerative changes of the nerve cells throughout associated with circumscribed areas of softening and tissue necrosis, are essentially regressive or degenerative. The interstitial changes are definitely progressive and proliferative in character, (hyperplastic and proliferative phenomena in the endothelial and adventitial cells, new formation of capillaries, hyperplastic changes of the leptomeninges).

COMMENT: While the parenchymatous changes are not specific in their character and may resemble those to be found in any type of structural vascular lesions, (arteriosclerosis, hypertensive encephalopathy) the mesodermal changes of the capillaries and leptomeninges are more characteristic and significant and are compatible with lead encephalopathy. The latter is characterized chiefly by prevalence of productive and proliferative phenomena in the endothelial and adventitial vascular tissue and in the leptomeninges.

DIAGNOSIS: Lead encephalopathy.

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