averaged at each point in the brain. Noticeable potentials were recorded in the following areas in the upper brain stem: ventromedial part of substantial reticularis mesencephalica, n. commissurae posterioris, centre médian, medial part of ventrobasal complex of the thalamus, Forel's field, n. reticularis, posterior and lateral hypothalamus, and ventral part of n. caudatus. Evoked potentials on the cortex were recorded in the lateral part of the first somatosensory and diffusely in the association areas as well. The potentials in these areas were of 15–30 msec in latency and less than 30 μv in magnitude.

b-2. Intracerebral Paraffin Injection and Changes in Fibrinolytic Activity of Cerebro-Spinal Fluid in Dogs

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Previous research\(^1\) carried out on dogs has indicated that injection of coagulating blood (and paraffin wax) into the brain can lead to rapid death; and that, at such times, there is a rapid acceleration of the fibrinolytic activity of cerebro-spinal fluid (CSF) probably due to release of tissue activator from around the "haematoma". Administration of trans-form aminomethylcyclohexane carboxylic acid (t-AMCHA)\(^2\) proved to have a beneficial effect, by its controlling action on fibrinolytic activity.

In the present study experiments were undertaken to further investigate the effect of injection of paraffin within the brains of dogs, in an attempt to elucidate the influence of a space-occupying intracerebral mass (and consequent increased intracranial pressure) on changes in the fibrinolytic activities of CSF and peripheral blood. In 25 healthy animals, a hole was first drilled into the temporal bone (2 cm anterior and 1.5 cm superior to the external auditory meatus) and varying amounts of paraffin (pre-melted) injected towards the base of the skull, just before solidification (c. 52°C.). Samples of CSF and peripheral blood were withdrawn at regular intervals and variations in the fibrinolytic activities of their euglobulin fractions estimated on standard and heated fibrin plates. (The remarks below refer directly to observations on standard plates: these were paralleled in the heated plates.) After death, autopsies were carried out and the masses of intracerebral, ventricular, and sub-dural paraffin estimated. The results allowed the dogs to be divided into two quite distinct groups. On the one hand, the 12 dogs dying within 3 days in general exhibited sharp rises in CSF fibrinolytic activity, and autopsies revealed comparatively large masses of intracerebral paraffin (128–1, 980 mg: average 520 mg). In the other 13 dogs, death (or sacrifice)
was in excess of 5 days after surgery, CSF fibrinolytic showed no sharp overall increases, and autopsies revealed comparatively small masses of intracerebral paraffin (0–220 mg: average 65 mg). There was no clear distinction in ventricular paraffin between the two groups, the figures being as follows: 0–2,045 mg (average 360 mg) in dogs dying rapidly, and 0–1,260 mg (average 570 mg) in dogs living more than 5 days. Furthermore, in the case of sub-dural paraffin, a similar lack of distinction was apparent: 0–1,640 mg paraffin (average 330 mg) in dogs dying rapidly, and 0–1,282 mg (average 310 mg) in dogs living more than 5 days. The fibrinolytic activities of peripheral blood in general showed irregular changes with no overall trend, except that in the single dog dying within 1 hour there was a rapid increase until death.

On the basis of these results, it is concluded that the volume of paraffin actually injected intracerebrally (as opposed to intraventricularly or sub-durally) is probably the most important deciding factor in the initiation of increased CSF fibrinolytic activity and rapid death. The limiting amount for a high rate of rapid deaths would appear to be about 120 mg (0.17 c.c.). (This figure compares with a total approximate volume of brain tissue of 80 c.c.). Concerning the mechanisms to changes induced in the brain tissue, it is thought that the injection of paraffin (intracerebrally) may lead to local disturbance of circulation and have a close relationship to the release of brain tissue activator into the CSF, along with damage of the tissue itself.

References

1) Mihara Hisashi, Fuji Tadao, & Okamoto Shosuke: Fibrinolytic Activity of Cerebrospinal Fluid and the Development of Artificial Cerebral Haematomas in Dogs; accepted for publication in “Thromb. Diath. Haemorrh.”

b-3. The Control of Antidiuretic Hormon (ADH) by Anterior Hypothalamus:

1. Unit Activity of the Supraoptic and Paraventricular Nuclei

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That there are many areas of the brain controlling the output of ADH is attested to by disturbances in aproprated ADH release with many diseases of the nervous system. Verney introduced one concept of the “osmoreceptor” in 1947 which had been supported by anatomical studies, and it is concerned that