A Comparison of Function of the First and Second Somatosensory Areas of the Dog[1]

It was found in a previous investigation that unilateral removal of the cerebral somatosensory receiving areas in the posterior sigmoid (SI) and anterior ectosylvian (SII) gyri of the dog caused transient impairement of a preoperatively established tactile conditioned reflex[2]. The impairment consisted in a reduction of the ability to elicit the reflex from the peripheral fields corresponding to the areas removed. The finding was taken to indicate that elements located in these areas participate in the conditioned reflex under normal conditions. It is, on the other hand, not known whether one area or both participate. Thus, it has now been examined whether a similar postoperative impairment of a comparable tactile conditioned reflex can be caused by removing SI or SII alone.

Dogs have been trained to react to a light tactile stimulus or a visual stimulus by pressing a button (33 mm diam eter) with the nose. The tactile stimulus consisted of puffs of air (100 msec duration, 3/sec) delivered through 1 of 4 nozzles, 2 of which were attached to each hindlimb; tibia and paw. The visual stimulus, used for control purposes, consisted of flashes of white light of the same duration and frequency as the tactile stimuli. The stimuli (14 each session, 3 tactile through each nozzle and 2 visual) were presented in pseudo-random order and at pseudo-random intervals. Correct responses (button-pressing performed during a stimulation not exceeding 10 sec) were reinforced with food; the dogs not having been fed for the last 24 h preceding a session. A 'blind', tactile stimulus, representing all parameters of the ordinary stimulus except for its tactile component, was used to check that the conditioned response was an effect caused by the activation of peripheral cutaneous receptors.

Lesions were made when a dog was performing accurately at a stable level; i.e. over 90% correct responses for the last 8 preoperative sessions. The lesions were restricted to the hindlimb area of SI or SII which was removed by subpial suction under aseptic conditions. The somatosensory areas were mapped with electrophysiological technique in each animal at the beginning of the operation.

In 6 dogs the following lesions were made initially: SI unilaterally, SI bilaterally, SII unilaterally, SII bilaterally in 1 animal and SI and SII together unilaterally in 2 animals. Of these animals only the 2 where both SI and SII had been removed showed any postoperative impairment (i.e. incidence of postoperative responses less than 50% of preoperative). It thus appeared as if SI and SII could replace one another functionally and that removal of both was necessary to obtain impairment[3]. However, when further lesions were made in these animals a different picture emerged.

For example, removal of the remaining SII in the dog where SII had already been removed unilaterally, caused transient impairment of the reflexes from the limb contralateral to the second operation. Similarly, contralateral impairment appeared after removal to the remaining SII of 1 dog which had recovered from a previous removal of SI and SII together on the other side. On the other hand, removal of the remaining SI in dogs with previous unilateral lesions in SI alone, or both SI and SII, caused no impairment (i.e. incidence of postoperative responses higher than 90% of preoperative). Likewise, unilateral removal of SII after bilateral removal of SI caused impairment, whereas unilateral removal of SI after bilateral removal of SII did not.

Thus postoperative impairment of the type observed previously after removal of both somatosensory areas[2] can be caused by removing SII but not by removing SI alone. It should, however, be pointed out that although this finding indicates a special significance for SII a role for SI is not excluded. For example, whereas unilateral removal of both SI and SII in the previous[2] and present material has always caused postoperative impairment, unilateral removal of SII in the present material, in one instance did not. The absence of impairment after bilateral removal of SII also remains to be explained. The present results agree with previous reports that the ability of dogs[4] and cats[5] to differentiate passively received tactile stimuli is impaired by lesions of SII but not SI.

Zusammenfassung. Ausgehend von der Annahme, dass beim Hund die somatosensiblen Gebiete im Gyrus sigmoideus posterior (SI) und Gyrus ectosylvius anterior (SII) an einem taktil bedingten Reflex beteiligt sind (Defekte am Reflex nach Abtragung des Gebietes), wurde die relative Bedeutung beider Gebiete durch Teilabtragungen studiert: Auftreten der Defekte nach Abtragung von nur SII, nicht aber von SI.

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Dehydration of the Brain by Intra-Peritoneal Injections of Hyper-Osmotic Solutions of γ-Aminobutyric Acid and DL-α-Alanine

Results of previous investigations have indicated that i.p. injections of large volumes of hyper-osmotic solutions of γ-aminobutyric acid (GABA), or other substances, can offer protection against convulsions produced by hyperbaric oxygen or by administration of chemical convulsants[1-4]. Although it was suggested originally that the effect of hyper-osmotic GABA was 'specific' to GABA and related to its entry into the brain[1], further results have indicated that this treatment causes significant dehydration of the brain[3]. Therefore, it has been postulated that the anti-convulsant action of hyper-osmotic GABA is related mainly to dehydration of the brain rather than to its penetration into the brain, and that this action is not specific for GABA[3]. Since injections of hyper-osmotic solutions of α-alanine dehydrate the brain and protect against convulsions at least as well as hyper-