EXPERIMENTAL OCCLUSION OF DURAL SINUSES

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Experience of one of the authors (A.M.M.) in the surgical treatment of wounds of the dural sinuses during the recent Korean conflict, increasing interest in the circulatory anatomy of the brain, and a paucity of specific experimental information led to this study. Efforts were directed toward accumulation of experimental data with regard to questions arising from sudden disruption of the major superficial venous system as encountered in penetrating cranioencephal trauma and also in the presence of space-occupying intracranial lesions.

TECHNICAL DATA

Twenty-eight Macacus rhesus monkeys, weighing from 6 to 10 lbs., were used. The dural sinus circulation was exposed by employment of biparietal or bifrontoparietal craniectomy. Intravenous pentobarbital (30 mg./kg.) was used as anaesthetic agent. Surgical alteration of the superficial venous outflow was accomplished by ligation, and by intraluminal insertion of skeletal muscle or paraffin.

Cerebral venous circulation was demonstrated before (Fig. 1), during, and after the experiments by direct sinography with 3–5 cc. of 35 per cent and of 75 per cent Diodrast. Inasmuch as direct sinography did not suffice in every instance to demonstrate collateral circulation, additional bilateral carotid arteriography was carried out in 5 animals.

Unipolar and bipolar electroencephalographic recordings were made in the parietal regions of 5 animals before, during, and after sinus occlusion, using an Offner 4-channel electroencephalograph. Simultaneous cerebrospinal fluid pressure readings were obtained through a #18 spinal needle inserted in the cisterna magna and connected to a water manometer.

Gross and microscopic examinations of specimens of brain and dural sinus were obtained from 16 animals, sacrificed from 5 days to 6 months after completion of the experiment. All types of occlusion were represented.

Microscopic studies of dural sinuses containing intraluminal skeletal muscle were made.

EXPERIMENTAL DATA

I. Occlusion of Rolandic and Bridging Veins (8 monkeys). Bilateral occlusion of the Rolandic veins was produced in 3 animals. This resulted im-

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Fig. 1. Monkey 345. Sinogram without alteration of venous system.

mediately in intense but transitory bilateral cortical swelling. Simultaneously marked distention of the superficial venous channels took place. A neurological deficit did not develop.

In 3 animals all venous tributaries of the superior longitudinal sinus were interrupted bilaterally. Again transitory, intense, bilateral cortical swelling and distention of the superficial cortical veins took place. The animals survived without development of a demonstrable neurological deficit.

In 2 animals unilateral interruption of the venous tributaries to the superior longitudinal sinus was produced. This resulted in homolateral edema and in homolateral distention of cortical veins. These animals also survived without development of a demonstrable neurological deficit.

Attempts at demonstrating the collateral network by means of sinography failed in all 8 animals. For that reason bilateral carotid arteriography was performed in 5 of the 8 monkeys in an effort to visualize in the venous phase the collateral network produced by occlusion of all venous tributaries to the superior longitudinal sinus. This was successful in 1 animal only (Fig. 2). The animal, however, expired immediately following the injection of the dye, which might account for the radiographic success. Postmortem examination of the brain of all 8 animals failed to reveal any significant gross or histological pathology.

II. Occlusion of Superior Longitudinal Sinus Anterior to Rolandoic Veins (3 monkeys). Occlusion of the superior longitudinal sinus anterior to the Rolandic veins was produced in 3 animals by ligation with 0000 silk. Occlusion of the sinus at site of ligation was demonstrated in each instance by sinography.
Ligation did not produce an alteration in the cerebrospinal fluid pressure. Electroencephalographic studies immediately after ligation revealed a variable momentary burst of slowing with elevations of slight amplitude. Postmortem examinations did not reveal any gross or microscopic pathological changes.

III. Occlusion of Superior Longitudinal Sinus Posterior to Rolandoic Veins (16 monkeys). Occlusion of the superior longitudinal sinus posterior to the Rolandoic veins was produced in 16 monkeys. This was accomplished by ligation in 8 animals, by injection of hot paraffin into the sinus in 3 animals, and by insertion of skeletal muscle into the lumen of the sinus in 5 animals. That portion of the sinus that had been occluded by insertion of skeletal muscle was excised in all 5 monkeys after a period of 15 days and the sinus was ligated proximally and distally. In each instance, occlusion of the posterior third of the superior longitudinal sinus was confirmed by sinography. The uniform response to occlusion consisted of cerebral edema and profound bilateral engorgement of cortical veins. A demonstrable neurological deficit did not occur.

In 5 animals electroencephalographic studies and determination of changes in the cerebrospinal fluid were carried out in order to obtain further information on the effect of sudden alteration in venous outflow. In each instance slow waves of high amplitude (200 to 300 μV., 3 to 5 per sec.)
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occurred instantaneously and returned gradually, within 20 to 90 sec., to the baseline pattern (Fig. 3). Associated with the appearance of the slow waves of high amplitude was a rise in cerebrospinal fluid pressure, with increases ranging from 70 to 100 mm. H₂O. A plateau of 70 to 100 mm. H₂O above the pre-occlusion pressure was reached in each case within 1 to 2 min. These levels remained unchanged at termination of the experiments 4 hours later.

Injection of hot paraffin into the lumen of the sinus produced immediate occlusion in 3 animals. At periods of 2, 4 and 8 weeks respectively, the paraf-

![Fig. 3. Monkey 10. Electroencephalographic and cerebrospinal fluid changes before, during and after occlusion of the superior longitudinal sinus posterior to Rolanic inflow.](image)

fin was removed. Formation of a thrombus was not induced by this method of occlusion. Removal of the obstructing paraffin resulted in restoration of normal venous outflow demonstrated by sinography.

Insertion of skeletal muscle into the lumen of the sinus resulted in complete obstruction which was demonstrated by sinography 1 week following occlusion. Relief of the obstruction and recanalization had taken place in all 5 animals when sinography was repeated 2 weeks after occlusion (Fig. 4). During these 2 weeks no demonstrable neurological deficit developed. The sinus was then resected at the site of muscle insertion for purpose of histological studies. It was of interest to note that ligation of the sinus proximally and distally did not produce a neurological deficit.

Histological studies revealed conversion of the muscle into collagenous scar tissue which was firmly adherent to the wall of the sinus except at the site of recanalization (Fig. 5). These sites were characterized by islands of
partly degenerated muscle fibers surrounded by spaces which were lined with endothelial cells and which contained blood elements (Fig. 6). Large spaces between scar tissue and wall of the sinus appeared to be continuous with the sinus. There was no evidence of thrombus formation.

Sinography was carried out at varying intervals following permanent occlusion of the sinus in 9 monkeys, demonstrating the development of collateral routes about the site of obstruction (Fig. 7).

A neurological deficit was not observed in any instance. Gross and micro-

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**Fig. 4. Monkey 7.** (A) Sinogram immediately following insertion of muscle occluding the superior longitudinal sinus posterior to Rolandic veins. (B) Sinogram 9 days following occlusion. Note appearance of Diodrast distal to site of occlusion. (C) Sinogram following excision of superior longitudinal sinus containing muscle.

**Fig. 5. Monkey 7.** Section through superior longitudinal sinus demonstrating endothelial lining of muscle. (×200)
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Fig. 6. Monkey 130. Section of superior longitudinal sinus showing muscle infiltrated with red blood cells and enlarging channels. Sinus excised 2 weeks following occlusion with muscle. (220×)

scopic examinations did not reveal any pathological changes other than those described in the presence of skeletal muscle.

IV. Occlusion of Lateral Sinuses (1 monkey). Ligation of both lateral

Fig. 7. Monkey 394. Sinogram 3 months following occlusion by ligation of superior longitudinal sinus posterior to Rolandic vein. Large numbers of collateral channels are present at site of occlusion.
sinuses was performed just proximal to the torcular Herophili. Electroencephalographic wave patterns and changes in cerebrospinal fluid pressure followed closely those seen in the presence of occlusion of the superior longitudinal sinuses. Time factors were similar, with the electroencephalogram returning to the pre-occlusion pattern within 70 sec. The cerebrospinal fluid pressure, when recorded after 4 hours, at time of termination of the experiment, remained elevated as had been observed in the presence of occlusion of the superior longitudinal sinus. Subsequent pressure readings, though desirable, were not obtained because it was felt that conditions would not be comparable.

V. Comments on Experimentally Attempted Induction of Sinus Thrombosis.

1. Intraluminal injection of hot paraffin, though productive of immediate permanent occlusion of the sinus, did not lead to formation of a thrombus.

2. Intraluminal insertion of skeletal muscle, though productive of transitory occlusion of the sinus, did not lead to formation of a thrombus.

3. Efforts to produce a thrombus by direct trauma applied to various portions of the superior longitudinal sinus in the presence of artificially induced hypotension failed to result in formation of a thrombus.

DISCUSSION AND CONCLUSION

In man the analysis of neurological sequelae to occlusion of dural sinuses or their tributaries has been complicated by associated gross cerebral lesions and simultaneous pathological alterations of collateral routes. Nevertheless Holmes and Sargent\(^3\) have been able to define a neurological syndrome occurring in man with occlusion of the superior longitudinal sinus posterior to the Rolandic veins. Merwarth\(^5\) described the Syndrome of the Rolandic Vein. The recent observations of Swanson and Fincher\(^7\) throw additional light upon the nature of the neurological deficit resulting from obstruction of the sinus. All authors agree that the superior longitudinal sinus can be ligated with impunity anterior to the point of entrance of the Rolandic veins. This has been confirmed by an analysis of 112 wounds of the dural sinuses incurred in the Korean War.\(^4\) There is considerable evidence, in the latter analysis and in Swanson and Fincher’s observations, that the dire neurological sequelae to occlusion of the dural sinus posterior to the Rolandic veins may be temporary, and that extent and duration of the neurological deficit may depend on the local damage that has been produced.

Beck and Russell\(^5\) have attempted to produce thrombosis in the superior longitudinal sinus of dogs and cats by introduction of agents usually productive of thrombotic lesions elsewhere. A neurological deficit did not result in those animals. Swanson\(^6\) was unable to produce thrombosis of the superior longitudinal sinus of the monkey by ligation. Even after resection of the posterior longitudinal sinus of 1 monkey, a neurological deficit did not develop during a 9-month follow-up period.

The present study confirms Beck and Russell’s observation in dogs and cats and those of Swanson in the monkey. Thrombus formation did not
occur in any of the animals. A neurological deficit did not result from occlusion of the Rolandoic veins or of the superior longitudinal sinus posterior to the Rolandoic veins.

The experimental evidence of cortical swelling and venous engorgement, associated with an increase in the cerebrospinal fluid pressure and with changes in the electroencephalographic pattern, indicate temporary alterations similar to those expected in man. The apparent rapid development of collateral venous outflow, as demonstrated in the monkey, seems to prevent permanent alterations. These experimental studies lend added weight to Swanson and Fincher's observations7 and to the analysis of war wounds involving dural sinuses to which we have referred at the outset of this discussion.4

As in the monkey, the electroencephalogram may be helpful in man in assaying the immediate effect of occlusion. Transient occlusion followed by gradual improvement of the electroencephalographic record may well provide a measure of the ability of the collateral circulation to return the pooling venous blood to the heart.

By the establishment of adequate venous drainage, Cairns et al.2 have shown that a large muscle stamp does not necessarily interrupt the flow of blood through a sinus so repaired. The analysis of wounds of the dural sinuses incurred in the Korean War4 revealed 1 instance of secondary thrombosis in 15 lacerations of the sinus that were repaired with a muscle stamp. That analysis furthermore suggested that the occurrence of secondary thrombosis depends not so much on the method of repair as on the maintenance of blood volume and pressure before, during, and immediately after operation. Beck and Russell1 have pointed out the effectiveness of reparative processes ending in organization and recanalization of the occluding muscle or of the cotton wool in the dog. The response of intraluminal striated muscle in the present series of experiments confirms the concept of recanalization. The value of the use of muscle stamps as method of choice for the repair of extensive lacerations of the sinus has been suggested previously on the basis of clinical observations. The current study demonstrates the process of recanalization which takes place in a sinus that has been repaired with a muscle stamp.

REFERENCES

6. Swanson, H. S. Personal communication.