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AJNR Am J Neuroradiol 1985, 6 (1) 19-22

<http://www.ajnr.org/content/6/1/19>

This information is current as
of April 19, 2024.

Experimental Production of Superior Sagittal Sinus Thrombosis in the Dog

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Prior efforts at experimental production of superior sagittal sinus (SSS) thrombosis generally have been unsuccessful. An experimental method was devised of inducing SSS thrombosis in the dog by use of double-lumen balloon catheters. Two balloon catheters, introduced into each internal jugular vein, are finally positioned at the torcular Herophili. The resulting stasis in the SSS, initiated by the inflated balloons, is augmented by injecting thrombin into the SSS. The stasis and the intraluminal thrombin cause thrombosis, which can be visualized on computed tomography (CT). This method may be used to study the natural history of SSS thrombosis as well as establish its CT and magnetic resonance imaging characteristics.

Superior sagittal sinus (SSS) thrombosis is an underdiagnosed disease [1-6] and is usually a complication of other primary disease processes (e.g., hypercoagulable state, meningocerebritis, asphyxia or dehydration in the neonate, and cardiovascular disease). The clinical symptomatology caused by SSS thrombosis is thus frequently superimposed on that of the primary disease, that is, the spectrum of its clinical presentation is not clear. Also, little is known about the chronology of the cerebral lesions produced by SSS thrombosis and its natural history: (1) the role of collateral venous pathways, (2) chronology and degree of recanalization, and (3) the incidence and extent of concurrent involvement of the adjoining cortical veins. These aspects of the disease are best studied in the animal in a controlled environment. To date, no noninvasive experimental procedure for inducing SSS thrombosis has been devised. Prior work consists mainly of ligating the dural sinuses and observing the effect on the brain of the sacrificed animal. In this setting, we devised a relatively innocuous experimental method for inducing SSS thrombosis in the dog.

Materials and Methods

The dogs were anesthetized with intravenous sodium pentobarbital. A cuffed endotracheal tube was positioned in the trachea and the animal ventilated with room air via a Harvard positive pressure respirator.

The method of SSS thrombosis production involved placing two double-lumen balloon catheters (Balloon Wedge Pressure Catheter, model JC-211, Critikon, Tampa, FL) into the internal jugular veins so that their tips were positioned at the torcular. First the jugular vein was exposed by cutdown at the level between the mandibular condyle and the external acoustic meatus. A small slit was made in one wall of the exposed internal jugular vein for introducing a 0.021 inch (0.53 mm) inner-diameter guide wire (Cook, Bloomington, IN). The guide wire was pushed gently until it crossed over to the contralateral transverse sinus (fig. 1). At this point, the double-lumen balloon catheter was threaded over the guide wire and was gently maneuvered forward until it could be palpated on the other side at a level between the external acoustic meatus and the mandibular condyle, where a venous cutdown was made to expose the catheter. The guide wire was then passed through the catheter until its end protruded a few centimeters beyond the catheter tip and out of the neck. The second

Received February 23, 1984; accepted after revision June 16, 1984.

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AJNR 6:19-22, January/February 1985
 0195-6108/85/0601-0019 \$00.00
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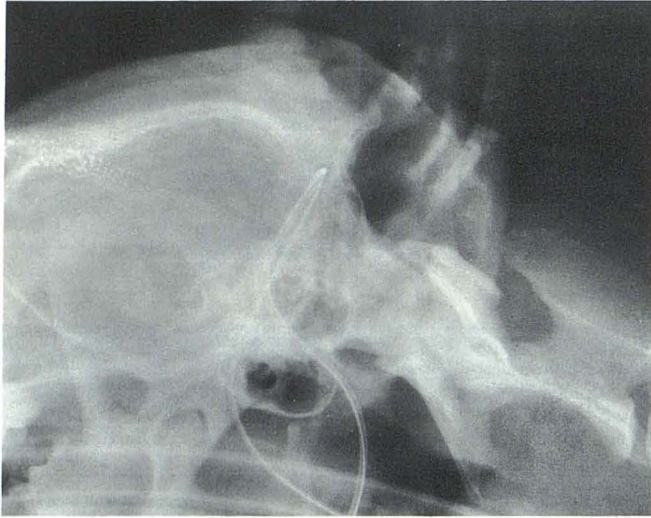


Fig. 1.—Lateral radiograph of dog skull. Guide wire-catheter combination crosses from one side to the other.

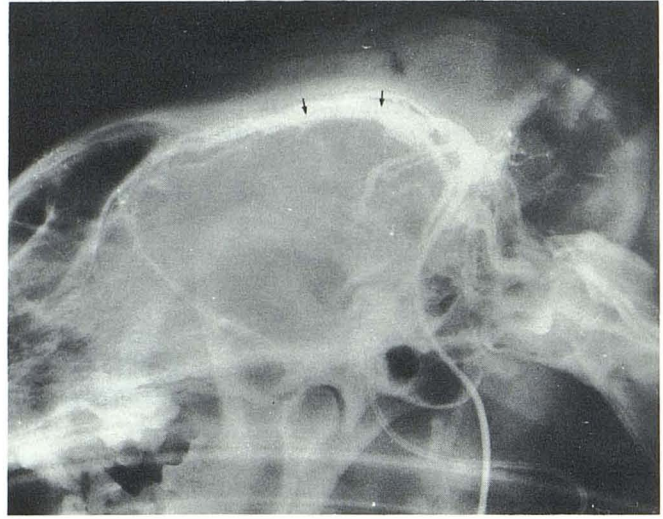
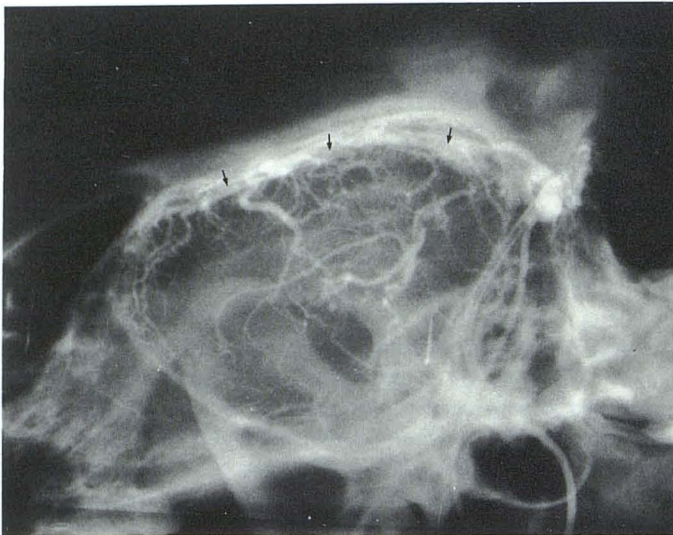


Fig. 2.—Normal superior sagittal sinogram (arrows) with noninflated catheter balloons at torcular. Minimal to no retrograde opacification of superficial cortical veins.

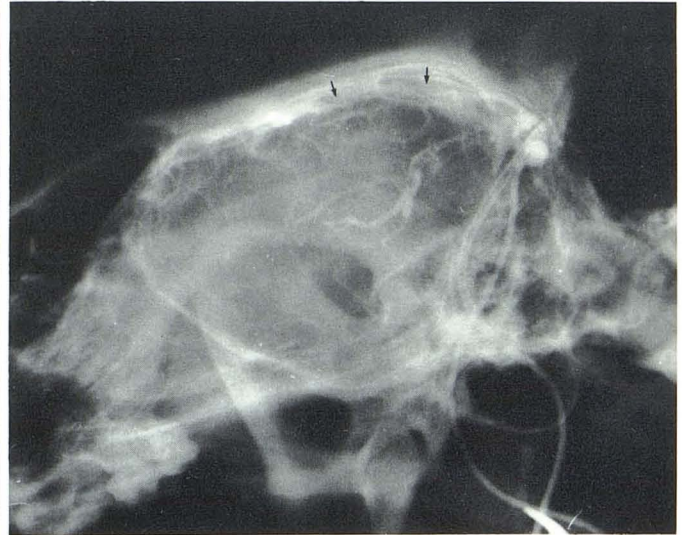


A

Fig. 3.—Superior sagittal sinogram after catheter balloons placed at torcular had been inflated to maximum capacity of 0.6 ml and thrombin injected into SSS. A, 1 sec after injection of contrast medium into SSS. Thrombosis is filling defect (arrows) within opacified sinus. Extensive retrograde filling of superficial

catheter was threaded over the protruded segment of the guide wire. By gently pulling on the first catheter and pushing the second catheter simultaneously, the tips of both catheters were brought together end-to-end at the torcular. The guide wire was then pulled out. The correct catheter tip position at the torcular was ascertained by injecting 0.25–0.5 ml of water-soluble contrast medium through each catheter. Then, 0.5–1 ml of the contrast agent was injected slowly into one catheter to obtain a control sinogram (fig. 2). Each catheter balloon was then inflated to its maximum capacity of 0.6 ml to induce stasis within the sinus. To facilitate thrombosis development, 0.75–1 ml of autologous blood containing 1000 U of thrombin was injected into the SSS through one catheter. A second sinogram was then obtained after about 45–60 min to document the thrombus development (fig. 3).

This technique of catheter placement at the torcular was tried in three dogs, and SSS thrombin induction in another three. Computed



B

cortical veins should not occur normally (cf. fig. 2). B, 9 sec after injection of contrast material into SSS. Abnormally prolonged opacification of SSS and cortical veins.

tomography (CT) was performed about 4 hr after thrombosis induction to assess its efficacy in diagnosing the thrombosis. After CT, the animal was sacrificed by administering an overdose of intravenous pentobarbital. The SSS and the brain morphology were studied in both the fresh and fixed states.

Results

The technique of catheter placement at the torcular without attempt at SSS thrombus induction was successful in each of the three dogs. In another three dogs, SSS thrombus induction was achieved and confirmed by pathologic examination. The development of thrombosis on the sinogram was seen as filling defects within the distended SSS in concert

Fig. 4.—A, Coronal CT scan of dog brain. Experimentally induced SSS thrombosis (*arrow*) (same dog as in fig. 3). Attenuation of thrombus-containing SSS is 175 H (compared with 1500 H of bone). This excessive density of SSS thrombus probably is partly from partial-volume averaging with bone and partly from iodine entrapment in clot. B, Bone-shift image. Triangular density of thrombus-containing SSS is not visible. (Normally there is no bony crest along inner aspect of dog skull.)

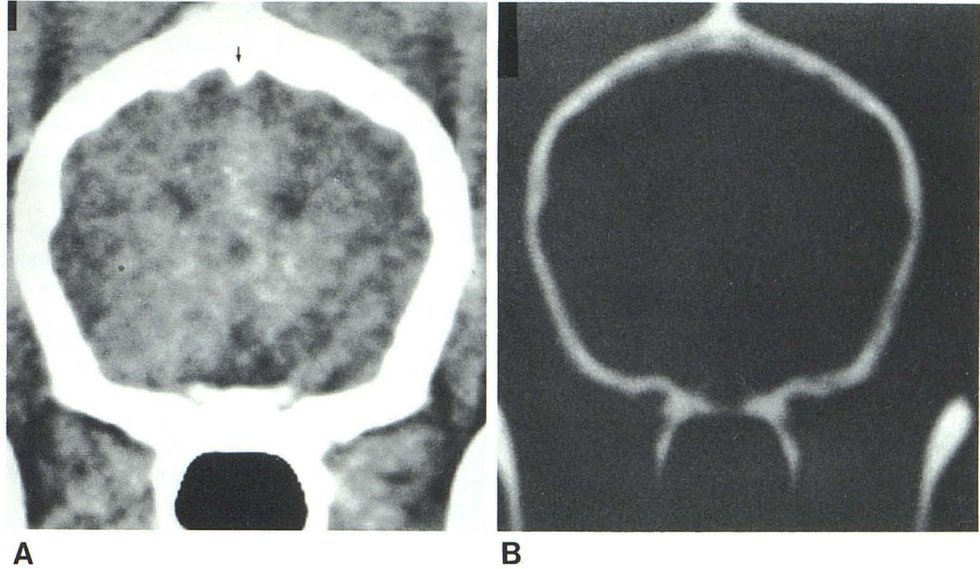
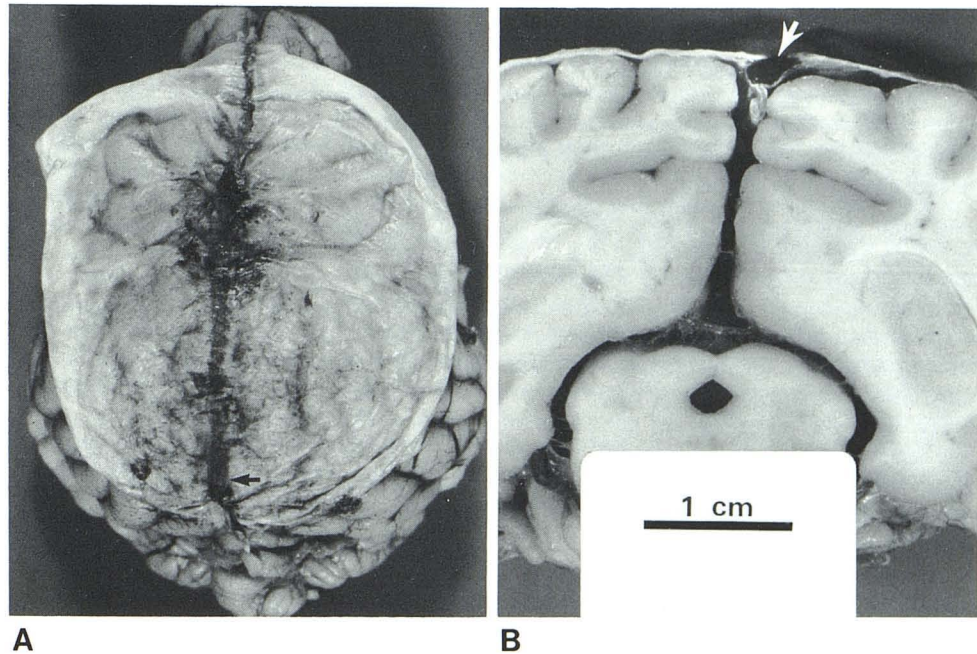


Fig. 5.—Autopsy specimen of dog brain in which SSS thrombosis was produced experimentally (same dog as in figs. 3 and 4). Superior (A) and coronal (B) views. SSS is distended with thrombus (*arrow*). No retrograde extension of thrombus into neighboring cortical veins.



with prominent retrograde filling of the cortical veins and delayed clearance of the contrast medium (fig. 3). On the CT scan, the thrombus was seen as a triangular high density (fig. 4). At autopsy, the thrombosis was manifested as an antemortem clot firmly adherent to the distended SSS (fig. 5). On gross examination of the brain, there was no retrograde extension of the clot into the adjoining cortical veins, nor was there evidence of brain lesion.

Discussion

Very little experimental work has been aimed specifically at producing SSS thrombosis. Prior studies have focused mainly on acutely occluding the posterior part of the SSS and the other dural venous sinuses by methods that consisted pri-

marily of applying ligatures around the venous sinus or introducing a foreign body or a coagulant within the lumen [7-16]. These experiments were designed essentially to produce a state of acute intracranial venous hypertension and to study the acute and chronic effects on the brain. The development of hydrocephalus was of major interest in these studies.

Beck and Russell [17] specifically attempted experimental induction of thrombosis by surgically exposing the SSS in 50 animals (29 rabbits, 13 dogs, four puppies, and four kittens) by (1) causing stasis after applying a clip on the sinus, (2) augmenting the stasis by introducing a coagulant into the sinus lumen, (3) blocking the sinus lumen by muscle or cotton-wool alone or steeped-in coagulant, (4) heat coagulation, and (5) inducing the Schwartzman phenomenon. (The Schwartz-

man phenomenon is an immune reaction that was elicited in two rabbits by exposing the SSS and scarifying its dorsal wall by needle point. A cotton-wool pledget soaked in potent *E. coli* filtrate was applied over the exposed SSS for 10 min and the wound then closed. After 24 hr a precipitating dose of 4 ml of the filtrate was injected into the marginal vein of the ear. The animals were sacrificed 3 days later. There was hemorrhage and necrosis of the soft tissues bordering the operation area. However, there was no SSS thrombosis.) In most (44 of 50), no SSS thrombosis was achieved. In the rest, segmental thrombosis was noted in one dog and granulation tissue in five. Except in cases of surgical trauma (two rabbits and one puppy), the animals remained healthy until sacrificed at various intervals (2 days to 10 weeks). The brain was essentially normal in all, except for slight ventricular dilation in five animals (two dogs and three puppies). Frustrated with their attempts at producing SSS thrombosis in most animals, Beck and Russell stated, "The idea that thrombosis may readily be induced in the superior longitudinal sinus evidently needs revision."

We believe that the experimental method of SSS thrombosis induction that we have designed causes no direct injury to the brain per se. This technique should help establish CT and magnetic resonance imaging criteria for diagnosing SSS thrombosis. Further, the method should help in studying the natural history, as well as the brain lesions and the neurologic deficits produced by SSS thrombosis.

The fact that no macroscopic brain lesions could be observed in the three animals might be explained by the fact that there was no concomitant retrograde extension of the thrombus into the cortical veins and that there was sufficient venous collateralization (which is known to be extensive in the dog) to preclude macroscopically recognizable ischemic brain lesions. Microscopic brain injury cannot be excluded totally, however, since we did not study the brain by this method; we plan to do so in the future. In this regard the observations made by Becker (cited in Friede [5]) are appropriate. He injected paraffin wax, which hardens at body temperature, into the SSS of puppies. He observed cerebral lesions only when the injected material extended into the superficial cortical veins. These animal observations are probably applicable to humans as well.

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