Since our report, the operative technique has been modified slightly. Rather than opening the dura, the outer layer is incised after its separation from the inner layer. This permits satisfactory decompression while minimizing possible complications.

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References

Arteritis due to EACA Therapy

To the Editor: In their article on arteriopathic complications of subarachnoid hemorrhage with EACA (J Neurosurg 40:480–485, 1974), Drs. Sonntag and Stein conclude from the clinical course and angiograms that arteritis occurred, perhaps as a result of the use of EACA. The radiographs are said to demonstrate arteritis, when actually they are quite compatible with the generalized, but discrete, lesions often seen with diffuse spasm. In the first case report it seems impossible to prove that the punctate lesion (a) in Fig. 1 is not also present as part of the earlier filling defect which was called “spasm.” The other radiographs referred to in the article also appear to be as consistent with diffuse spasm as with arteritis. The clinical improvement of the latter two patients may well represent the natural history of their illness or be the result of other therapeutic measures such as shunt placement, rather than the result of discontinuing EACA. Furthermore, without pathological correlation it is difficult to substantiate the presence of an arteritis.

The authors are to be commended for their awareness of potential hazards of EACA therapy. The clinical effectiveness of EACA for subarachnoid hemorrhage secondary to ruptured aneurysm is certainly still controversial. However, it does not seem justifiable to implicate EACA in these cases on the basis of the circumstantial evidence presented in this article.

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Response: Dr. Hood rightfully emphasizes the difficulty in differentiating spasm from the diffuse arteritis seen in our patients treated with EACA. However, we think that these changes do not look like spasm but are more in keeping with the findings seen in proven cases of arteritis and that when such changes are seen, the surgeon should consider EACA as a possible cause.

Spasm usually has its peak between the fifth and twelfth days after a subarachnoid hemorrhage. The punctate narrowing of the internal carotid artery, the occlusion of an ascending frontal branch of the middle cerebral artery, and the alternate narrowing and dilatation of many of the cerebral arteries in our Case 1 were seen in an arteriogram performed 18 days after the subarachnoid hemorrhage and after the patient had been on EACA for 14 days. In Case 3, similar changes were seen 17 days after the subarachnoid hemorrhage, but 7 days after EACA was started.

It is well known that spasm occurs in the vessels adjacent to the ruptured aneurysm and may spread throughout major vessels at the base of the brain. In all three cases we saw changes as described above not only quite distant from the aneurysm but also distant from the circle of Willis.

Two of our patients improved after EACA was stopped and follow-up arteriograms did not reveal any of the previous changes present while on EACA. It is true that the third patient had a shunt placement at the same time that EACA was discontinued; however, arteriographic abnormalities were present 17 days after the second subarachnoid hemorrhage and not present on the next arteriogram, 9 days after the EACA was stopped.

It is our contention that EACA indeed may cause clinical as well as arteriographic changes and that when the condition of patients receiving this drug begins to
deteriorate, follow-up arteriograms are warranted. If the changes described in our paper are present, EACA should be discontinued.

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Carotid Ligation for Aneurysms

To The Editor: This note is prompted by the article by Kak, et al., in the October, 1973, issue of the Journal (Kak VK, Taylor AR, Gordon DS: Proximal carotid ligation for internal carotid aneurysms. A long-term follow-up study. J Neurosurg 39:503–513, 1973). This paper again demonstrates that proximal occlusion of the cervical carotid artery is an effective method for preventing rebleeding from intracranial carotid aneurysms; it also showed that common carotid occlusion was just as effective as internal carotid artery occlusion and carried with it considerably less morbidity. Although blood flow and pressure measurements were recorded, no indication was given about the systemic arterial blood pressure at the time of ligation; therefore the relationship of pressure to ischemic changes is not clear. We are writing primarily to comment on this point.

On our service for over 20 years various types of proximal ligation of arteries feeding saccular aneurysms have also been carried out; we, too, find that these procedures are significant protection against recurrent bleeding. To diminish the risk of ischemia, we have carried out staged ligation of the common carotid artery under local anesthesia and made special note of the pressor effect on systemic arterial blood pressure. It is this latter feature, the presence or absence of pressor effect, that we feel constitutes an essential difference between internal and common carotid artery occlusion and so influences the potential for subsequent ischemia.

In common carotid ligation under local anesthesia, if there is an immediate pressor effect on systemic arterial blood pressure, we proceed with ligation with the feeling that this decreases the risk of subsequent cerebral ischemia. The pressor effect may last for only 24 to 72 hours, but this, as Kak, et al., indicate, is the time of most post-ligation ischemic episodes. The cause of the pressor effect is the decreased pressure sensed by the baroreceptors in the region of the carotid sinus. With internal carotid artery ligation, the opposite occurs. The pressure sensed at the sinus is increased; this tends to abolish or inhibit any compensatory systemic pressor effect. Although there are other reasons for post-ligation ischemic events, this significant difference between common and internal carotid artery ligation in relation to potential ischemic episodes deserves emphasis. It is for this reason that it would be desirable to know the relationship of the arterial blood pressure to the incidence of ischemic events in the different categories reported by Kak, et al. Moreover, the manner of carrying out the ligation is significant; the use of local or general anesthesia, the presence or absence of certain drugs, etc., are all factors that may modify the response of the carotid sinus. In this regard it is significant that Purves 1 has demonstrated the important direct effect of the carotid sinus on cerebral blood flow through the neurogenic mechanism.

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Reference


Response: Early in our series most of the operations for carotid ligation were done under local anesthesia, but from 1960 onward most were done under general anesthesia. The systemic blood pressure was measured at the same time as the intraarterial pressure recordings taken in the carotid artery distal to the point of occlusion. The systemic pressure rarely varied more than a few mm Hg when either the common or internal carotid artery were occluded at operation. Carotid artery pressures before and after occlusion could not be compared unless the systemic blood pressure had remained constant. Most of the complications occurred within 48 hours; during this time the pressor effects or their absence would be most noticeable. No significant pressor effect was noted in this period. We never carried out a staged ligation based on systemic arterial pressure changes. The ligation was staged either because the patient in some way could not tolerate a total occlusion...