CLINICAL COMPLICATIONS OF CEREBRAL ANGIOGRAPHY

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(Received for publication November 26, 1951)

The purpose of this paper is to summarize the untoward effects of cerebral angiography as we have encountered them clinically in 150 patients upon whom we have performed 174 cerebral angiograms. We wish further to emphasize the several factors or mechanisms that may be etiologic in the development of these complications. This report is not intended in any way to condemn the use of cerebral angiography for we feel that it is one of the greatest advances in our means for the diagnosis of intracranial lesions since the introduction of pneumoencephalography and ventriculography by Dandy.

With the present extensive use of cerebral angiography as an aid in the diagnosis of many different intracranial neurologic problems, there is a need for an adequate evaluation of the coincidental reactions and complications of this very valuable diagnostic procedure. Although there are many reports in the literature concerning various complications that have arisen consequent to cerebral angiography, it would appear that there is a need again to emphasize that cerebral angiography may not be as innocuous a procedure as some have indicated. Our experience has shown, and we wish to herein emphasize, there can be no doubt that not a few of these complications are a matter of ill-advised or poorly executed techniques employed, while other untoward manifestations may be assigned to the contrast media, and finally a few of these must always be inherent dangers in doing cerebral angiography.

In our series of 150 cases in which angiography was performed there were 32 tumors, 25 aneurysms, 13 subarachnoid hemorrhages (no aneurysm found), 3 subdural hematomas, 4 intracerebral hematomas, 14 cerebral thromboses, 3 with thrombosis of the internal carotid artery,† 17 cerebral degenerative syndromes (arteriosclerosis, atrophy, etc.), 1 meningovascular syphilis, and 38 miscellaneous (8 normal vascular patterns and a heterogeneous group of cases).

In this group serious untoward complications were encountered in 17 cases with 5 fatalities. In 2 instances less serious though alarming signs were noted.† The following case reports detail these major complications. Other less serious and transitory complications will be discussed.

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† One with thrombosis of one internal carotid artery and thrombosis of the contralateral middle cerebral artery.
† Since this was submitted for publication we have performed over 100 more percutaneous cerebral angiograms, observing the precautions herein outlined, and in only 1 instance were there even transitory neurologic complications.
COMPlications of cerebral angiography

REPORT OF CASES

I. FATAL CASES

(A) Delayed onset of unconsciousness, respiratory failure and subsequent death occurring with a normal cerebrovascular system.

Case 1. An angiogram was done because of fixed pupils and bilateral facial and orbital pain of undetermined etiology with negative pneumoencephalogram in a 38-year-old white female. After failure in finding the right common carotid artery by the percutaneous technique, the open method was used. In this case only the right carotid artery was exposed. A total of 40 cc. of 35 per cent diodrast was employed. Because of restlessness and difficulties in the x-ray technique 5 cc. of 2½ per cent pentothal were given intravenously and two 5 cc. injections of 70 per cent diodrast were made into the right carotid artery. Excellent roentgenograms were obtained in the AP and lateral views. The patient awakened immediately after this complaining of a severe pain in her eyes, an accentuation of one of her chief complaints. There were no other untoward complaints or neurologic sequellae, until an hour and 20 minutes after the last angiogram was taken when central respiratory failure suddenly developed, and 2 hours later complete respiratory failure ensued. In spite of the aid of a respirator, stellate ganglion procaine blocks, serum albumin, plasma, lumbar punctures, metrazol and caffeine, death occurred. Postmortem examination confirmed the clinical evidence of cerebral edema with additional pathologic findings: 1) Bilateral gasserian ganglionitis, chronic, severe; 2) marked cerebral edema with pressure cone formation at the foramen magnum; 3) persistent thymus and relatively large lymph nodes; 4) hypoplasia of the breasts and mild hirsutism; 5) probable mild platybasia. (A review of the roentgenograms revealed a grade I platybasia.)

Delayed (30 minutes) loss of consciousness, sudden respiratory failure and death occurring in 2 cases of atherosclerosis and thrombosis of the internal carotid with cerebral infarction.

Case 2. A 53-year-old white male was studied because of progressive left hemiparesis. Difficulties were encountered in doing carotid angiography by the percutaneous method and therefore the open method was used. It was recognized that the right carotid appeared firmer than usual but the needle was inserted into it anyway. Blood flowed only fairly well and although it was suspected that it might be thrombosed, 10 cc. of 35 per cent diodrast were injected into the carotid. The angio- grams disclosed a "blotch" of dye, measuring 5×2½ cm. in its greatest diameter, in the region of the distribution of the middle cerebral artery. This was thought to be a diffusion of the diodrast into the infarcted area. There was only a thin streak of dye seen in the thrombosed internal carotid artery. Central respiratory failure occurred about 30 minutes after the last diodrast injection followed in a few minutes by cardiac arrest. At subsequent examination there was found an infarction of the major portion of the right hemisphere with obvious thrombosis of the right common and internal carotid arteries and with healed ancient coronary thrombosis and myocardial fibrosis.

Case 3. A 38-year-old white male was hospitalized because of right hemiplegia and total aphasia. This came on approximately a week after severe hemorrhage from a duodenal ulcer. Bilateral cerebral angiography was done under pentothal anesthesia, using 60 cc. (!) of 35 per cent diodrast on each side. He failed to regain consciousness and died 12 hours later. Autopsy disclosed an atherosclerotic plaque in