Hazard of Retrograde Aortography in Dissecting Aneurysm

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AORTOGRAPHY by translumbar, retrograde femoral injection, and retrograde catheterization, are all now considered moderately safe and efficacious diagnostic procedures. It is our objective to describe a potential hazard in retrograde arterial catheterization for the diagnosis of dissecting aneurysm, that to our knowledge, has not previously been described. Two cases are presented in which the retrograde catheter entered the false lumen of a dissecting aneurysm.

Case Reports

Case 1
The patient, a 64-year-old white man, was admitted to Wadsworth General Hospital on July 10, 1961, with a chief complaint of unrelenting chest pain of several hours' duration. There was a 2-year history of hypertension. No prior history of heart disease was elicited.

The blood pressure was 220/110 in both arms, and there was a pulse rate of 110 per minute. The entire examination was within normal limits, including the heart, lungs, abdomen, peripheral vessels, and nervous system.

The hemoglobin value was 39 per cent and the white blood-cell count 9,500, with 85 per cent polymorphonuclear leukocytes. On the fifth hospital day it was 15,500 cells with 88 per cent polymorphonuclear leukocytes. Urinalysis revealed 2+ albumin, 1+ glucose, and 10 to 15 red blood cells per high-power field were found. The creatinine was 1.5 mg. per cent and the amylase 82 Somogyi units (upper normal 120 units). Serum glutamic oxaloacetic transaminase on five successive days was less than 40 units. The fasting blood sugar was 154 mg. per cent (upper normal 90 mg. per cent). Serial electrocardiograms were interpreted as showing left ventricular hypertrophy with occasional ventricular premature contractions. Chest x-rays showed a dilated aorta.

In spite of large doses of meperidine the patient complained of persistent back pain. On the eighth hospital day an aortogram was done by retrograde catheterization of the femoral artery. The films revealed the catheter to be within the false lumen of a dissection that extended from the distal arch of the aorta to the diaphragm (Fig. 1). On the ninth hospital day the patient was subjected to thoracotomy with retrograde perfusion of the distal aorta by partial bypass through a pump oxygenator. The aorta from the distal arch to the diaphragm was replaced with a Teflon sleeve prosthesis. Postoperatively the patient did poorly and died within 24 hours.

Case 2
The patient, a 61-year-old Filipino was admitted to Wadsworth General Hospital on December 13,

Figure 1
Retrograde femoral angiogram of case 1 showing catheter and contrast material in false passage of the dissecting aneurysm. The true aortic lumen has been outlined. The configuration of contrast material remained unchanged over several seconds.

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The patient had recovered uneventfully from a myocardial infarction in 1947. In 1954 the patient suddenly developed vertigo, nausea, and vomiting, and a diagnosis of Meniere's disease was made. Subsequently he experienced recurrent bouts of lightheadedness. In 1955 he developed a left hemiplegia with a residual left hemiparesis. Caloric testing revealed a nonfunctioning labyrinth on the right.

On admission the patient denied any impairment of speech or vision. There had been no increase in motor impairment nor had there been further sensory difficulties.

The blood pressure was 160/104 mm Hg bilaterally. The pulse was 80 per minute and regular. Bilateral areus senilis was present. No carotid or cranial bruits were heard. The heart, lungs, and abdomen were within normal limits. A left hemiparesis was noted.

The blood count, urinalysis, and fasting blood sugar were within normal limits. The serologic test for syphilis was negative. Serum cholesterol was 384 mg. per cent. The electrocardiogram was consistent with an old diaphragmatic myocardial infarction and left ventricular hypertrophy. The electroencephalogram was abnormal with waves of 4 to 6 cycles per second over the right hemisphere. The skull films were within normal limits. Chest x-ray showed aneurysmal dilatation of the descending aorta.

Retrograde aortography was performed via a catheter inserted into the right brachial artery and threaded into the aortic arch. The subsequent films showed that the catheter was located within the false passage of the aneurysm and that the dye in the false passage was obliterating the carotid arterial lumens (fig. 2). Immediately after the procedure the patient developed convulsions, followed by disorientation. Cerebral function gradually returned. Five days after aortography the patient developed severe retrosternal pain with blood pressure elevation to 220/135 mm. Hg. There was no change in the electrocardiogram, and serial transaminase determinations were normal. It was thought that there had been further aortic dissection. The patient gradually improved and was transferred to a long-term care unit.

Discussion

Aortography was introduced in 1929 by dos Santos. He first used a translumbar approach and later employed retrograde injection or femoral catheterization.

Unfortunately complications occur frequently enough to make the physician weigh the merits to be derived against the potential risks of the procedure. Abrams listed 29 deaths in 1,076 thoracic aortograms (1.7 per
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These were due most commonly to cerebral damage from the iodinated contrast agents. Respiratory, cardiac, and renal deaths were also noted. One patient died of hemorrhage. Morbidity in this procedure involves hematoma formation, thrombosis, iodism, and transient depression of renal function. A recent report by Gudbjerg and Christensen presents data indicating intimal damage, including subintimal deposition of dye with dissection, in 36 of 126 cases in which retrograde lumbar aortography was performed on arteriosclerotic vessels. No persistent damage was recognized in these patients.

Within a 6-month period we encountered two cases in which the false passage of a dissecting aneurysm was inadvertently cannulated during retrograde aortography. While the sudden release of 40 to 60 ml. of contrast material into the false passage by the Gidlund pressure syringe may extend the dissection, rupture the false passage, or occlude the main channel by the wall of the false injection into the false channel, the procedure was uneventful in case 1. However, the second patient suffered cerebral ischemia resulting from bilateral carotid occlusion by the distended false passage.

The false passage can be entered via catheter insertion from the brachial or femoral arteries. This would seem to preclude either route as the "safe" approach.

Since the diagnosis of dissecting aneurysm often depends on aortography, the injection of contrast material into the left atrium by transseptal catheterization might eliminate the danger of direct injection into the false passage.

**Summary**

During retrograde catheterization via the femoral artery and the brachial artery, the false lumen of a dissecting aneurysm was inadvertently entered in two patients. Bilateral carotid occlusion was produced in one by the high-pressure injection of contrast material into the false passage. A method is suggested to avoid this hazard.

**Acknowledgment**

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**References**


**Surgery and New Knowledge**

Whenever a region of the body has been made accessible to surgery, a new insight into its pathology has resulted from the fresh experience that has been acquired of morbid processes in the living and especially of the earlier stages of disease. This has been abundantly shown in the case of the abdomen and in the case of the thorax.—*The Collected Papers of Wilfred Trotter, F.R.S.* London, Oxford University Press, 1946, p. 48.
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