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Amnesia after Femorocerebral Angiography

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R. K. Coates¹

The charts and radiographs of 12 of 1,520 patients who developed amnesia after transfemoral cerebral angiography performed with local anesthesia and minimal sedation were retrospectively reviewed. In three younger patients, exacerbation of organic or idiopathic temporal lobe epilepsy was thought to be the cause of the amnesia, while the amnesia of the nine patients over 40 years old had the characteristics of transient global amnesia. Eight of the patients had had similar episodes before angiography, and three others had other evidence of vertebrobasilar disease. Emboli from atheromatous debris or catheter clot due either to catheter manipulation problems, an inexperienced operator, or the use of a large catheter were considered likely causes in 11 cases, while in three patients contrast emboli or toxicity may have been the etiologic factor.

Amnesia after angiography is seldom mentioned in reviews of the complications of cerebral angiography performed by transfemoral catheterization [1]. Wishart [2] briefly mentioned six cases in 231 vertebral angiograms. Wales and Nov [3] described two cases of transient global amnesia complicating cerebral angiography. Memory disturbance was described in nine patients after vertebral angiography by de Tribolet et al. [4], while two cases have been recorded during coronary angiography with the catheter positioned in the aorta [5]. Our experience is that amnesia is more common than the literature suggests; this paper presents 12 cases seen in our department over the past 4 years.

Materials and Methods

Between April 1977 and April 1981, 1,520 patients had cerebral angiography performed by transfemoral catheterization under local anesthesia with premedication using either 10 mg Valium or 30 mg Serax given orally about 1 hr before the angiogram. The posterior circulation was studied in 1,321 of the patients. Forty percent of the patients were examined for suspected carotid stenosis, 11% for possible vertebrobasilar ischemia, 30% for possible intracranial bleeding, 10% for suspected brain tumors, and 9% for miscellaneous reasons.

One neuroradiologist used a red Kifa 6 French catheter for 750 angiograms. The other neuroradiologist obtained 313 angiograms with a 7.3 French Cook Head Hunter catheter on cases presenting with transient ischemic attacks and vertebral basilar disease, and used a Mani 5 French catheter for the other 457 angiograms. Forty-eight angiograms were obtained by a resident under supervision, and the two experienced neuroradiologists obtained the other 1,472.

Conray-260 was used in all cases; the catheters were frequently flushed with heparinized saline (4,000 U/L). Subclavian or innominate injections were made in 519 patients; selective vertebral injections were made in 802. For each subclavian or innominate artery series, 10 ml of contrast material was used per injection, while 2½–6 ml of contrast material was injected each time a vertebral artery was selectively studied. The total amount of contrast material injected into the vertebrobasilar circulation by the neuroradiologist using the Mani and Cook catheters was 20 ml, and the other neuroradiologist never injected more than 35 ml. If both vertebral arteries needed to be catheterized, both investigators...
studied one before and the other after the anterior circulation had been examined so that there was a ½–1 hr interval between the two series of vertebrobasilar injections. The smallest artery catheterized in each examination of the posterior circulation is recorded in Table 1.

Results

The central nervous system (CNS) complications are listed in Table 2 and are divided into four types as described by Mani et al. [1]. A transient minor complication lasted less than 10 days and did not significantly affect the health of the patient, while a transient major complication was of the same duration, but the activity and health of the patient were affected. A permanent complication persisted for more than 10 days. Technical problems during angiography were recorded on our radiologic reports. The data concerning amnesia and other complications were recorded at the time of the angiogram and from information supplied during our daily neuroscience case conferences. Details of the course of amnesia were derived retrospectively from the patients’ charts.

Twelve patients developed sudden amnesia for recent events and could not remember where they were, why they were there, or what had happened to them (Table 3). Frequently, they asked a question that they had asked just a few minutes before and to which they had been given an answer. Some felt vaguely that something was wrong and others felt shivery.

In three younger patients (cases 1–3), the amnesic episodes were thought to be due to a temporal lobe epilepsy because the patients were more agitated, restless, or belligerent, and because they had previously had a similar attack labeled as temporal lobe epilepsy. These patients, two men and one woman aged 22–36 years, had no evidence of cerebrovascular disease. The temporal lobe epilepsy was idiopathic in one patient, the result of a viral encephalitis in another, and produced by a thalamic glioma in the third. The amnesia started during or at the end of the angiograms and lasted 3, 6, and 24 hr, respectively. The posterior circulation was studied angiographically in two patients, but in the patient with the thalamic glioma only a right carotid angiogram was obtained. In this patient the right carotid artery supplied the right posterior cerebral artery.

In nine older patients, six men and three women 43–72 years old (mean, 58 years), transient global amnesia developed after angiography. This was recognized during or at the end of angiography in eight patients, while in one patient (case 12), the amnesia was recognized only 8 hr after

TABLE 3: Etiologic Factors in Amnesia after Femorocerebral Angiography

<table>
<thead>
<tr>
<th>Case No.</th>
<th>1</th>
<th>2</th>
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<th>4</th>
<th>5</th>
<th>6</th>
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<th>10</th>
<th>11</th>
<th>12</th>
<th>Totals</th>
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<tbody>
<tr>
<td>Temporal lobe epilepsy</td>
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<td>X</td>
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<tr>
<td>Transient global amnesia</td>
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<td>X</td>
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<td>9</td>
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<tr>
<td>Clinical (C) or radiologic (R) evidence of disease in the territory of the posterior cerebral basilar and vertebral arteries.</td>
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<td>C</td>
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<tr>
<td>Previous amnesic episode</td>
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<td>Other evidence of vertebrobasilar disease</td>
<td>R</td>
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<td>Possible technical problems:</td>
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<td>Catheter manipulation</td>
<td>X</td>
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<td>Inexperienced resident</td>
<td>X</td>
<td>X</td>
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<td>Big catheter—7.3 French in subclavian (S) or 6 French in vertebral (V)</td>
<td>V</td>
<td>S*</td>
<td>V</td>
<td>V</td>
<td>S</td>
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<td>Possible contrast problems:</td>
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<td>“Bad batch”</td>
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<td>“Overdose”</td>
<td>X</td>
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* Right carotid angiography only; this supplied the right posterior cerebral artery.
† 5 French Mani used.
angiography. The nine patients had both their anterior and posterior circulations studied. Cases 4–7 and 10–12 had clinical and/or radiologic evidence of cerebrovascular disease. Five of the nine cases (cases 4, 6–8, and 12) also had had a previous episode of transient global amnesia. The precise duration of that amnesia was difficult to evaluate, but five patients had recovered within 4 hr and all had recovered by 24 hr.

Seven patients (cases 1, 2, 6, 7, and 9–11), questioned at various times during their hospital stay, could not recall the angiogram. Four patients (cases 3, 5, 8, and 12) were not asked if they could remember the angiogram. Case 4 was amnesic for 3 hr after angiography, but later claimed to remember the angiogram. One of the older patients, case 7, developed transient signs indicative of vertebrobasilar ischemia during his amnesia. No new signs occurred in any other patient. Computed tomography (CT) was performed on all patients before angiography and was normal in all except case 2, the patient with the thalamic glioma.

Clinical vertebrobasilar disease and a history of transient global amnesia was prominent in those patients who developed transient global amnesia after angiography. Seven (4%) of 167 patients presenting with vertebrobasilar disease developed postangiographic transient global amnesia. Excluding all patients who may conceivably have had temporal lobe epilepsy, only one (0.1%) in 1,200 of those patients without vertebrobasilar disease developed postangiographic transient global amnesia. A patient who developed postangiographic transient global amnesia was at least 10 (and not more than 30) times more likely to have a history of transient global amnesia than one who did not develop this complication.

In case 4, a patient with spasm of the basilar artery in whom it was important clinically to prove that a basilar tip aneurysm was present, four injections of 6 ml of contrast material were made into the left vertebral artery, and it was estimated in this case that 30 ml of the total 50 ml of contrast agent used for the investigation entered the posterior circulation.

Technical problems with catheter manipulation were recorded in four patients (cases 5, 7, 10, and 11). All the amnesic complications occurred in the hands of experienced neuroradiologists with the exception of two of the younger patients (cases 1 and 3), who were catheterized by a resident under supervision.

In five patients the amnesic episodes followed subclavian injections using a 7.3 French Head Hunter catheter. There were no episodes of amnesia when subclavian angiography was carried out using 6 French Kifa or 5 French Mani catheters. In five patients the amnesic episodes followed selective vertebral injections using a 6 French Kifa catheter. One patient developed amnesia after a selective vertebral injection with a 5 French Mani catheter. In the patient in whom the posterior cerebral artery originated from the carotid artery, the amnesic episode followed carotid angiography using a 7.3 French Head Hunter catheter. When the relation of catheter size and the smallest artery injected in age-matched groups is studied (table 1), a soft size 6 catheter is safer than a stiff size 7.3 catheter in a subclavian artery and a size 5 catheter is safer than a size 6 catheter in a vertebral artery.

Cases 6 and 9 developed amnesia with the same batch of contrast material (table 3), and at that time six of nine consecutive patients developed complications. Five of these had neurologic complications, and one had an allergic complication. Three of the patients had the presenting neurologic symptoms reproduced. The probability that two of five consecutive patients would develop amnesia after angiography when the approximate incidence of amnesia is 1% (12 in 1,520) is $9.7 \times 10^{-4}$, and the probability of five of nine patients developing neurologic complications when our neurologic complication rate is 2.2% (table 2) is $6 \times 10^{-7}$ using binomial probability.

Three patients had a history of temporal lobe epilepsy and five patients had a history of transient global amnesia. Seven patients had either clinical or radiologic evidence of vertebrobasilar disease (table 3). Overall, therefore, 11 of 12 patients had evidence of disease in the territories of the posterior cerebral arteries; case 9 was the only exception. These 11 patients were regarded as having "susceptible brains." Situations likely to produce atheromatous debris or catheter clot emboli occurred in 11 cases (table 3). In three patients contrast emboli or toxicity may have contributed to the development of the amnesia, and in case 9 it was most strongly suspected. This 60-year-old man presenting with amaurosis fugax had no radiologic or clinical evidence of vertebrobasilar disease, and his technically uneventful angiogram was obtained by an experienced operator using a 6 French Kifa catheter in the vertebral artery. This patient was one of the six of nine consecutive patients who developed complications during angiography using the same batch of contrast material.

Discussion

Transient global amnesia is a syndrome described by Fisher and Adams [6] in 1964 in which there is a sudden loss of memory for the present and recent past in individuals in or past middle age, and the patients cannot retain new facts although the remote memory is intact. Characteristically, the patient keeps asking the same question. There is no loss of consciousness, no physical signs, and the patient recovers in several hours but has amnesia for the period of the attack. Single occurrences are the rule but multiple attacks have been reported [7–9]. The authors speculated that the cause of the transient global amnesia might be a localized epileptic discharge in the temporal lobe, a theory subsequently favored by few [10, 11], or an episode of ischemia involving the limbic system of the medial temporal lobe. The generally accepted theory is that it is due to transient vascular insufficiency of the arteries supplying the medial temporal lobe [12]. Our series has two distinct groups of patients—a younger group of three labeled as temporal lobe epilepsy and an older group of nine classified as transient global amnesia.

Of our 1,520 patients, 12 (0.8% or one in 125) developed postangiographic amnesia, yet this complication is rarely recorded by others [1–5]. The complication rate for patients

**Angiographic Amnesia**

*Case 9*
with cerebrovascular disease and subarachnoid hemorrhage in the 2,316 cases of Mani and Eisenberg [13] was 1.7%. They did not say whether amnesia occurred. Eisenberg et al. [14] reported a complication rate of 1.3% in 301 cases of cerebrovascular disease, but they did not attempt either vertebral or subclavian catheterization. They reported no amnesic complications. Although our CNS complication rate was 2.2%, we had a high number of minor transient CNS complications (0.9%) compared with Mani et al. [1], possibly due to our light sedation. Because our overall complication rates are similar to those of other investigators, we speculate that others may not have recognized amnesia due to heavier sedation. We would have been unaware of these complications if the patients had been premedicated with neuroleptanalgesia [15] or Demerol or had the investigation been performed under general anesthesia. The type of sedation used by Mani et al. [1] and by Eisenberg et al. [14] is not recorded. Palmer et al. [16], who injected the innominate and subclavian arteries, may not have noticed amnesic complications because of the neuroleptanalgesia. Silent radiologic emboli from cerebrofemoral angiography were described by Cronqvist [17].

Our statistics for vertebral and subclavian angiography suggest that a smaller, softer catheter will produce the fewest complications. Eisenberg et al. [14] stated that a 5 French catheter has a surface area only half that of a 7 French catheter, which would correspondingly decrease the potential for emboli, and that the stiffer catheters, whose torque facilitates catheterization, may also damage the intima by flailing of the catheter tip. Wishart [2], who recorded six cases of amnesia in 447 cases, used an 8 French Hinck Head Hunter catheter [18]. De Tribolet et al. [4], who described Korsakoff syndrome in nine of 832 vertebral angiograms, used a 6.5 French BD red catheter, and we used a 7.3 French Head Hunter in six of our cases. Lin [19] recommended only using bigger Head Hunters for difficult cases, preferring, as do Mani et al. [1] and Eisenberg et al. [14], the soft 5 French catheter.

Wishart [2] also stated that most of his cases were done by residents, and Mani et al. [1] found in 5,000 cases that residents produced 4.5 times as many complications as more experienced operators. Although Olivecrona [20], who reviewed a similar number of cases, disagrees, comparisons suggest that residents in California and Stockholm have similar complication rates. Two of our cases occurred in the hands of a resident.

In Europe, five common brands of contrast medium have been found to contain intrinsic particles [21]. In rubber-tipped vials there are between 61 and 369 particles measuring 5–10 μm, 45 to 183 measuring 10–30 μm, and three to five measuring greater than 30 μm in various brands in 1 ml of contrast material. These small particles could embolize into the territories of both posterior cerebral arteries, the regions thought by Horel [22] to be responsible for amnesia, and could account for our cases of amnesia in which vertebrobasilar disease or temporal lobe epilepsy was present in 11 of our 12 patients. Two cases described by Wales and Nov [3] occurred while using the same batch of contrast medium (Conray-60), and it was theorized that the complications were due to contrast emboli. We had two similar cases associated with one batch of contrast medium.

One of our cases of amnesia occurred when about 30 ml of contrast material was injected into a vertebral artery to diagnose a basilar aneurysm in the presence of some spasm. This amnesia may be due to contrast toxicity in a brain made susceptible by atherosclerosis and arterial spasm. Toxic effects of contrast medium can occur with very high doses of modern agents [23].

In 14 patients with transient global amnesia reported by Matthew and Meyer [7], 11 had clinical and 12 had radiologic evidence of vertebrobasilar vascular disease; and in our nine cases of transient global amnesia, seven had clinical and three had radiologic evidence of vertebrobasilar disease. Five of our nine cases of transient global amnesia had a previous amnesic episode, and all of our cases diagnosed as temporal lobe epilepsy had had a similar episode. All these factors suggest that there are areas in the hippocampi made susceptible by disease processes that can be triggered either by an operator or contrast-induced factor. Overdoses with diazepam may produce transient global amnesia [24], but in our cases 10 mg oral Valium or 30 mg Serax was chosen to produce mild tranquilization with minimal drowsiness.

REFERENCES

2. Wishart DL. Complications of vertebral angiography as compared to non-vertebral cerebral angiography in 447 studies. AJR 1971;113:527–537
14. Eisenberg RL, Bank WO, Hedgcock MW. Neurologic compli-