Occlusive Disease of the Carotid Arteries

By Allen Silverstein, M.D.

Within the last 10 years there has been increasing recognition of the frequency of spontaneous occlusions of the carotid arteries as a cause for neurologic deficit, and several reports of successful therapy instituted early in the disease have appeared. Since many patients with symptoms due to carotid occlusion will be seen initially by the general practitioner or internist, increased awareness and knowledge of the condition seem warranted. The clinical and laboratory features of 50 patients with proved carotid occlusions are presented, the varying clinical pictures produced by such occlusions are described, and the potential means of therapy are discussed.

The recent application of arteriography to the study of cerebrovascular disease has resulted in a revision of several previously held concepts concerning the diagnosis and therapy of the so-called "strokes." One of the most significant of these new concepts has been the acceptance among neurologists of the relative frequency of occlusions of the internal and common carotid arteries as a cause for neurologic deficit. Similarly, the more commonly discussed subjects in the current neurologic literature include many suggested diagnostic and therapeutic means of managing the condition. The purposes of this communication are to analyze the salient features of 50 patients admitted to the Mount Sinai Hospital in whom the diagnosis of carotid occlusion was proved, to restress the frequency and great clinical variability of the disease, and to discuss some therapeutic implications for this common cause of a "stroke."

Materials and Methods

The presence of a carotid occlusion was established beyond doubt in all 50 of the patients who form the basis for this report. The occlusion was demonstrated by carotid arteriography in 47 patients and by autopsy in 3. Additional confirmation of the angiographically demonstrable occlusion was available at surgery in 6 patients and at autopsy in 1.

Forty-five of the 47 arteriograms were performed percutaneously, and 2 by cut-down technic. The contrast medium employed routinely since 1956 has been sodium diatrizoate (Hypaque), and all studies have been performed with the aid of serial radiography. Since 1957 the major portion of the carotid arteries in the neck have been included on the x-ray films of the cerebral angiogram. The criteria for angiographic proof of a carotid occlusion have included visualization of the tip of the Courmand needle on the film.

Thirty of the patients in the present series have been personally observed by me during the last 2 years, and form the basis for a report concerning collateral circulation. The details of all 50 patients have been obtained from the charts of the Mount Sinai Hospital. Where a discrepancy in neurologic findings was recorded during the same period of observation, the findings of the senior observer have generally been accepted. Three of the 50 patients have previously been reported.

Results

Age, Sex, and Incidence. The patients varied in age from 26 to 82 years; 42 were over the age of 50. Thirty-seven of the patients were males, while only 13 were females.

The frequency of recognition of carotid occlusion increased markedly after 1956, almost certainly as a reflection of the increased utilization of arteriography (fig. 1). Two other minor factors may contribute to the more frequent discovery of carotid occlusion as a cause of neurologic deficit: the increased awareness of the condition by pathologists, and thus the more frequent examination of the proximal portions of the internal carotid artery at autopsy; and routine visualization of the neck at the time of cerebral arteriography, and the consequent detection of many partial carotid occlusions.

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*The cassette changer and special table utilized for routine angiography have been designed by Dr. Leonard Mais.
Contributing Factors. There was clear evidence of vascular disease elsewhere in 38 of the 50 patients with verified carotid occlusions. Twenty-six patients gave a history of hypertension, or were hypertensive with a resting blood pressure greater than 150/100. A history of angina or electrocardiographic changes indicating myocardial damage were obtained in 24 patients. Sixteen patients gave a history of intermittent claudication in the lower extremities or had diminished to absent peripheral pulses; 3 of them had previous femoral artery occlusions. Severe arteriosclerotic changes were observed in the fundi of 8 patients, and 9 demonstrated significant calcification of the aorta or carotid arteries on routine chest or skull films. Five patients were diabetic, 3 had positive blood serologic tests (probably true positives in all), and the diagnosis of disseminated lupus erythematosus was established in another.

In addition to generalized vascular disease, the history of a few patients suggested other predisposing factors. Thus, one patient had sustained severe head trauma shortly before her symptoms began; a second showed neurologic symptoms 4 days after undergoing general anesthesia for an appendectomy; and a third had undergone neck surgery several years previously. Still another patient—the only one in the present series whose occlusion was thought to be embolic in nature—suffered from chronic empyema and was being treated for septicemia at the time of his occlusion.

Symptoms. The onset of symptoms was quite sudden in 36 patients, 10 of whom rapidly improved, only to develop recurrent episodes prior to admission. In 9 patients the sudden onset was followed by a progressive course.

Fourteen patients had a gradual onset and slow progression of symptoms, as is usually seen with neoplasms. In 3 of these, despite the over-all progression, the symptoms were somewhat intermittent.

The duration of symptoms prior to admission varied from under 1 day in 4 patients to over 2 years in 3. The symptoms included weakness of 1 side of the body or of an upper extremity in 40 patients; unilateral sensory disturbances such as numbness or paresthesias in 18; difficulty with communication in 19; and organic personality changes in 14. In addition, in 14 patients there were seizures,
which were almost always unilateral, involving the paretic arm or face. Ten patients complained of headache, and 4 patients reported dizziness or a depressive reaction. Visual symptoms were described in 9 patients; in 5 of these there was transient diminution or loss of vision in the eye ipsilateral to the carotid occlusion.

**Signs.** Motor weakness was by far the most common neurologic finding in this series: hemiparesis or hemiplegia was present in 49 of the 50 patients. The deficit was always greater in the upper extremity than the lower, and the face was commonly involved. Reflex abnormalities, such as unilateral hyperactive deep tendon reflexes or extensor plantar responses, were noted in 35 patients; in a few patients the plantar responses were bilaterally extensor. A hemisensory syndrome was detected in 32 patients, and a visual field defect was established in 26. An organic mental syndrome of varying severity was evident in 22 patients, and 21 exhibited some degree of aphasia. Pupillary changes were recorded in 3 patients; in 2 of these the pupil contralateral to the occluded carotid was dilated, while the ipsilateral pupil was dilated in the third. Ataxia, gaze palsy, and extrapyramidal signs were each described in 1 patient. Papilledema was not noted. Optic atrophy on the same side of the occlusion was noted in only 1 patient, and occurred several weeks after his hospitalization.

A statement concerning the equality of the 2 carotid pulses was available in 36 patients; in 11, at least one observer noted unilaterally diminished pulsations; in most cases, however, subsequent examiners could not confirm this finding. An attempt was made to hear a bruit over the carotid arteries, eyeballs, and head in 30 patients, and was successful in only 4. The limited value of auscultation for a bruit or palpation of the carotid pulse has been emphasized previously. Manual compression of the patent carotid artery was recorded in 34 patients, and syncope or seizures were produced by this maneuver in 22 patients. Of the 20 patients in whom ophthalmodynamome-try was performed, 16 had significantly diminished retinal artery pressures on the side of their occlusions. The usefulness of these procedures has also been discussed. Distention of the superficial vessels of the face on the side of a carotid occlusion—presumably the effect of collateral circulation—was noted in only 1 patient.

**Laboratory Data.** Lumbar punctures were performed in 46 patients. In 1 the cerebrospinal fluid was grossly bloody, and had a pressure of 340 mm. water; in 5 other patients the fluid was xanthochromic; in the remaining 40 patients the fluid was clear and under normal pressure. The protein content of the spinal fluid was under 50 mg. per cent in 26 patients, from 50 to 80 mg. per cent in 14 patients, from 80 to 110 mg. per cent in 5 patients, and 111 mg. per cent in 1 patient.

Electroencephalograms were performed in 44 patients. Twenty-nine of these had focal abnormalities (usually slow wave activity) on the side of the occlusion; 6 patients had diffuse abnormalities, and 8 had normal records. In 7 of these last patients, an abnormal record was produced by manual compression of the patent carotid.

X-rays of the skull revealed a significant pineal shift in 2 patients, and films of the neck demonstrated calcification of the carotid arteries in only 2 of the 21 patients in whom such calcification was sought. Pneumoencephalograms were performed in 13 patients; they were normal in 4, showed symmetrically dilated ventricles in 7, and possibly indicated slight displacements in 2 others.

**Arteriography.** Carotid arteriography was performed in 47 patients. In 23 of these patients the contralateral carotid was also injected, and in 3 vertebral studies were performed. The clinical courses of only 2 patients tended to progress immediately following arteriography. In another 2 patients there were transient neurologic deficits following angiography of the patent carotid; however, these deficits lasted less than 24 hours. No other complications were reported following angiography.
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Pathology. The brains of the 4 patients with carotid occlusions who came to autopsy revealed more or less extensive softening in the distribution of the middle cerebral artery and a lesser extent in the anterior. Secondary brain stem hemorrhages were present in all 4, and 2 had significant uncal and cingulate herniations. The surgically resected portions of the carotid arteries of the 6 patients who underwent surgery revealed fairly severe atheromatous changes with thrombus formation.

From the arteriographic and autopsy findings, the location and nature of the carotid occlusions were divided as follows: 28 of the occlusions were in the right carotid alone, 20 were in the left alone, and in 2 patients bilateral complete carotid occlusions were demonstrated. Of the 50 patients, 36 had complete occlusions (fig. 2), while 14 had significant partial occlusions (fig. 3). The common carotid artery was the site of occlusion in 2 patients, the carotid “siphon” was occluded in 4 patients, and the distal intracranial portion of the internal carotid was the site of 1 occlusion. The most common site for occlusion, however, was the internal carotid artery in the neck, where such lesions were demonstrated in the remaining 43 patients. These occlusions occurred most commonly at the origin of the internal carotid.

Prognosis and Therapy. Ten of the 50 patients in the present series have died. Death occurred from 48 hours to 2 years after the onset of symptoms, probably due to carotid occlusion. Seventeen patients showed some neurologic improvement and have maintained such improvement. The remaining 23 patients showed no definite change from their condition at the time of admission, or have been lost to follow-up study.

Fourteen patients in the present series have been placed on anticoagulant therapy in an attempt to prevent further vascular occlusion. Six patients underwent neck surgery in an attempt to correct the existing carotid occlusion. The procedures employed were thrombendarterectomy in 4, and carotid-jugular anastomosis in 1 (in 1950). The clot was too extensive for any definitive procedure in the sixth patient. One patient in the present series was treated with intramuscular trypsin.
TABLE 1.—Frequency of Carotid Occlusions as Determined by Several Means of Study

<table>
<thead>
<tr>
<th>Method of study</th>
<th>Author, reference</th>
<th>Number of patients studied</th>
<th>Percentage with carotid occlusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>I Routine consecutive autopsies</td>
<td>Hultquist¹¹</td>
<td>1300</td>
<td>4.4</td>
</tr>
<tr>
<td>I Routine consecutive autopsies</td>
<td>Fisher⁹</td>
<td>432</td>
<td>9.5*</td>
</tr>
<tr>
<td>II Autopsies in patients with cerebrovascular disease</td>
<td>Samuel¹⁵</td>
<td>82</td>
<td>6.1</td>
</tr>
<tr>
<td>II Autopsies in patients with cerebrovascular disease</td>
<td>Hutchinson and Yates¹⁶</td>
<td>83</td>
<td>39.2*</td>
</tr>
<tr>
<td>III Consecutive arteriograms in patients suspected of having intracranial mass lesions</td>
<td>Moniz et al.⁶</td>
<td>500</td>
<td>0.8</td>
</tr>
<tr>
<td>III Consecutive arteriograms in patients suspected of having intracranial mass lesions</td>
<td>Feiring¹⁷</td>
<td>500</td>
<td>1.4</td>
</tr>
<tr>
<td>III Consecutive arteriograms in patients suspected of having intracranial mass lesions</td>
<td>Thomson¹⁸</td>
<td>1800</td>
<td>1.2</td>
</tr>
<tr>
<td>III Consecutive arteriograms in patients suspected of having intracranial mass lesions</td>
<td>Batley¹²</td>
<td>730</td>
<td>1.0</td>
</tr>
<tr>
<td>III Consecutive arteriograms in patients suspected of having intracranial mass lesions</td>
<td>Norris et al.²⁰</td>
<td>349</td>
<td>2.6</td>
</tr>
<tr>
<td>IV Consecutive arteriograms in patients suspected of having cerebrovascular disease</td>
<td>Riishede²⁰</td>
<td>100</td>
<td>14.0*</td>
</tr>
<tr>
<td>IV Consecutive arteriograms in patients suspected of having cerebrovascular disease</td>
<td>Tatelman²²</td>
<td>200</td>
<td>21.5*</td>
</tr>
</tbody>
</table>

*Includes significant stenosis as well as complete occlusions.

Twenty-nine patients received no specific therapy.

Because anticoagulant and surgical therapy have been employed largely only in the last 18 months, the follow-up period is relatively short. At this time, therefore, data cannot be presented concerning the effects of therapy on the natural course of the disease.

**Discussion**

*Historical Background.* The first pathologic description of a carotid occlusion dates back to Willis³ in 1664. An early report of the condition was made by Cushing,⁴ in 1900, and Hunt³ in 1914 described the classical syndrome produced by occlusion of the common carotid artery and stressed the value of palpation of the carotid pulsations in the neck as a means of diagnosis. Interest in carotid occlusion, however, remained slight until the development of cerebral arteriography, and the report of 4 patients so diagnosed by Moniz et al.⁶ in 1937. By 1951 Johnson and Walker⁷ had collected 101 proved cases of carotid occlusion from the literature and added 6 more. The entity of carotid occlusion was firmly established with the subsequent writings of Fisher,⁸–¹⁰ Webster and Gurdjian¹¹–¹³ and others.

*Incidence.* Carotid occlusive disease is not rare (table 1). The incidence of carotid narrowing or occlusion in routine, unselected autopsies may be as high as 9.5 per cent,¹⁰ whereas 39 per cent of patients with cerebrovascular disease may have significant carotid stenosis.¹⁶ Carotid occlusion may be diagnosed arteriographically in 14 to 21.5 per cent of patients presenting the acute "stroke" syndrome.²¹,²² Fields et al.²³ found that 25 per cent of patients with cerebrovascular disease have extracranial (i.e., carotid and vertebral) occlusive disease.

Atherosclerosis is by far the most common cause of carotid occlusion. Significant atherosclerosis in the internal carotid artery has been found in 46 per cent of patients over 45,²¹ and as many as 80 per cent of older age patients have such atheromas.¹⁵,²⁵

*Etiology.* The various etiologies or predisposing conditions associated with carotid occlusions as reported in the literature are listed alphabetically in table 2. In the present series, moderate to severe degrees of atheroma formation were noted in almost all 10 (6 surgical and 4 autopsy) specimens of the carotid arteries studied. Evidences of atherosclerosis elsewhere in the body, or conditions predisposing to its development, were present in 76 per cent of the 50 patients in the present series.

*Clinical Features.* The signs and symptoms of carotid occlusion are quite variable, and can mimic those of several other diseases of the brain. Thus, patients presenting progressive neurologic deficit (46 per cent of the present series) may well be suspected of having cerebral neoplasms, especially when headaches (20 per cent) or focal seizures (28 per cent) occur. Although not observed in the present series, papilledema has been reported...
to occur with carotid occlusion.\textsuperscript{7, 37} Spinal fluid protein may be quite high. If air studies are undertaken as the first definitive diagnostic procedure, the erroneous diagnosis of brain tumor may be further supported by significant displacements of the ventricular system.\textsuperscript{41, 42} Such displacements, as well as the 2 shifted pineal bodies noted in the present series, are undoubtedly the results of cerebral edema.

Another common onset of carotid occlusion in a sudden vascular accident (72 per cent in the present series), which may be completely indistinguishable from middle cerebral artery occlusion.\textsuperscript{21, 30} The pathologic changes in the brain at autopsy with carotid occlusion are similar to those found with middle cerebral artery occlusion.\textsuperscript{32, 37} Thus, hemiparesis, the most constant finding with carotid occlusion (98 per cent in the present series), is characterized by greater severity in the upper than in the lower extremity.

Many patients with carotid occlusions (26 per cent of the present series) have recurrent unilateral manifestations of cerebral dysfunction, the syndrome of "intermittent insufficiency of the carotid arterial system,"\textsuperscript{143} or "transient ischemic attacks."\textsuperscript{144} Because this syndrome occurs with partial carotid occlusions,\textsuperscript{43} it is mistakenly considered to be almost diagnostic of carotid occlusive disease. We have recently seen many patients with intermittent episodes of unilateral cerebral dysfunction and patent carotid arteries at arteriography; in a few of these patients a cerebral neoplasm was eventually demonstrated. An incomplete history or lack of observation during an episode may make differentiation impossible between "ischemic" episodes and postictal phenomena.\textsuperscript{44} For these reasons, the increasing use of anticoagulant therapy is not approved in patients with recurrent cerebral episodes—without adequate investigation to determine etiology.

There are other clinical manifestations of carotid occlusions that seem to occur more commonly in the textbooks than in patients. Thus the association of transient monocular blindness with contralateral hemiplegia, although suggestive of carotid occlusion,\textsuperscript{9} occurred in only 10 per cent of the present series. The same may be said about the diagnostic significance of an ipsilateral Horner’s syndrome\textsuperscript{37, 46} (4 per cent), ipsilateral optic atrophy\textsuperscript{5} (2 per cent), and dilatation of the superfi cial vessels of the face\textsuperscript{37} (2 per cent). Among the more unusual fi ndings in our series and in the literature are subarachnoid hemorrhage, extrapyramidal signs, depression and impairment of eye movements. It should be noted that completely asymptomatic carotid occlusions may not be rare.\textsuperscript{3, 10, 15, 20}

Four reportedly diagnostic bedside tests of carotid occlusion have been frequently discussed in the recent literature. These procedures are (1) palpation of the carotid pulse in the neck and in the pharynx; (2) auscultation of a carotid or intracranial bruit; (3) measurement of the retinal artery pressures (ophthalmodynamometry) and (4) manual compression of the contralateral carotid. In a study\textsuperscript{1} of 30 personally observed patients from this series, diminished carotid pulsations were present in only 17 per cent of the pa-

### Table 2.—Reported Etiologies and Predisposing Conditions for Carotid Occlusion

<table>
<thead>
<tr>
<th>Condition and reference no.</th>
<th>Condition and reference no.</th>
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</thead>
<tbody>
<tr>
<td>Atlantoid compression\textsuperscript{24}</td>
<td>Lupus erythematosus (present series)</td>
</tr>
<tr>
<td>Alcoholism\textsuperscript{27}</td>
<td>Mucormycosis\textsuperscript{24}</td>
</tr>
<tr>
<td>Aneurysm with thrombosis\textsuperscript{22}</td>
<td>Periarteritis nodosa\textsuperscript{27}</td>
</tr>
<tr>
<td>Atherosclerosis\textsuperscript{*}</td>
<td>Polycythemia vera\textsuperscript{25}</td>
</tr>
<tr>
<td>Cardiac surgery\textsuperscript{7}</td>
<td>Pulsatile disease\textsuperscript{136}</td>
</tr>
<tr>
<td>Cervical ril\textsuperscript{29}</td>
<td>Rheumatic fever\textsuperscript{27}</td>
</tr>
<tr>
<td>Compression by neoplasms\textsuperscript{26}</td>
<td>Sinusitis, cavernous\textsuperscript{26}</td>
</tr>
<tr>
<td>Congenital abnormalities\textsuperscript{21}</td>
<td>Syphilis\textsuperscript{31}</td>
</tr>
<tr>
<td>Embolism\textsuperscript{22}</td>
<td>Temporal arteritis\textsuperscript{27, 26}</td>
</tr>
<tr>
<td>Erythroblastosis\textsuperscript{31}</td>
<td>Thromboangiitis obliterans\textsuperscript{26, 35, 36}</td>
</tr>
<tr>
<td>Infections\textsuperscript{27, 28}</td>
<td>Trauma\textsuperscript{39, 40}</td>
</tr>
</tbody>
</table>

\*Atherosclerosis is considered by almost all authors the major etiologic factor for carotid occlusion.
tients. Palpation of pharyngeal pulsations was of no diagnostic aid. A carotid bruit was heard in 13 per cent of the patients. Significant differences of retinal artery pressure were present in 80 per cent, and compression of the patent carotid artery gave "positive" results in 70 per cent of the patients studied. The nature, extent, and rapidity of development of collateral circulation are probably the significant factors responsible for the varied clinical pictures of carotid occlusion. ¹

Angiography. It should be apparent that a definitive diagnosis of carotid occlusion cannot be made on clinical grounds alone. To establish such a diagnosis, and also to exclude space-occupying lesions that may be excised or radiosensitive, carotid arteriography must be performed. The safety of this procedure, even in elderly patients with "vascular disease," is being accepted at increasing numbers of neurologic centers since the development of several newer, less toxic contrast media.

There were only 2 possible persisting complications from the 73 (47 in the occluded carotid, 23 bilateral, and 3 vertebral) angiograms performed in the present series. The recently reported 12 per cent incidence of serious complications in 500 consecutive angiograms at this hospital was derived entirely from experience with older contrast media. ²⁷ Since the completion of this study, 1,000 further angiograms have been performed with the newer, safer contrast media. The incidence of complications in the present series of arteriograms, although the findings are not yet tabulated, is definitely much lower. British workers have reported the incidence of serious complications with Hypaque to be 0.7 per cent. ⁴⁸ Similar conclusions concerning the newer contrast media, even when employed in patients with cerebrovascular disease, have been reached by many others. ¹³, ²¹-²³, ²⁶

Though rare, occasional complications do follow arteriography. In general, in patients with evidence of a focal brain lesion, the amount of information to be obtained by arteriography in most cases outweighs the risks of the procedure. It is recommended, therefore, that every patient with unilateral cerebral disease be evaluated for arteriography after appropriate preliminary study. Only by such means can a presently correctible lesion be excluded, or the diagnosis of a potentially correctible carotid occlusion be established. The evidence that prompt therapy for carotid occlusion, once confirmed, may improve the existing symptomatology or at least prevent further disability will be reviewed subsequently.

Comparison of Present Series with Other Reports. The clinical manifestations of carotid occlusion in this series have not appreciably differed from those previously described (table 3).

### Table 3.—Incidence of Common Clinical Findings Reported with Carotid Occlusion

<table>
<thead>
<tr>
<th>Author and reference no.</th>
<th>No. of patients</th>
<th>Progressive course</th>
<th>Intermittent course</th>
<th>Hemiparesis</th>
<th>Sensory deficit</th>
<th>Aphasia</th>
<th>Field defect</th>
<th>Organic mental syndrome</th>
<th>Headache</th>
<th>Seizures</th>
<th>Ischemic Blindness</th>
<th>Optic atrophy</th>
</tr>
</thead>
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<tr>
<td>Johnson and Walker</td>
<td>107*</td>
<td>25</td>
<td>40</td>
<td>80</td>
<td>20</td>
<td>60</td>
<td>11</td>
<td>15</td>
<td>50</td>
<td>20</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Shapiro and Peyton</td>
<td>17</td>
<td>12</td>
<td>59</td>
<td>100</td>
<td>41</td>
<td>53</td>
<td>12</td>
<td>18</td>
<td>6</td>
<td>0</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>Thomson</td>
<td>23</td>
<td>48</td>
<td>48</td>
<td>91</td>
<td>70</td>
<td>60</td>
<td>39</td>
<td>9</td>
<td>70</td>
<td>17</td>
<td>4</td>
<td>4</td>
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<tr>
<td>Webster et al.</td>
<td>63</td>
<td>10</td>
<td>27</td>
<td>81</td>
<td>17</td>
<td>36</td>
<td>4</td>
<td>19</td>
<td>21</td>
<td>9</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>Boldrey et al.</td>
<td>24</td>
<td>17</td>
<td>33</td>
<td>88</td>
<td>43</td>
<td>17</td>
<td>8</td>
<td>?</td>
<td>83</td>
<td>38</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Jacobsen and Skinhos</td>
<td>27</td>
<td>37</td>
<td>11</td>
<td>70</td>
<td>52</td>
<td>44</td>
<td>22</td>
<td>44</td>
<td>37</td>
<td>4</td>
<td>?</td>
<td>11</td>
</tr>
<tr>
<td>Sastrasin</td>
<td>65</td>
<td>63</td>
<td>63</td>
<td>92</td>
<td>31</td>
<td>52</td>
<td>22</td>
<td>37</td>
<td>57</td>
<td>8</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Silverstein</td>
<td>50</td>
<td>46</td>
<td>26</td>
<td>98</td>
<td>64</td>
<td>42</td>
<td>52</td>
<td>44</td>
<td>20</td>
<td>28</td>
<td>10</td>
<td>2</td>
</tr>
</tbody>
</table>

*One hundred and ten of these patients were collected from the literature.
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Two significant differences between the present series and the majority of other reports deserve comment. In the present series the right carotid artery was found occluded considerably more often than the left. In almost all of the previously published series, left carotid occlusions predominated. No immediate explanation is available for this discrepancy. The sites of carotid occlusion in this series are otherwise similar to those reported elsewhere. As a prelude to a discussion of therapy, it should be stressed that the overwhelming majority of carotid occlusions occur in the neck, a most accessible site.

The second major difference between this and other series of carotid occlusions is the observation here of predominantly symmetrical ventricles in most of the 13 patients in whom pneumoencephalograms were performed. Several previous writers have described unilateral cerebral atrophy on the side of the occlusion.7, 52 Again the explanation for these conflicting findings is obscure.

Therapy. The results of therapy in the present series have not been presented because the number of patients adequately treated is small, and the follow-up periods are brief. At the present time there are insufficient data to determine which of the 2 major forms of currently recommended therapy for carotid occlusion is to be preferred.

Most writers agree now that previously suggested therapies such as carotid sinus denervation, cervical sympathectomy, carotid-jugular anastomosis, simple resection of a portion of the occluded carotid, and other measures, are probably of little value. The currently accepted forms of therapy for carotid occlusion are anticoagulant therapy and reconstruction of the occluded vessel.

Anticoagulants. Most of the present exponents of anticoagulant therapy for cerebrovascular diseases usually do not perform definitive diagnostic, i.e., arteriographic, studies. Most of these workers would agree that there is little to be gained from such therapy in patients with complete carotid, or other occlusions and maximal neurologic deficit. At least one patient, however, with an angiographically verified complete carotid occlusion had partial patency on repeated angiography following anticoagulant therapy.53 If the mechanism of action of anticoagulant therapy in cerebral vascular disease is the protection of collateral circulation,54 then even patients with complete occlusions and maximum deficit should be treated. No adequate data, however, have yet been published to settle this point. In a small series55 no differences in recovery were detected on the basis of anticoagulant therapy, and 1 patient went on to develop a carotid occlusion (confirmed at autopsy) while on adequate amounts of therapy.

There is evidence, however, that patients with recurrent "ischemic" attacks, as may be seen with partial carotid occlusions, are benefited by anticoagulants.44, 56-59 It must be stressed that intermittent symptoms are not pathognomonic of carotid or other cerebral occlusions. However, several of the patients in whom intermittent symptoms were relieved by anticoagulant therapy, had findings suggestive of carotid occlusion, i.e., positive ophthalmodynamometric readings that reverted to normal following therapy,58 unilateral blindness and contralateral paresis,59 etc. It also seems that patients with progressive neurologic deficit due to carotid and other occlusions are helped by anticoagulant therapy.44, 58

The other major area in which anticoagulants appear to be effective is in the reduction of embolic occlusions in the cerebral vessels.60-63 Carotid occlusions from emboli, as in patients with rheumatic heart disease, have been reported only occasionally,28, 32 although they may have occurred much more frequently than previously suspected.

The need for strict control of anticoagulant therapy in neurologic patients who may be aphasic or have organic mental syndromes must be particularly stressed. If the once commonly discussed belief64 is valid, that massive cerebral hemorrhage is precipitated by diseased cerebral vessels and ischemic softening, the hazards of anticoagulant therapy in
patients with occlusive cerebral vascular disease may be great. The most recently reported incidence of cerebral hemorrhage in such patients on anticoagulant therapy was 5 per cent. 63 This hazard may or may not be greater with recent occlusions.

Reconstructive Surgery. Complications may also occur with surgical therapy for carotid occlusion. 65 These may include further cerebral damage from either ischemia or hypotension; the release of emboli during manipulation of the occluded carotid; and the production of postoperative thrombi with extension intracranially or proximally into the aorta. 66 Such complications, however, have been reported rather infrequently with carotid surgery, and probably can be prevented somewhat by utilization of hypothermia during surgery, the avoidance of excessive damage to the vessel wall, and postoperative anticoagulant therapy. 66

There are 5 currently reported means of reconstruction for carotid occlusion: thrombectomy, 65, 67 thrombo-endarterectomy, 23, 53, 66, 68-72 side-to-side anastomosis between the external and internal carotid arteries; 53, 73 resection of the occluded portion of the carotid and end-to-end anastomosis, 66, 74-76 or replacement with a (venous) graft, 77 or internal-external carotid anastomosis, 65, 70, 78 and bypass grafts made of nylon, 79 Dacron, 23 or a homograft. 80, 81 A temporary polyvinyl shunt has also been employed to facilitate thrombo-endarterectomy. 69

Although experience is insufficient at the present time to evaluate these procedures, there is probably little value to be gained from simple thrombectomy or internal-external carotid anastomosis. The relative merits of anticoagulant and surgical therapy for carotid occlusive disease are also unsettled. There is some evidence, however, that patients operated on for partial occlusions or immediately after the onset of a complete occlusion may improve to a greater degree than would occur without therapy. Thus, the recurrent “ischemic” symptoms may completely subside after surgery. 23, 67, 71, 78 postoperative angiograms may show patency for at least several months, 53, 66, 67, 71, 73, 77, 78 and previously abnormal results with ophthalmodynamometry and carotid compression may return to normal. 71, 82

On the other hand, the majority of patients with complete occlusions of more than several hours’ duration, have not tended to show significant improvement from reconstructive procedures. Some of the few patients who have improved following surgery, were subsequently demonstrated to have occluded vessels at postoperative angiography.

To summarize the reported results of anticoagulant and surgical therapy for carotid occlusive disease, (1) both forms of treatment are probably ineffective for long-standing complete occlusions with major neurologic deficit; (2) both therapies may help patients with acute complete occlusions and progressing deficit; (3) both therapies can eliminate the recurrent “ischemic” symptoms frequently seen in patients with partial carotid occlusions; and (4) it is not known at the present time which form of therapy is to be preferred, and under what circumstances each should be employed. The data are also incomplete concerning the complications from either form of therapy.

Conclusions

An attempt has been made to demonstrate how frequently carotid occlusions are responsible for so-called “strokes.” The varying clinical pictures produced by such occlusions have been discussed, and the relatively rare occurrence of previously considered diagnostic features was noted. The only 2 such features that occur with significant regularity are positive results with ophthalmodynamometry and carotid compression; even these are not always present and false-positive tests do occur. The procedure of choice for the diagnosis of a carotid occlusion is arteriography. The reasons why the current application of this procedure is considered safe are presented.

There is evidence that both anticoagulant and surgical reconstructive therapy are of
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aid in the management of the intermittent or mild symptoms of partial carotid occlusion; they may also help the severe or progressive symptoms associated with a fresh complete occlusion. Unfortunately, these particular patients—those with only a slight hemiparesis, or those asymptomatic in between recurrent “ischemic” attacks—are the very ones upon whom most physicians have been reluctant to perform arteriography.

It is recommended that arteriography be employed in such patients as a definitive diagnostic procedure, because (1) possibly corrective space-occupying lesions can produce such symptoms; (2) anticoagulant and surgical therapy for carotid occlusions may be effective only in such patients; and (3) such patients seem not to be significantly hurt by arteriography with the newer contrast media. This procedure, however, should follow certain basic preliminary studies including a detailed history, complete physical examination, electrocardiogram, and lumbar puncture. The performance of the procedure on an outpatient basis is not recommended.

The institution of potentially hazardous therapeutic measures, such as anticoagulant therapy or neck exploration under anesthesia, prior to the establishment of the diagnosis, is not consistent with the usually accepted principles of proper medical practice. Prompt institution of therapy, once the diagnosis is established, however, may make a significant difference in the recovery of lost function, or in the prevention of further deficit in patients with carotid occlusive disease.

SUMMARY

Occlusions of the carotid arteries may be responsible for over 20 per cent of all acute cerebral vascular lesions. The clinical and laboratory features of 50 proved patients with this disease are presented. The clinical pictures produced by carotid occlusions are quite variable, and can mimic those of brain tumors, middle cerebral artery occlusions, and other conditions. There are few clinically diagnostic features for carotid occlusions. Definitive diagnosis requires arteriographic study. There is some evidence that early institution of anticoagulant or surgical reconstructive therapy in certain patients with carotid occlusions is indicated.

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SUMMARIO IN INTERLINGUA

Il es possibile que occlusiones del arterias carotidic es responsable pro plus que 20 pro cento de omne acute lesiones cerebro-vascular. Es presentate le caracteristicas clinic e laboratorial de 50 patientes confirmate de iste morbo. Le tableaus clinic producite per occlusion carotidic es multo variabile. Illos pote similar tumores cerebral, occlusion de arteria centro-cerebral, e altere conditiones. Il existe pauc aspectos clinico-diagnostic de occlusion carotidic. Le definitive diagnose require studios arteriographic. Certe observationes supporta le theses que le precoce institutione de therapia anticoagulante o chiruro-reconstructive es indicare in seligite patientes con oclusion carotidic.

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Medical Eponyms

By Robert W. Buck, M.D.

Bright's Disease. Richard Bright (1789-1858), Lecturer on the Practice of Medicine, and one of the physicians to Guy's Hospital, included in his Reports of Medical Cases, Selected with a View of Illustrating the Symptoms and Cure of Diseases by a Reference to Morbid Anatomy (London, Longman, Rees, Orme, Brown, and Green, vol. 1, 1827), the results of his investigations of the pathological conditions associated with albuminous urine. Their epoch-making character has served to attach his name permanently to the whole group of nonsurgical diseases of the kidney. The following quotation is taken from the introductory remarks of the author (page 2):

"The different diseases of the heart and of the lungs on which dropsy depends, and the various changes to which the liver is subject rendering it a cause of impediment to the circulation, are still open to much investigation. . . ."

"There are other appearances to which I think too little attention has hitherto been paid. They are those evidences of organic change which occasionally present themselves in the structure of the kidney, and which, whether they are to be considered as the cause of the dropsical effusion or as the consequence of some other disease, cannot be unimportant. Where those conditions of the kidney to which I allude have occurred, I have often found the dropsy connected with the secretion of albuminous urine, more or less coagulable on the application of heat. I have in general found that the liver has not in these cases betrayed any considerable marks of disease, either during life or on examination after death, though occasionally incipient disorganization of a peculiar kind has been traced in that organ. On the other hand, I have found that where the dropsy has depended on organic change in the liver, even in the most aggravated state of such change no diseased structure has generally been discovered in the kidneys, and the urine has not coagulated by heat. I have never yet examined the body of a patient dying with dropsy attended with coagulable urine, in whom some obvious derangement was not discovered in the kidneys."
Occlusive Disease of the Carotid Arteries
ALLEN SILVERSTEIN

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