Internal Carotid Artery Occlusion: Clinical and Therapeutic Implications

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SUMMARY Five hundred patients referred to the Cerebrovascular Clinic of the Johannesburg Hospital were examined by a battery of noninvasive tests and angiography. Thirty-four occlusions of the internal carotid artery were found in 32 patients. These patients were prospectively evaluated, including clinical examination, analysis of risk factors and subsequent management. This group of patients was followed up for a mean period of 18 months, and the clinical and laboratory findings and follow up data of this group were compared to an age and sex matched group of patients with matched presenting symptoms, but with patent internal carotid arteries on angiography. Four clinical patterns emerged in the patients with occluded carotid arteries: asymptomatic (3), TIA's (17), initial fixed stroke (7), and TIA with subsequent stroke (5). Follow up of the occluded group revealed 19 patients (59%) with no further symptoms and no indication for surgical intervention. Nine patients required surgery; 4 external carotid endarterectomies (ipsilateral), 4 internal carotid endarterectomies (contralateral), and one extracranial to intracranial bypass. Two were lost to follow up and one died. After 18 months mean follow up 29 patients (91%) were well and asymptomatic. Follow up for a similar period of the non-occluded group revealed three deaths, three late strokes and three myocardial infarctions. None were lost to follow up. After 19 months mean follow up 26 patients (81%) were well with no new neurological symptoms. The prognosis of appropriately treated patients with total occlusion of the internal carotid artery does not appear to be worse than in patients with similar presenting features and patent carotid arteries. The role of surgery in preventing further emboli despite the presence of a total internal carotid occlusion is highlighted.

OCCLUSION OF THE INTERNAL CAROTID artery has aroused clinical interest as a result of the appreciation of the poor correlation which has been demonstrated to exist between the angiographic findings and the associated neurological symptoms.1,2 The number of patients with significant strokes and residual symptoms after the occlusion of an internal carotid artery are less impressive than initially suspected, with only 27% having severe complicated strokes and 73% being asymptomatic or experiencing only transient or mild focal neurological disorders.2 Occlusion of the internal carotid artery may, however, be associated with transient or fixed neurological symptoms after the occurrence of the occlusion, and the evidence strongly suggests that such symptoms are frequently related to embolic disease originating from the external carotid artery3 or the proximal blind stump of the internal carotid artery.3 This concept is in direct conflict with the opinion of Gomensoro et al. that "when a lesion progressed to complete thrombosis, there was no longer any possibility for emboli to pass into the distal circulation."6

The benign clinical course and the importance of the external carotid artery collateral circulation and the consequent therapeutic implications, became evident to us in our series of 500 patients presenting to the Cerebrovascular Clinic of the Johannesburg Hospital predominantly with transient or minor focal cerebral vascular events. Forty two patients of 700 studied in the Vascular Laboratory7 were demonstrated to have occlusion on angiography. Thirty two of these patients were prospectively evaluated and followed up by at least one of the authors at the Cerebrovascular Clinic of the Johannesburg Hospital between January 1980 and December 1983. This paper presents the clinical picture and related investigations and management of these 32 patients (34 internal carotid occlusions) and documents the benign follow up course of this group of patients provided appropriate attention is paid to the remaining collateral vessels supplying the brain.

Patients and Methods

Five hundred patients with carotid or vertebrobasilar transient ischaemic attacks, minor strokes or asymptomatic carotid bruits were evaluated prospectively at the Cerebrovascular Clinic of the Johannesburg Hospital between 1980 and 1983. All patients were assessed clinically, including a full cardiac evaluation and electrocardiogram. Blood tests performed routinely included haemoglobin, haematocrit, clotting profile, blood sugar, cholesterol, uric acid, thyroid function and Wasserman Reaction. Each patient underwent a combination of noninvasive tests7 consisting of carotid phonoangiography with split frequency display (Narco Biosystems), Doppler ultrasound carotid velocity assessment utilising the Medasonic D9 directional Doppler unit with zero crossing detector analog output, directional Doppler evaluation of the supraorbital flow...
including response to superficial temporal artery compression and "turbulence index" with internal carotid artery signal identification. Conventional contrast angiography was only carried out in selected patients where a specific clinical indication existed. Angiography was carried out in patients with one or more episodes of carotid territory transient ischaemic attacks, amaurosis fugax, carotid territory reversible ischaemic neurological deficits and carotid territory strokes provided that the latter underwent a major degree of recovery. Angiography was not as a rule carried out in patients totally unfit for surgical therapy, patients over 75 years of age and patients who presented with clinical features of vertebrobasilar insufficiency and with normal noninvasive carotid artery studies.

One hundred and sixty four of these patients have undergone conventional contrast arteriography, yielding 320 contrast investigations (sides) of good diagnostic quality. Patients who had angiography prior to being referred to the Clinic are included in this study but were excluded in another study analysing the sensitivity of the noninvasive tests in detecting occlusion of the carotid artery. This study analyses the clinical and laboratory features of 32 patients (34 occlusions, two bilateral) in whom follow up data of not less than 6 months was available. The results of the analysis of the clinical and laboratory findings and follow up data of this group were compared to those obtained from a nonoccluded series of patients. This nonoccluded group consisted of age and sex matched patients selected from the same population of patients presenting with matched clinical symptoms, but documented to have patent internal carotid arteries on the basis of conventional contrast arteriography. Follow up data of patients with angiographic occlusion not prospectively evaluated and of patients with occlusion on noninvasive testing without confirmatory angiography, are included for comparison.

Results
Clinical Features
There were 21 males and 11 females with a mean age of 58.84 years (sd 6.89, range 44–69) compared to the nonoccluded group mean age of 58.09 years (sd 7.81, range 44–70). The presenting clinical symptoms of the occluded group were divided into four categories:

i. asymptomatic
ii. transient ischaemic attack
iii. initial fixed stroke
iv. initial transient ischaemic event followed by fixed stroke.

The symptoms of the nonoccluded group were selected to match those of the occluded group and the same number of cases were included in each category of symptoms.

Asymptomatic
Three patients were documented to have totally asymptomatic occlusions when angiography was performed for an unrelated clinical indication. In one patient the indication for angiography was the presence of an asymptomatic carotid bruit in a patient due to undergo aorto-iliac surgery. In two other patients unsuspected occluded carotid arteries were found when angiography was performed for a transient ischaemic event appropriate to a contralateral stenosed internal carotid artery.

Transient Ischaemic Episode
Seventeen patients presented with between one and twenty five episodes of transient ischaemic attacks. In nine patients amaurosis fugax alone was the presenting feature. Six patients had cerebral transient ischaemic attacks only with no visual disturbances. All these episodes occurred in the left carotid artery territory. Two patients had combined amaurosis fugax and carotid territory transient ischaemic episodes, one appropriate to the right and one to the left carotid arteries.

Initial Fixed Neurological Deficit
Seven patients presented with a fixed initial neurological loss. One patient suffered permanent blindness of the right eye due to central retinal artery occlusion which presumably occurred at the time of the carotid thrombosis. A further two patients presented with amaurosis fugax of the left eye followed within two hours by a right hemiparesis. The remaining four patients presented with carotid territory stroke as the initial event, two appropriate to each side.

Initial Transient Ischaemic Event Followed by Fixed Stroke
Five patients had initial transient carotid territory events which were ignored by the patient. In all cases these were multiple episodes either of amaurosis fugax or of transient limb paresis appropriate to the carotid artery which was subsequently documented to have undergone occlusion. These patients presented with a permanent neurological deficit.

Risk Factors
The risk factors were compared between the occluded group and the matched group with angiographically patent arteries. This comparison, detailed in table 1 suggests that factors which are a marker for diffuse systemic vascular disease, are a more prominent feature of the occluded group than the nonoccluded group.

Follow Up
The 32 patients with angiographically documented occlusion in this study were followed up for a mean period of 18.9 months (range 6 to 40 months, s.d., 9.8 months). Table 2 contains the details of this group. Two were lost to follow up after six months. Four patients continued to have transient focal ischaemic events or episodes of amaurosis fugax after the occlusion was documented angiographically. In each case this was proven to be due to disease of the carotid bifurcation (stump or external carotid artery origin stenosis) and no further episodes occurred after external carotid endarterectomy. Three patients developed...
contralateral symptoms, one dying after contralateral stroke, one occluding the contralateral artery, and one requiring a contralateral endarterectomy for these symptoms. Prophylactic elective carotid endarterectomy was carried out in 3 asymptomatic patients with contralateral carotid artery stenosis of >50 percent. One patient with bilateral occlusions had extracranial to intracranial bypass surgery for persistent nonhemispheric symptoms. Thus 19 patients have, on regular follow up, remained well and have not developed focal neurological symptoms or required surgery. To this can be added the 3 patients who had prophylactic contralateral carotid endarterectomy but have remained asymptomatic.

However, following appropriate medical or surgical therapy, 29 of the original 32 cases assessed at a mean period of 18 months from the commencement of this study were found to be well and symptom free (table 3) other than the residual neurological deficits remaining after the presenting episode. The only patients who have not been documented to be well after 18 months are the two lost to follow up and the single death.

Ten patients diagnosed on noninvasive tests alone as having internal carotid artery occlusion, but not confirmed angiographically, have been followed up for a similar period. Only one of these patients has had a contralateral stroke which resulted in death during this follow up period. The others have remained well.

A further ten patients identified as having occlusion angiographically in the Vascular Laboratory, but not followed clinically by the Cerebrovascular Clinic were traced through their referring physicians. All have remained well and asymptomatic after a mean 18 month follow up period.

During a similar mean 19 month follow up period in the nonoccluded group, nine underwent uneventful carotid endarterectomy for symptomatic carotid disease and have remained well. The remainder were treated with appropriate medical therapy as no surgically appropriate lesion was present. Of these three have died — one following a stroke, one due to a myocardial infarct and one due to an unrelated malignancy. Three further patients have had late strokes. Two patients developed myocardial infarcts but recovered during follow up. Thus following appropriate medical or surgical therapy 26 patients remain free of neurological symptoms after a mean follow up period of 19 months (table 3).

Management of the Opposite Side

Although 26 of the 30 patients followed for longer than 6 months remained without symptoms referable to the occluded side, a high proportion of all the patients had contralateral carotid disease. In 2 patients the contralateral carotid artery was occluded, and in a further

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**TABLE 1** Risk Factors in 32 Patients with Occluded Carotid Arteries Compared with 32 Age and Sex Matched Patients with Patent Carotids

<table>
<thead>
<tr>
<th>Factor</th>
<th>Nonoccluded group</th>
<th>Occluded group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous stroke</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>Cardiac ischaemia</td>
<td>16</td>
<td>22</td>
</tr>
<tr>
<td>Cardiac valvular</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td>Peripheral vascular disease</td>
<td>11</td>
<td>15</td>
</tr>
<tr>
<td>Hypertension</td>
<td>21</td>
<td>25</td>
</tr>
<tr>
<td>Smoking</td>
<td>23</td>
<td>29</td>
</tr>
<tr>
<td>Hormonal alteration</td>
<td>9</td>
<td>11</td>
</tr>
<tr>
<td>Alcohol</td>
<td>5</td>
<td>10</td>
</tr>
</tbody>
</table>

| Glucose*                | Mean 16.1         | Std Dev 1.66 | Mean 15.7 | Std Dev 2.23 |
| Haematocrit %           | Mean 47.8         | Std Dev 5.50 | Mean 46.5 | Std Dev 6.64 |
| Glucose*                | Mean 6.15         | Std Dev 1.34 | Mean 5.34 | Std Dev 1.22 |
| Uric acid*              | Mean .41          | Std Dev .10  | Mean .43  | Std Dev .09  |
| Cholesterol*            | Mean 6.8          | Std Dev 1.28 | Mean 7.74 | Std Dev 2.18 |
| Free Thyroxin pmol/L    | Mean 16.8         | Std Dev 2.99 | Mean 16.5 | Std Dev 5.28 |
| Wasserman reaction      | all negative      |               | all negative |               |

*mmol/L.

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**TABLE 2** Details of Follow Up of 32 Patients with Occlusion

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Therapy</th>
<th>Outcome</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymptomatic</td>
<td>Conservative</td>
<td>Well</td>
<td>19</td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>Elective contralateral endarterectomy</td>
<td>Well</td>
<td>3</td>
</tr>
<tr>
<td>Ipsilateral TIA</td>
<td>External endarterectomy</td>
<td>Well</td>
<td>4</td>
</tr>
<tr>
<td>Contralateral TIA</td>
<td>Internal carotid endarterectomy</td>
<td>Well</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Conservative (artery occluded)</td>
<td>Well</td>
<td>1</td>
</tr>
<tr>
<td>Non hemispheric (bilateral occlusion)</td>
<td>EC/IC bypass</td>
<td>Well</td>
<td>1</td>
</tr>
<tr>
<td>Contralateral stroke</td>
<td>Conservative</td>
<td>Died</td>
<td>1</td>
</tr>
<tr>
<td>Lost to follow up</td>
<td></td>
<td>—</td>
<td>2</td>
</tr>
</tbody>
</table>

**TABLE 3** Long Term Results of Occluded and Nonoccluded Groups

<table>
<thead>
<tr>
<th></th>
<th>Occluded group</th>
<th>Nonoccluded group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean follow up</td>
<td>18 months</td>
<td>19 months</td>
</tr>
<tr>
<td>Range</td>
<td>6-40 months</td>
<td>6-36 months</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>9.8 months</td>
<td>9.4 months</td>
</tr>
<tr>
<td>Neurologically asymptomatic (Myocardial infarctions)</td>
<td>29</td>
<td>26</td>
</tr>
<tr>
<td>Late strokes</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Deaths (cause)</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>(stroke) (stroke)</td>
<td>(stroke)</td>
<td>(stroke)</td>
</tr>
<tr>
<td>(myocardial infarct) (myocardial infarct)</td>
<td>(malignancy)</td>
<td></td>
</tr>
<tr>
<td>Lost to follow up</td>
<td>2</td>
<td>0</td>
</tr>
</tbody>
</table>
11 cases a stenosis of 50% or greater was present in the opposite carotid. In 5 patients low grade stenosis or angiographic ulceration was present in the opposite carotid bifurcation. In only 14 cases was the opposite carotid angiographically normal at the time of presentation.

Surgical endarterectomy was performed on the contralateral carotid artery in 4 patients. In three patients, elective endarterectomy was performed for a tight stenosis in the absence of ipsilateral symptoms. The fourth patient with 70% stenosis was initially treated conservatively, and seven months later developed transient ischaemic episodes appropriate to the stenosis for which an endarterectomy was curative. A further patient with tight stenosis refused surgery and went on to have the only subsequent stroke in these 32 patients followed up for a mean period of 18 months. One patient with 50% stenosis treated conservatively, developed an appropriate transient ischaemic event and was shown on repeat angiography to have occluded the second internal carotid artery. He remains well on follow up.

Surgical Therapy
As described above a total of 9 patients in the occluded internal carotid artery group were treated surgically. Four had ipsilateral external carotid endarterectomy, four had contralateral internal carotid endarterectomy and one had an extracranial to intracranial bypass. The single extracranial to intracranial bypass in this series was performed on a patient who had presented with a history of bilateral strokes, was documented on angiography to have bilateral occlusions and who continued to have nonhemispheric symptoms.

Discussion
This series of cases documents the relatively benign neurological course of patients with complete carotid artery occlusion evaluated prospectively in our unit. In our patients the atheroma risk factors demonstrated a trend towards being more severe than the same risk factors in an age and sex matched control group with similar presenting features and patent carotid arteries. This increased incidence of generalised atheromatous disease was also demonstrated by Dyken1 in a similar study of patients with complete carotid artery occlusion. The important implication reported by others is the associated increased risk of coronary artery disease which accounts for the major morbidity in these patients.1,12 Although in our series coronary artery disease was not a major complicating factor, attention should always be given to the associated coronary artery disease. Adams et al19 has gone so far as to suggest that coronary angiography should be performed as a routine in all patients with TIA.

Twenty of our patients presented initially with no symptoms or with transient neurological symptoms. Twelve presented with completed strokes. All these patients have made a good recovery and remain with only minor residual weakness. Of importance is the fact that 29 of 32 patients treated either conservatively or with surgical attention to the residual vascular supply remain well and asymptomatic over a mean 18 month follow up period. The two patients lost to follow up had remained well for an initial period of 6 months. A further 20 patients diagnosed as internal carotid occlusion have had an equally benign course although not studied in such detail.

These results compare favourably with the collected studies analysed by Cole et al12 In that survey only the retrospective study of Bogousslavsky et al14 showed no deaths or strokes in a mean 27 month follow up period. Eight of their patients had delayed TIAs, and each of these had disease of the ipsilateral external or common carotid arteries, or an abnormality of the internal carotid artery stump. The finding that the nonoccluded group yielded four strokes on follow up compared to a single stroke (contralateral after refusing surgery) in the occluded group is notable. The higher incidence of strokes may be related to the fact that in the nonoccluded group the aetiology of the symptoms was not uniformly due to carotid artery disease. The possibility exists that correctly managed patients with symptoms due to total carotid occlusion may be at a lesser risk for stroke than those with similar symptoms due to diseases other than occlusion of the internal carotid artery.

The importance of the remaining external carotid circulation in patients with internal carotid artery occlusion is highlighted by the four patients in our series who developed subsequent ipsilateral focal symptoms. In each of these cases the external carotid disease presented was documented to be the source of the symptoms and responded promptly to external carotid endarterectomy. The importance of external carotid endarterectomy has previously been identified by other authors.11,13,14 Occlusion of the internal carotid artery is almost invariably associated with stenotic disease of the origin of the external branches of the carotid artery.12 Only if symptoms persist after such attention to the external carotid origin, provided that the contralateral internal carotid is free of disease, would we consider extracranial to intracranial bypass surgery.

The importance of the contralateral carotid artery in patients with carotid artery occlusion has been stressed by Riles et al.17 This is particularly pertinent when both hemispheres can be demonstrated by noninvasive testing or angiography to be supplied by the single remaining patent diseased internal carotid artery.

In the Joint Study of extracranial arterial occlusion18 those patients surviving contralateral endarterectomy had a markedly reduced incidence of stroke in the follow up period when compared to medically treated patients. The operative complications of such surgery have progressively improved and many authors report excellent immediate and long term results with contralateral endarterectomy in patients with occlusion.17 In our study, on evaluation of the contralateral carotid artery 14 were normal and 6 had less than 50% stenosis. These cases all remained asymptomatic on follow up. Eleven patients had contralateral stenosis of great-
er than 50%, and two had contralateral internal carotid occlusion. Of importance is that all the patients who continued to be symptomatic after the initial event had >50% contralateral stenosis or contralateral occlusion. Of these 13 cases 9 underwent surgery, 1 died of stroke after refusing surgery, 1 had a TIA at the time of occluding the contralateral artery, and only two remained asymptomatic.

From these data we would suggest that in patients suspected of having an internal carotid occlusion, specific noninvasive tests and angiography are important to evaluate the status of the ipsilateral common, external and residual stump of the internal carotid artery, as well as the status of the contralateral internal carotid artery. In the absence of disease of these arteries we have demonstrated a good neurological prognosis in this series. In the presence of ipsilateral external carotid or contralateral internal disease we have demonstrated a marked reduction of focal neurological events after appropriate surgery. Similar results have been reported by other authors. 11, 13–16

We believe that surgical intervention in 9 of the 13 patients demonstrating severe contralateral internal and ipsilateral external disease contributed to the good long term results achieved in our series. Nevertheless, we concede that the exclusion of some patients medically unfit for angiography may have introduced a favourable bias in our results particularly in terms of associated coronary artery morbidity and mortality. The absence of a common aetiology of the symptoms of the nonoccluded group may account for the higher stroke rate in that group. A remarkably benign course has been shown after 18 month mean follow up in our group of patients with total carotid artery occlusion. Although our series is too small to draw definitive conclusions, these results support the mounting of a larger controlled series to establish:

a. The true role of such surgery in patients with total occlusion of the internal carotid artery.

b. Whether such a group of patients has a better prognosis than patients presenting with similar symptoms and a patent carotid artery.

Acknowledgment

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References

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