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Recognising Stroke Prone Patients with a Poor Collateral Circulation*

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The prognosis in carotid disease is extremely variable and is influenced by the availability of collateral circulation. This study investigates the possibility of recognising patients with a poor collateral potential by using non-invasive tests.

Preoperative OPG and EEG were compared with intraoperative EEG during test clamping in 208 carotid endarterectomies. Clamping ischaemia occurred in 29 patients (14%). Preoperative EEG had a sensitivity of 62% and a specificity of 82%. OPG showed a sensitivity of 96% and a specificity of 54%. Combined OPG and EEG resulted in a sensitivity of 93% and a specificity of 73%.

Both tests are safe and easy to perform and interpret. These techniques can be used to identify those patients with carotid stenosis who have an increased risk of stroke due to a poor collateral circulation and may help to refine the indications for carotid endarterectomy.

Key Words: Stroke; Collateral Circulation; OPG; EEG.

Introduction

"The prognosis in carotid disease is extremely variable, depending presumably on the availability of collateral circulation through the many potential channels." This statement of C. Miller Fisher[1] reflects the thoughts that form the basis of the present study. Progression of a carotid stenosis to occlusion may occur without symptoms, may lead to transient symptoms or result in catastrophic neurological deficit, depending on the availability of collateral pathways. Assessment of the risk of a carotid stenosis should therefore include an investigation of the collateral potential. Patients with a very poor collateral potential show signs of cerebral ischemia on EEG during intraoperative carotid test clamping.

This study investigates whether it is possible to recognise these patients preoperatively by two non-invasive tests: EEG and ocular-pneumoplethysmography (OPG), both with carotid compression.

If so, this may help to identify those patients with carotid stenosis who have an increased risk of stroke and who may therefore benefit from carotid endarterectomy.

Materials and Methods

The preoperative EEG was performed with a 21 lead system and a 21 channel recording on awake patients in the supine position. Carotid compression was performed low in the neck to avoid stimulation of the baroreceptors or dislodging of atheroma. Right and left sides were compressed alternately, while a photoelectric pulse detector on the earlobe served to monitor the efficiency of the compression. Carotid compression was maintained for at least 30 s but was stopped when signs of cerebral ischaemia were noted.

OPG measures left and right ophthalmic artery pressures simultaneously. Its most common application is for detection of haemodynamically significant or pressure reducing lesions. Ophthalmic artery pressure measure-

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ment during carotid compression provides information on the collateral circulation. The pressure on the side of compression is called the Collateral Ophthalmic Artery Pressure (COAP).[2]

The two preoperative tests were compared to 4 channel intraoperative EEG recording during carotid test clamping.

Carotid endarterectomy was performed under general anaesthesia while the blood pressure was maintained at the preoperative level. Heparin was administered systemically before the common and external carotid arteries were clamped. The circulation was restored whenever EEG changes such as cortical slowing or lateralising were noted by the neurophysiologist. Otherwise, testclamping was maintained for 3 min. A shunt was inserted only in patients with intraoperative EEG changes during clamping.

Preoperative OPG and EEG were compared to intraoperative EEG in 208 patients; 156 male, 52 female (mean age 63 years). Indications for surgery were TIA (69 cases), completed stroke (46), nonhemispheric symptoms (35) and haemodynamically significant asymptomatic stenoses with poor collateral potential, mostly in cases of multivessel disease and/or the need for coronary bypass (58 cases). None of the operations was performed for acute stroke. With rare exceptions all lesions on the operated side were haemodynamically significant; according to our criteria this means a transverse diameter stenosis of greater than 65%.

Results

Intraoperative signs of insufficient collateral circulation were seen during test clamping in 29 of 208 patients (14%), who were then shunted. Preoperative compression EEG correctly identified 18 of these 29 patients, giving a sensitivity of 62% and 147 of the 179 patients who did tolerate clamping, giving a specificity of 82% (Table 1).

The COAP as determined preoperatively by carotid compression and OPG was indicative of a good collateral potential if it was 60 mmHg or higher, whereas pressures less than 60 mmHg were considered to represent a poor collateral circulation.

The COAP was less than 60 mmHg in 28 of the 29 patients with intraoperative EEG changes (sensitivity 96%) and was 60 mmHg or more in 97 of the 179 patients with normal intraoperative EEG (specificity 54%) (Table 2).

By progressively lowering the COAP threshold from 60 mmHg to a figure of 20 mmHg, the specificity of this test can be increased to 88% at the cost of a decreased sensitivity of 48%.

This dynamic interrelationship is shown graphically as a ROC analysis in Fig. 1.

Finally, both preoperative tests were combined and compared with the intraoperative findings (Table 3, Fig. 2).

The best results were obtained with a COAP threshold of 30 mmHg. With either a positive preoperative EEG or a COAP less than 30 mmHg, the sensitivity was 93% (27 of 29 cases with intraoperative positive EEG). The corresponding specificity was 73% (131 of 179 cases with intraoperative negative EEG).

No complications occurred from carotid compression.

Table 1. Preoperative EEG with carotid compression compared to intraoperative EEG during carotid clamping in 208 patients

		Intraoperative EEG		
		+	−	Total
Preoperative EEG	+	18	32	50
	−	11	147	158
Total		29	179	208

Sensitivity 18/29 = 62%, specificity 147/179 = 82%.

Table 2. Sensitivity and specificity of preoperative OPG as compared to intraoperative EEG for various COAP thresholds

COAP threshold (mmHg)	OPG sensitivity (%)	specificity (%)
60	96	54
50	79	65
40	76	74
30	65	85
20	48	88

Discussion

It is widely acepted that the collateral potential is one of the factors that influence the outcome of progressive carotid obstruction. The main collateral pathway is the circle of Willis, which is not a complete circle in about 50% of all cases. The anterior and posterior communicating arteries may be stringlike or even completely absent. Autopsy studies show that these patients have a higher

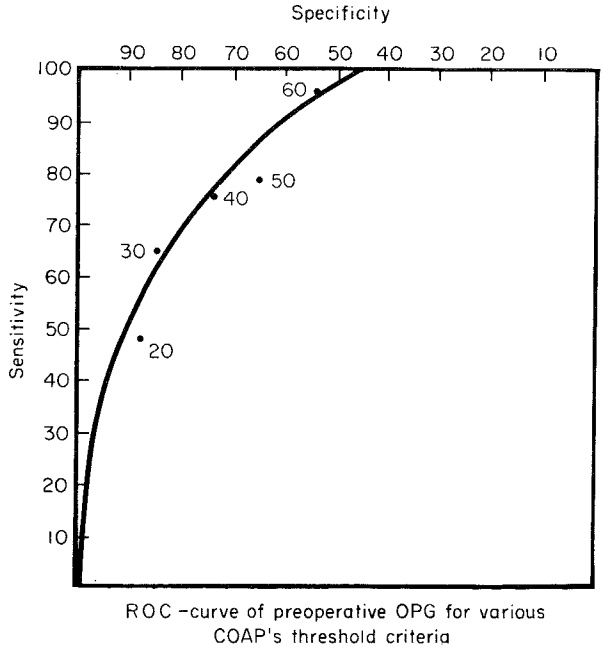

Fig. 1. ROC curve of preoperative OPG as compared to intraoperative EEG for various COAP thresholds.

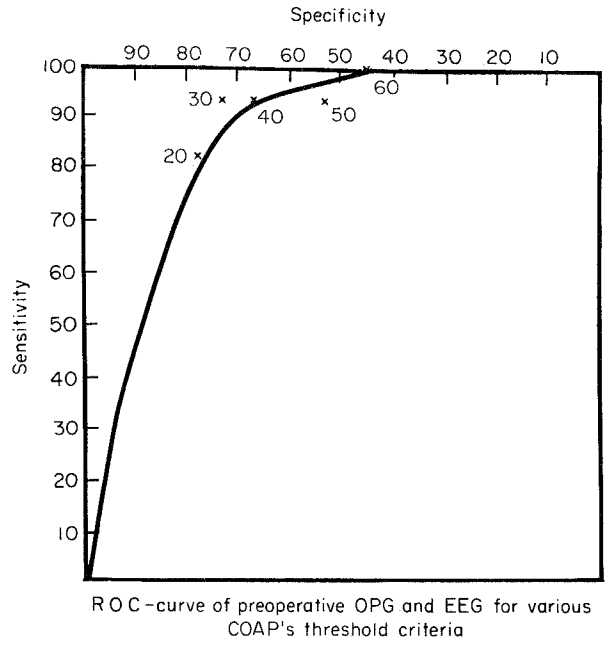

Fig. 2. ROC curve of combined preoperative OPG and EEG as compared to intraoperative EEG for various COAP thresholds.

incidence of cerebral infarction compared to those with a normal circle.[3]

One may wonder whether dilatation of several component parts of the circle of Willis takes place during the progression of extracranial stenosis. It has been shown to occur in animal models following carotid ligation.[4] However, one would prefer this to happen before the moment of occlusion. Afterwards it might be too late because cerebral ischaemia could be present. In a prospective study using OPG, we discovered that the COAP does not increase with the progression of carotid stenosis.[5] The more extensive the disease the greater the chance that carotid compression or clamping will not be tolerated or at least will result in a poor COAP.[2]

Table 3. Sensitivity and specificity of combined preoperative OPG and EEG as compared to intraoperative EEG for various COAP threshold

COAP threshold (mmHg)	Combined OPG and EEG	
	sensitivity (%)	specificity (%)
60	100	45
50	93	53
40	93	67
30	93	73
20	82	78

Compensation for carotid occlusion may take place by an increase in flow in the other vessels. Roberts[6] showed that there is a considerable variation in flow change in different individuals due to differences in the circle of Willis. The same observations have been made by neurosurgeons using gradual carotid occlusion with the Silverstone clamp.[7]

Ischaemic phenomena as a result of carotid occlusion more often occur in older patients than in younger ones.[8] This may be due to more extensive extracranial obstructive disease in older patients. Moreover collateral vessels lose their capacity to dilate with ageing because of the atrophy of elastic tissue and other degenerative changes.

Whether adaptation of the circle of Willis can take place is an individual variable. Carotid ligation for aneurysms results in ischaemic complications in 30% of cases.[8] The same observations have been made in carotid resection for carcinoma of the neck. In these patients, OPG has been used to assess the tolerance to interruption of the carotid circulation.[9]

Some clinical information is available from prospective studies on symptomatology when stenosis progresses to occlusion. Roederer[10] found in a total of 10 such cases 3 strokes and 1 TIA, while 6 occlusions occurred without symptoms.

Chambers[11] documented 17 carotid occlusions, of which 8 resulted in no symptoms, 7 gave TIAs and 2 were followed by a stroke.

It has been assumed that the risk of late ischaemic

symptoms on the side of carotid occlusion is low. Several recent studies, however, have shown that it is considerable and comparable to the risk of stroke in patients suffering from TIAs i.e. around 5% annually,[12,13] even in patients who present themselves with asymptomatic occlusion.[14] There is accumulating evidence that these symptoms occur when the collateral flow through the circle of Willis to the area of the brain above the occlusion is insufficient. These patients have a maximum vasodilatation and have lost their ability to respond to periods of hypotension with vasodilatation.[15] The patients at risk of stroke can be selected by transcranial Doppler with CO_2 augmentation.[16] The aim of our present study is similar. A poorly functioning collateral circulation is probably a risk factor for stroke. Intraoperative EEG changes during test clamping indicate a poor collateral potential and OPG and EEG offer ways to predict whether carotid occlusion will be tolerated.

For this application one prefers to have a highly sensitive noninvasive test with a reasonable specificity. EEG alone is not sensitive enough. OPG with a threshold of 60 mmHg offers a high sensitivity at the cost of an unacceptable specificity. Although the choice is rather arbitrary, the best result of both tests combined is obtained with a COAP threshold of 30 mmHg, giving a sensitivity of 93% and a specificity of 73%.

The final proof of the value of these tests has to come from a prospective follow-up study on patients with high grade stenosis who are treated conservatively.

When patients with a poor collateral potential develop a stroke, it will be clear that these simple noninvasive tests have a great value in refining the indications for carotid surgery.

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