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Internal Carotid Artery Stump Pressures during Regional Anesthesia

James W. Sublett, M.D., (MAJ, MC)* Anthony B. Seidenberg, M.D. (MAJ, MC)* ROBERT W. HOBSON, II, M.D. (LTC, MC)†

In recent years there has been controversy concerning the appropriate anesthetic management of patients undergoing carotid endarterectomy. It has been suggested that general anesthesia is superior to regional techniques.1.2 The effects of hypercarbia versus hypocarbia, induced hypertension, and the effect of general anesthetics on cerebral oxygen consumption (CMR₀) have been documented3-9; however, we are unaware of similar studies conducted in the awake patient under regional anesthesia.

Recent studies have indicated that during clamping of the internal carotid artery, the blood pressure cephalad to the clamp is representative of blood pressure in the proximal parts of the middle and anterior cerebral arteries of the same side.3 There is also evidence indicating a linear relationship between internal carotid artery occlusion pressures (stump pressure) and regional cerebral blood flow. Boysen and associates have also reported that a stump pressure below 60 torr increases the risk of cerebrovascular in-

Address reprint requests to: James W. Sublett, M.D. (MAJ, MC), Anesthesia and Operative Service, Walter Reed Army Medical Center, Washington, D.C. 20012.

sufficiency during the period of internal carotid artery occlusion during general anesthesia.3

The purpose of the present study was to correlate stump pressures during regional anesthesia with clinical signs of cerebrovascular insufficiency in the awake patient. We also measured Pacos, Paos and mean systemic blood pressures in an attempt to determine whether adequate oxygenation, ventilation and blood pressure control could be maintained during regional anesthesia.

MATERIAL AND METHOD

Subjects of the study were 18 patients undergoing carotid endarterectomy under regional anesthesia. Four patients had bilateral staged procedures and were studied on each occasion. Indications for operation were transient ischemic attacks, previous cerebrovascular accident with recurrent transient symptoms, or an abnormal carotid arteriogram obtained prior to undergoing aortoiliac reconstructive surgery. Preoperative arteriograms to rule out a stenotic lesion in the distal intracranial circulation were performed in all patients. The mean age of the patients was 59 years (45-77).

Anesthetic management consisted of a superficial and deep cervical plexus block with 1.5 per cent mepivacaine or 1.5 per cent lidocaine with 1:200,000 epinephrine, using the technique described by Moore.10 The block was supplemented by local infiltration

^{*} Anesthesia Resident.

[†] Vascular Fellow.

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TABLE 1. Gloup 1, Stamp 1. Comment										
	Stump Pressure (torr)	Control Mean Arterial Blood Pressure* (torr)	Operative Mean Arterial BBlood Pressure* (torr)	Pa _{o,} (torr)	Pa _{co} , (torr)	Clinical Response ICA Occlusion				
Patient A	30	(120/60) 80	(110/75) 85	135	41.6	None				
Patient B	25	(120/70) 85	(115/70) 85	71	31.8	None				
Patient C	35	(120/60) 80	(180/90) 120	209	34.0	None				
Patient D	30	(135/90) 105	(160/110) 125	1-1-1	41.6	None				
Patient E	25	(120/70) 85	(190/90) 125	134	34.7	None				
Patient F	25 30	(140/80) 100 (130/80) 95	(145/90) 100 (130/65) 85	88 145	38.1 29.1	None None				
Mean ± SD	28.5 ± 3.7	90 ± 10	105 ± 19.3	132 ± 44.5	35.8 ± 4.8	None None				

^{*}Cuff systolic and diastolic pressures are given in parentheses.

with 0.5 per cent lidocaine as necessary Premedication consisted of either diazepam (10 mg, im) or Innovar (1-2 ml, im) and glycopyrrolate (0.2-0.3 mg, im). Diazepam was used for sedation intraoperatively as needed.

In two patients mean arterial blood pressure was obtained from a catheter inserted into a radial artery and transduced with a Statham transducer to an Electronics for Medicine three-channel recorder. In 16 patients mean pressure was calculated from values obtained from a standard blood pressure cuff (diastolic pressure plus one third pulse pressure). Arterial samples for bloodgas analysis were drawn on arrival in the operating room, following which patients were given oxygen via nasal prongs at flow rates of 3 l/min. Arterial samples were again taken at 15-minute intervals during the procedure, and when stump pressures were measured. Pao, and Paco, were determined using an Instrumentation Laboratories 313 blood-gas analyzer.

A 4-minute period of test clamping was used prior to performing carotid arteriotomy. During this period, stump pressures were measured from a 20-gauge needle inserted above the internal-carotid-artery-occlusion clamp, using a Statham transducer and a Sanborn Model 296 dual-channel recorder.

The patient's clinical status was simultaneously assessed for signs of cerebrovascular insufficiency by asking him to move all four extremities, to perform simple mathematics, i.e., counting, serial sevens, and to answer questions concerning time and place. If the patient developed signs of cerebrovascular insufficiency, the operation was postponed for approximately two days, at which time operation was performed under general anesthesia with use of an internal shunt. Stump pressures were not used as a guide to performing endarterectomy with an internal shunt.

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RESULTS

The seven patients whose stump pressures were less than 50 torr are listed in table 1. None of these patients developed clinical symptoms of cerebrovascular insufficiency intraoperatively or postoperatively. The mean Pa_{O_2} of this group was 132 torr and the mean Pa_{O_2} of this group was 132 torr and the mean Pa_{O_2} of torr. Systemic blood pressure in this group increased from a mean control value of 90 torr to 105 torr intraoperatively. Pa_{O_2} decreased intraoperatively in one patient secondary to intraoperative pulmonary edema. Postoperatively, it was found that the patient had suffered silent myocardial infarction in the three months prior to surgery.

TABLE 2. Group II, Stump Pressures Greater Than 50 Torr

	Stump Pressure (torr)	Control Mean Arterial Blood Pressure* (torr)	Operative Mean Arterial Blood Pressure* (torr)	Palor (torr)	Pacu, (torr)	Clinical Response to ICA Occlusion
Patient A	75	(120/80) 95	(130/75) 95	140	38.2	Hemiplegia, slurring of speech, unresponsive
Patient B	75	140	130	135	31.8	Hemiplegia, slurring of speech
Patient C	85	(135/80) 98	(125/70) 87	195	35.4	None
Patient D	65	(150/85) 107	(180/100) 126	153	33.9	None
Patient E	65	(130/90) 105	(130/90) 105	89	33.4	None
Patient F	60	(120/80) 93	(120/70) 86	so	43.6	None
Patient G	50	(160/90) 113	(140/100) 113	78	39.8	None; control Pa _{0z} = 57 torr
	60	(120/70) 86	(130/70) 90	107	38.1	None
Patient H	75	(170/60) 96	(195/85) 130	76	47.5	None; control Pa ₀ , = 55 torr
	52.5	(180/100) 133	(200/110) 150	89	41.3	None
Patient I	75	(160/100) 120	(150/120) 130	148	37.3	None
Patient J	65	(200/100) 130	(150/90) 110	201	33.4	None
Patient K	85	(180/90) 120	(140/90) 105	98	38.8	None
Patient L	60	100	95	203	33.6	None
Patient M	50	(125/80) 95	(150/85) 105	77	41.6	None
MEAN SD	66 ± 11.4	108 ± 16.4	112 ± 19.6	125 ± 49.9	37 ± 4.4	

^{*}Cuff systolic and diastolic pressures are given in parentheses.

Two patients of the 12 patients with stump pressures greater than 50 torr (table 2) developed clinical signs of cerebrovascular insufficiency, consisting of slurred speech, hemiplegia, and, in one patient, complete unresponsiveness. These changes were transient in one patient, but the other patient developed persistent hemiparesis. In both cases stump pressures were greater than 70 torr. There was no associated hypercarbia or hypocarbia, and the mean blood pressure was not appreciably changed from control levels in these two patients.

DISCUSSION

While one cannot contest the relationship of stump pressures to regional cerebral blood flow and Pa_{Co.} previously determined during

general anesthesia, our data indicate that in the awake patient stump pressure is not a reliable guide for predicting the need for an internal shunt during the period of occlusion of the internal carotid artery. Boysen and associates3 have indicated that cerebrovascular symptoms during occlusion of the internal carotid artery are more likely to be the result of embolic occlusions in the distal cerebral circulation arising from the stenotic lesion in the internal carotid artery than from occlusion of the artery itself. This would explain the lack of correlation between stump pressures and symptomatology of cerebrovascular insufficiency seen in our patients. In the two patients who developed clinical symptoms. these appeared within 2 minutes following occlusion of the internal carotid artery. In Patient A, there was no improvement with

unclamping of the artery; he continues to have slight residual hemiparesis. Patient B, however, showed marked improvement with unclamping, and all signs of cerebrovascular insufficiency disappeared within three hours. While the clinical course of Patient A is compatible with an embolic phenomenon, that of Patient B cannot be as readily explained in this manner. We conclude that clinical symptoms of cerebrovascular insufficiency are of varied etiology and that their occurrence may not be reliably predicted by stump pressure.

The stump pressures in Group I are lower than the normally expected 2:1 ratio between mean arterial systemic blood pressure and stump pressure. The explanation for these findings remains unclear, although this may be related to the awake state. Our data are comparable to stump pressure measurements found during regional anesthesia by Moore and Hall.¹¹ However, these investigators were able to relate a stump pressure of 25 torr to adequacy of collateral cerebral circulation, which was not confirmed by our study.

Many investigators have indicated that general anesthesia should be employed as the technique of choice for carotid endarterectomy. 12 The reasons include: 1) a decrease in CMR₀, with general anesthesia: 2) improved ability to manipulate regional cerebral blood flow via changes in Pa_{CO}, and systemic blood pressure. We feel that the mild hypocarbia and the slight increase in mean blood pressure seen in our patients indicate that some of the disadvantages previously attributed to regional anesthesia do not exist. This, and the obvious advantage of being able to evaluate the awake patient's neurologic status, have led us to believe that

there remains a place for regional anesthesia in the anesthetic management of patients undergoing carotid endarterectomy.

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