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動力學之追蹤研究

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主持人：高明見　　執行機構及單位名稱：台大醫學院外科

' 八十六年度及以前的一般國科會專題計畫(不含產學合作研究計畫)亦可選擇適用，惟較特殊的計畫如國科會規劃案等，請先洽得國科會各學術處同意。

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Changes in hemodynamics of the carotid and middle cerebral arteries before and after endoscopic sympathectomy in patients with palmar hyperhidrosis: preliminary results

JIANN-SHING JENG, M.D., PING-KEUNG YIP, M.D., SHENG-JEAN HUANG, M.D., AND MING-CHIEN KAO, M.D., D.M.SC.

Department of Neurology and Division of Neurosurgery, Department of Surgery, National Taiwan University Hospital, Taipei, Taiwan

Object. The purpose of this study was to analyze the change in carotid and middle cerebral artery (MCA) hemodynamics before and after endoscopic upper thoracic sympathectomy in patients with palmar hyperhidrosis (PH).

Methods. Sixty-eight patients with PH (35 males and 33 females) for whom the average age was 24.5 ± 10.7 years (± standard deviation) were recruited into this study. These patients all underwent routine upper T-2 sympathectomy to treat their PH. Ultrasonography studies of the carotid arteries (CAs) and MCA were obtained in each patient before and after T-2 sympathectomy. The blood flow volume, flow velocity, and resistivity index (RI) in the bilateral common CAs (CCAs), internal CAs (ICAs), and external CAs (ECAs) were evaluated using duplex ultrasonography. The systolic peak velocity, mean velocity, diastolic peak velocity, pulsatility index, and RI of the bilateral MCAs were evaluated using transcranial Doppler ultrasonography. Blood pressure and heart rate were also recorded during this study. The Student paired t-test was used to analyze the differences between studies before and after bilateral T-2 sympathectomy. There was a significant reduction in diastolic pressure after T-2 sympathectomy ($p = 0.003$), but not in systolic pressure or heart rate. The vessel diameter was increased after sympathectomy in the left CAs and right CCA. The T-2 sympathectomy led to significant elevation of blood flow volume and RI in the left CCA, ICA, and ECA ($p < 0.05$). The authors found significant increases in maximum flow velocity and RI in the left MCA ($p < 0.05$).

Conclusions. Patients who underwent T-2 sympathectomy demonstrated a significant increase in blood flow volume and flow velocities of the CAs and MCA, especially on the left side. Asymmetry of sympathetic influence on the hemodynamics of the CAs and MCA was noted. The usefulness of sympathectomy for the treatment of ischemic cardiovascular and cerebrovascular disease deserves further investigation.

KEY WORDS • **carotid artery** • **middle cerebral artery** • **hyperhidrosis** • **sympathectomy** • **ultrasound**

NUMEROUS studies have demonstrated the influence of the sympathetic nervous system on arterial blood flow. Stimulation or inhibition of the sympathetic nerves may affect both the small and the large arteries. Findings in animal studies have indicated that electrical or chemical stimulation of the sympathetic nervous system may induce cerebral vasoconstriction,[6] decrease the ipsilateral cerebral blood flow (CBF),[13,26,28] and significantly reduce arterial stiffness and distensibility.[9,11] In contrast, sympathetic denervation attenuates the development of hypertrophy and the distensibility in the pial arterioles of stroke-prone spontaneously hypertensive rats.[3] However, distensibility is markedly increased in the large arteries of sympathectomized rats.[27]

A few studies in humans indicate that an increase in sympathetic influence may lead to a reduction of radial artery distensibility.[5,12] For patients with palmar hyperhidrosis (PH), endoscopically guided transthoracic T-2 sympathectomy can increase palmar skin perfusion and temperature, reduce the amplitude of sympathetic skin response, and concomitantly reduce craniofacial sudomotor activity.[20,22–25] Thoracic sympathectomy may reduce the frequency of angina attacks, raise parasympathetic activity, and lower sympathetic activity.[34,37] Direct stimulation of the superior cervical sympathetic nerve trunk increases the blood flow velocity of the middle cerebral artery (MCA),[36] whereas blockade of the stellate ganglion increases the ipsilateral CBF.[35] Since we began using this minimally invasive procedure to treat a large number of patients with palmar and craniofacial hyperhidrosis, we have obtained very satisfactory results. We investigate in this paper the influence of the upper thoracic sympathetic chain on the blood flow volume (BFV) and flow velocity of the carotid arteries (CAs) and MCA in patients with PH who have undergone sympathectomy in which noninvasive sonographic techniques were used.

TABLE 1

Blood pressure and heart rate before and after T-2 sympathectomy in 68 patients with PH

Factor	Preop (mean ± SD)	Postop (mean ± SD)	Difference (mean ± SED)
systolic blood pressure	111.0 ± 14.3	107.9 ± 12.9	−3.1 ± 1.6*
diastolic blood pressure	72.0 ± 13.5	67.5 ± 12.7	−4.5 ± 1.4†
heart rate	72.1 ± 9.7	70.0 ± 9.8	−2.1 ± 1.3

* $p = 0.06$.
† $p < 0.05$.

Clinical Material and Methods

Study Participants

Between January and July 1997, 68 patients with PH (35 males and 33 females) whose average age was 24.5 ± 10.7 years (± standard deviation [SD]) were recruited for this study. Patients were excluded for the following reasons: if they were receiving any drug that might affect autonomic function directly or indirectly, or if they had ischemic cardiac or cerebral diseases. This research was approved by the National Science Council, and all patients gave informed consent for the operation and the ultrasonography study.

Thoracic Sympathectomy for Patients With PH

The technique of endoscopic upper thoracic sympathectomy has been well developed by Kao and colleagues[20,22,24,25] and is described in detail elsewhere. Since 1991, more than 200 patients with PH have undergone this operation at our hospital each year. Almost all patients obtained satisfactory results with minimal morbidity.[21] The sympathectomy technique is briefly described as follows: the patients with PH received endotracheally induced general anesthesia and were placed supine with both arms in abduction. Transthoracic video-guided endoscopic ablation of the T-2 sympathetic segment was performed on each side successively in a one-stage operation. The operative procedure was monitored along with palmar skin perfusion by using a laser Doppler flowmeter, and palmar skin temperature was determined using the skin probe of a telethermometer (model 43 TA; Yellow Springs Instrument Co., Yellow Springs, Ohio). A significant increase in the skin perfusion and then the skin temperature (usually 3°C) indicated a successful sympathectomy that would lead to sufficient and long-lasting relief of PH. Both sides of the sympathectomy were accomplished within 30 minutes.

Ultrasonography Studies

Each patient underwent an ultrasonography study 1 day before the operation and 2 to 4 weeks postoperatively. This study included duplex ultrasound of extracranial CAs and color-coded transcranial Doppler (TCD) ultrasonography of the intracranial MCA bilaterally. A Diasonic Gateway series workstation was used for measurement. A 7.5-MHz B-mode imaging transducer, combined with a 5-MHz pulsed-Doppler transducer, was used for measurement of the CAs. The diameter of the vessel lumen, peak systolic velocity, end-diastolic velocity, and resistivity ndex (RI) were obtained in the common CA (CCA), internal CA (ICA), and external CA (ECA) bilaterally. The insonation angle of the sampling vessels was fixed at 60° to avoid the effect of varied angles on velocity. The BFV was automatically calculated as BFV = (time average flow velocity) × (cross-sectional area of the vessel lumen).

A 2.0-MHz real-time and pulse-Doppler transducer was used for the TCD study. The proximal segment of MCA was insonated at a depth of 50 to 55 mm through the posterior temporal window.[1,4] The systolic peak velocity, mean velocity, diastolic peak velocity, pulsatility index (PI), and RI were obtained bilaterally. The depth of the sampling MCA for the same patient was identical at different examinations. The average bilateral brachial blood pressure and heart rate were recorded during the ultrasonography study.

Statistical Analysis

We used commercially available software (SPSS for Windows; SPSS, Inc., Chicago, IL) for the statistical analyses. Values are expressed as mean ± SD and mean ± standard error of difference (SED). Student's paired t-test was used for comparison between the flow velocity, BFV, RI, and PI of the bilateral carotid and cerebral arteries before and after T-2 sympathectomy. Differences were considered significant at a probability level of less than 0.05.

Results

We obtained ultrasonography studies before and after surgery in all 68 patients. The reduction of the diastolic blood pressure after sympathectomy was significant (mean difference ± SED, 4.5 ± 1.4 mm Hg; $p = 0.003$), but the changes in the systolic blood pressure and heart rate were less pronounced (Table 1).

The duplex ultrasonography study revealed significant hemodynamic differences in the CAs before and after thoracic sympathectomy. After bilateral T-2 sympathectomy, there was a pronounced increase in the diameter of the CCA bilaterally and of the left ICA ($p < 0.05$). The peak systolic velocity was increased in the left CCA and ICA and in the ECA bilaterally ($p < 0.05$), but the end-diastolic velocity was not. These increases were accompanied by significant changes in RI ($p < 0.01$). Due to the increase in both the diameter and the flow velocity, the BFV in the left CCA, ICA, and ECA ($p < 0.01$), and the right ECA ($p = 0.03$) was significantly elevated (Table 2).

The TCD study showed significantly increased maximum flow velocity (mean ± SED, 7.6 ± 3.4 cm/sec; $p = 0.03$) and increased PI and RI ($p = 0.04$) in the left MCA after sympathectomy. The differences in the mean velocity of the left MCA and the hemodynamic changes in the right MCA after sympathectomy were not significant (Table 3).

Discussion

The cerebral circulation is highly and hierarchically controlled by autoregulatory, metabolic, and neurogenic

TABLE 2

*Hemodynamics of CAs before and after T-2 sympathectomy in 68 patients with PH**

Vessel & Factor	Preop (mean ± SD)	Postop (mean ± SD)	Difference (mean ± SED)
lt CCA			
diameter (mm)	5.77 ± 0.4	5.88 ± 0.36	0.11 ± 0.03†
BFV (ml/min)	340.6 ± 65.4	373.7 ± 33.1	33.1 ± 11†
PSV (cm/sec)	98.0 ± 20.8	105.0 ± 21.1	6.9 ± 2.6‡
EDV (cm/sec)	25.1 ± 6.1	27.8 ± 28.4	2.8 ± 3.5
RI	0.73 ± 0.04	0.75 ± 0.05	0.02 ± 0.01†
lt ICA			
diameter (mm)	4.45 ± 0.38	4.57 ± 0.29	0.12 ± 0.04§
BFV (ml/min)	259.3 ± 49.4	282.9 ± 64.1	23.6 ± 7.8†
PSV (cm/sec)	74.2 ± 17.4	80.8 ± 17.7	6.6 ± 2.4§
EDV (cm/sec)	29.2 ± 7	29.2 ± 8.7	0 ± 0.9
RI	0.6 ± 0.06	0.63 ± 0.06	0.03 ± 0.01†
lt ECA			
diameter (mm)	3.78 ± 0.41	3.84 ± 0.29	0.06 ± 0.05
BFV (ml/min)	112.2 ± 39.5	131.0 ± 34.5	18.7 ± 4.8†
PSV (cm/sec)	70.8 ± 22	84.7 ± 23.4	13.9 ± 2.8†
EDV (cm/sec)	13.4 ± 3.6	14.7 ± 4.3	1.3 ± 0.6‡
RI	0.8 ± 0.04	0.82 ± 0.03	0.02 ± 0.01†
rt CCA			
diameter (mm)	5.79 ± 0.55	6.0 ± 0.57	0.21 ± 0.04†
BFV (ml/min)	365.1 ± 113.8	386.2 ± 85.4	21.1 ± 13.8
PSV (cm/sec)	95.7 ± 31.3	101.9 ± 19.4	6.2 ± 4.1
EDV (cm/sec)	25.2 ± 6.4	22.8 ± 3.9	−2.4 ± 0.8‡
RI	0.74 ± 0.0	0.77 ± 0.05	0.03 ± 0.01†
rt ICA			
diameter (mm)	4.49 ± 0.51	4.56 ± 0.4	0.07 ± 0.04
BFV (ml/min)	273.3 ± 55.5	272.8 ± 63.1	−0.5 ± 6.5
PSV (cm/sec)	77.3 ± 19.4	76.5 ± 17.7	−0.8 ± 2
EDV (cm/sec)	29.3 ± 7.9	28.4 ± 6.6	0.9 ± 1
RI	0.6 ± 0.1	0.62 ± 0.06	0.02 ± 0.01
rt ECA			
diameter (mm)	3.78 ± 0.44	3.84 ± 0.38	0.06 ± 0.05
BFV (ml/min)	127.2 ± 40	138.0 ± 36.1	10.8 ± 5‡
PSV (cm/sec)	79.4 ± 22	88.1 ± 21.2	8.7 ± 2.8†
EDV (cm/sec)	14.9 ± 4.9	15.5 ± 4	0.6 ± 0.7
RI	0.81 ± 0.04	0.82 ± 0.04	0.01 ± 0.01‡

* EDV = end diastolic velocity; PSV = peak systolic velocity.
† $p < 0.005$.
‡ $p < 0.05$.
§ $p < 0.01$.

TABLE 3

Hemodynamics of the MCA before and after T-2 sympathectomy in patients with PH

Factor	Preop (mean ± SD)	Postop (mean ± SD)	Difference (mean ± SED)
lt side (50 patients)			
peak systolic velocity	102.7 ± 29	110.4 ± 29.7	7.6 ± 3.4*
peak diastolic velocity	49.2 ± 17.1	50.5 ± 22.6	1.2 ± 2.3
mean velocity	71.8 ± 21.4	74.6 ± 22.6	2.7 ± 2.6
PI	0.72 ± 0.16	0.77 ± 0.16	0.05 ± 0.02*
RI	0.5 ± 0.09	0.53 ± 0.09	0.02 ± 0.01*
rt side (64 patients)			
peak systolic velocity	101.5 ± 32.7	106.9 ± 26.3	5.4 ± 3.3
peak diastolic velocity	48.5 ± 17.8	48.5 ± 17.8	0 ± 2.1
mean velocity	70.9 ± 24	73.7 ± 19.1	2.8 ± 2.4
PI	0.76 ± 0.13	0.8 ± 0.14	0.03 ± 0.02
RI	0.52 ± 0.06	0.54 ± 0.07	0.02 ± 0.01

* $p < 0.05$.

mechanisms.[16,30,31] Although cerebral vessels are richly innervated, the role of neurogenic regulation of cerebral circulation is undetermined. The walls of the cerebral arteries are densely innervated by nerve fibers, among which sympathetic fibers originating from the superior cervical ganglion and extending to the cerebral arteries through the ICA have been well demonstrated.[10,15,18]

Studies of sympathetic influence on the cranial vessels in humans are rare and the importance and significance of sympathetic effects on cerebral hemodynamics is still controversial.[16] During normotensive conditions, electrical stimulation of sympathetic nerves apparently has little effect on CBF in cats and dogs,[2,17] and only a modest effect in rabbits and monkeys.[17,33] There is evidence that large cerebral arterioles are controlled neurogenically, whereas smaller vessels are controlled chemically.[17] Some studies have revealed that electrical or chemical stimulation of the sympathetic nervous system in animals may induce cerebral vasoconstriction and decrease CBF significantly.[6,13,26,23] Umeyama, et al.,[35] reported that blockade of stellate ganglia would increase CBF in the ICA territory. Wahlgren, et al.,[36] demonstrated an increase in flow velocity in the MCA after superior cervical sympathetic nerve trunk stimulation.

Since 1990 we have treated a large number of patients with PH by means of transthoracic T-2 sympathectomy and have noticed that most patients also experienced concomitant reduction in their craniofacial sweating postoperatively.[20,22,24,25] Subsequently, we have used endoscopic T-2 sympathectomy to treat patients with distressing craniofacial hyperhidrosis and this has been proven to be a simple and effective therapy.[23] Based on this observation, we believe that T-2 sympathectomy might also have a substantial effect on vasomotor activity in the craniofacial region.

However, to our knowledge, there has been no report concerning cerebral hemodynamic change in patients with PH after thoracic sympathectomy. In the present study we used noninvasive sonographic techniques to study the cerebral hemodynamic change in our patients with PH before and after sympathectomy: duplex ultrasound for extracranial arteries and color-coded TCD for intracranial arteries. We found that ablation of the bilateral T-2 ganglia leads to a significant increase in the diameter, flow velocity, and BFV of the CAs, and the left MCA flow velocity. The increase in CA BFV was probably caused by vasodilation and increased flow velocity after sympathectomy.

Anatomically, the upper third or fourth thoracic, stellate, and cervical sympathetic ganglia receive the preganglionic fibers from the upper four thoracic segments of spinal cord and direct the postganglionic fibers to the targets, including the cranial vessels and heart.[7] Although the cerebral arteries are innervated by sympathetic nerve fibers that originate mainly from the superior cervical ganglion, blockade of the stellate ganglion still can increase CBF[37] and removal of a part of the upper thoracic sympathetic chain would induce some changes in vasomotor activity in the cranial vessels.[23] Our study clearly confirmed that limited T-2 sympathectomy for the treatment of PH could result in an increase in the arterial diameter,

flow velocity, and BFV of the CAs, and the systolic peak velocity, RI, and PI of the MCA, especially on the left side.

Asymmetry in the changes in the CA and MCA hemodynamics caused by sympathectomy was also found in this study; the effect was more prominent on the left than on the right side. Lateralization of sympathetic function has been found at the level of the inferior cervical ganglion.[29,38] In addition, based on our observation of a large number of patients with postsympathectomy PH, it is very impressive that more than 90% of the complications of Horner's syndrome occurred in the left eye; nevertheless, all of these patients were treated using the same method, namely transthoracic T-2 sympathectomy. We postulate that the left T-2 segment may send more sympathetic fibers to the craniofacial region than the right segment, so that left T-2 sympathectomy may result in a higher risk of producing Horner's syndrome than right T-2 sympathectomy.[19] The sympathetic innervation of the heart is also asymmetrical.[14] Stimulation of the left stellate ganglion produces a higher incidence of arrhythmia than stimulation of the right.[32] Right/left hemisphere inactivation delivers asymmetrical sympathetic consequences,[14,38] probably because the distribution and density of the sympathetic nerves on the cerebral arterial walls vary at the same ganglion level. The left T-2 ganglion may have greater sympathetic influence on the carotid and cerebral arteries than the right T-2 ganglion.

Open thoracic sympathectomy was performed in patients with hyperhidrosis or angina pectoris in the era of the 1940s to 1960s.[8] This open method constituted major surgery that led to a high complication rate, and it was abandoned until the new technique of transthoracic endoscopic sympathectomy emerged. Because of its simplicity and minimal invasiveness, endoscopic thoracic sympathectomy was recently advocated for treatment of angina pectoris.[34,37] Our study demonstrated that even limited T-2 sympathectomy can increase flow volume and velocity in the cerebrovascular system. It is reasonable to expect that more extensive sympathectomy including the lower portion of the T-1 (to avoid the risk of producing Horner's syndrome) and T-2 segments might induce a more substantial effect on cerebral hemodynamics. Consequently, application of this minimally invasive sympathectomy technique to the treatment of ischemic cardiovascular and cerebrovascular diseases deserves further investigation.

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Address reprint requests to: Ming-Chien Kao, M.D., Division of Neurosurgery, Department of Surgery, National Taiwan University Hospital, No. 7 Chung-Shan South Road, Taipei, Taiwan.