

Case Report

Severe hypertension after stellate ganglion block

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Haemodynamic effects may occur after stellate ganglion block (SGB) arising from autonomic imbalance and local anaesthetic infiltration to barosensitive areas. We report seven patients who developed severe hypertension (systolic arterial pressure >200 mm Hg) after SGB in our pain clinic service. We postulate that diffusion of the local anaesthetic along the carotid sheath may produce vagal blockade causing unopposed sympathetic activity as a result of attenuation of the baroreceptor reflex. We recommend close monitoring of arterial pressure measurement in patients who received SGB.

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Stellate ganglion block (SGB) may be of benefit in some increases in pain and ischemic states. It is associated with some rare life-threatening complications arising from accidental subarachnoid block and intravascular injection:^{1,2} these lead to hypotension in most cases. SGB is also reportedly useful in open-heart surgery by effectively preventing postoperative hypertension.^{3–5} Hypertension after SGB has been documented in very few cases after SGB. We reported a patient who showed severe hypertension after SGB from an unknown cause.⁶ In the current report, we describe a series of seven cases who showed unexpected severe hypertension after SGB over a 14-yr period in our pain clinic service (Table 1).

Case reports

A standardized SGB was performed in all cases using the paratracheal technique with a 25-gauge needle (3.5-cm length) inserted at Chassaignac's tubercle on the sixth cervical vertebra. The aspiration test was repeated many times during injection. The concentration and volume of local anaesthetic were carefully adjusted on an individual basis by each physician. Development of Horner's syndrome (ptosis and myosis) was considered objective evidence of a successful block. Patients were monitored with oscillometric arterial pressure measurement, as and when appropriate. Values of arterial pressure and heart rate were taken from each patient's record retrospectively, and the heart rate recordings in three patients were not available.

Case 1

A 61-yr-old woman was admitted to our hospital because of numbness in the left side of her face. As she did not have any medical history, and appeared to suffer from left facial nerve palsy without any other symptoms, she was diagnosed as having idiopathic facial nerve palsy. The patient was to undergo SGB twice daily: morning and evening. On admission day 10, the systolic/diastolic arterial pressures (SDBP) of the patient increased from 130/78 to 230/140 mm Hg and she complained of a throbbing headache. ECG, neurological examinations, and CT scan of the head revealed no abnormalities. Urinary catecholamines were normal, excluding pheochromocytoma. Notably, we did not use local anaesthetics containing epinephrine in our clinic. Subsequent SGB by lidocaine 1% as a substitute for mepivacaine, also was associated with hypertension. As SDBP thereafter increased at every attempt, SGB was discontinued and oral steroid therapy was commenced.

Case 2

A 72-yr-old woman complained of excruciating headaches for several years. The neurologist diagnosed tension headache. As headache and stiff muscles in both shoulders were relieved by application of local anaesthetics, the patient was introduced to our outpatient clinic. Trigger point block in combination with SGB alleviated her neck stiffness effectively. On the third SGB attempt, SDBP elevated from 140/70 to 245/132 mm Hg. As SDBP did not decrease

Table 1 Patient characteristics and methods of stellate ganglion block. SBP, systolic arterial pressure; DBP, diastolic arterial pressure; HR, heart rate; SGB, stellate ganglion block; NM, not measured

No.	Age (yr)	Gender	Side	Local anaesthetics	No. of previous block	SBP/DBP (HR) (mm Hg, beats min ⁻¹) before SGB	Maximum SBP/DBP (HR) (mm Hg, beats min ⁻¹)	Other complications after SGB	Relevant medications	Previous complications
1	61	F	Left	Mepivacaine 1%, 5 ml	18	130/78 (NM)	230/140 (NM)	Headache	Mecobalamin Adenosine triphosphate	None
2	72	F	Left	Mepivacaine 1%, 5 ml	2	140/70 (54)	245/132 (66)	None	Diazepam Eperizone	None
3	56	F	Right	Mepivacaine 2%, 5 ml	1	140/80 (71)	230/110 (84)	Hoarseness	Predonine Carbamazepine	Behcet's syndrome
4	57	F	Left	Mepivacaine 1%, 5 ml	20	146/80 (NM)	202/110 (NM)	Headache	None	Diabetes mellitus
5	71	F	Left	Mepivacaine 1%, 5 ml	2	135/67 (53)	218/129 (91)	Hoarseness	Imipramine	Moderate hypertension
6	26	F	Left	Mepivacaine 2%, 8 ml	5	140/80 (81)	196/121 (90)	Hoarseness	Buprenorphine Pentazocine	None
7	73	F	Left	Mepivacaine 1%, 5 ml	19	146/74 (NM)	200/100 (NM)	Hoarseness	None	Labile hypertension

even after 1 h of rest, nasal drip with 5 mg nifedipine in divided doses decreased systolic arterial pressure to 140 mm Hg. After vital signs were restored to normal levels, the patient was allowed to go home. Subsequent SGB attempts often elevated her SDBP, and post-SGB hypertension was managed successfully with bed rest.

Case 3

A 56-yr-old woman complained of recurrent headache after a head injury at the age of 7 yr. Besides the head injury, she suffered from a gastric ulcer and uveitis because of Behcet's syndrome. Persistent headache, non-responsive to analgesics, originated from the right temporal region. Neurological abnormalities were not detected in various examinations conducted by the neurosurgeon. She was thus diagnosed as having traumatic right occipital pain. On the second SGB attempt, SDBP increased from 140/80 to 230/110 mm Hg 20 min after SGB. As the patient complained of giddiness, she was allowed to rest for 1 h. General signs stabilized when SDBP recovered to 167/94 mm Hg. From the third to the seventh SGB attempt, SDBP increased slightly from 140/80 to about 160/90 mm Hg. After the eighth SGB attempt, hypertension was alleviated with oral administration of a calcium channel antagonist (nitrendipine, 5 mg), and SGB slightly raised the SDBP from about 120/70 to 140/80 mm Hg. The headache was relieved and the patient was able to enjoy a higher quality of life without analgesics.

Case 4

A 57-yr-old woman with a previous history of diabetes mellitus complained of painful sensations on her left cheek. As neurological examinations did not reveal any abnormalities, she was diagnosed with atypical facial pain. Treatment with SGB was then initiated, and the area of pain gradually narrowed. On the 21st SGB, SDBP of 146/80 increased to

202/110 mm Hg accompanied by headache 10 min after SGB, and continued for about 1 h. The systolic arterial pressure attenuated to levels of 170 mm Hg after 1 h of resting. Subsequent attempts with SGB were done with close monitoring of BP. Symptoms were improved and therapy was terminated subsequently.

Case 5

A 71-yr-old woman with a previous history of diabetes mellitus and hypertension was infected with herpes zoster on her left upper limb (C6 region) 2 months previously. She complained of severe pain in her left upper extremity. Post-herpetic neuralgia was diagnosed. SGB was used as an analgesic therapy. On the third SGB attempt, SDBP gradually increased from 135/76 to 218/129 mm Hg accompanied by an increase in heart rate from 53 to 91 beats min⁻¹ 45 min after SGB. The arterial pressure was normalized with nasal drip of 5 mg nifedipine. The patient had travelled a long distance to the clinic, and was thus admitted to the hospital and thereafter underwent continuous cervical epidural block for 3 weeks.

Case 6

A 26-yr-old woman, with a previous history of left radial fracture at the age of 17 yr, presented swelling of the left forearm since 18 yr of age. Symptoms were not relieved with surgical treatment of lymph-venous anastomosis, and she was diagnosed as suffering from lymph edema in the left upper limb. After continuous cervical epidural block for 3 weeks, pain and edema were dramatically relieved. SGB was substituted for epidural block and repeated on a daily basis. The patient complained of discomfort and hoarseness accompanied by elevated SDBP (196/121 mm Hg) with increased heart rate of 90 beats min⁻¹ after the sixth attempt. Follow-up observation after 1-h resting

revealed normalized SDBP (138/94 mm Hg) and heart rate (82 beats min⁻¹). Hypertension did not occur thereafter.

Case 7

A 73-yr-old woman had a previous history of hypertension. She was diagnosed with post-herpetic neuralgia in the left thorax (T2 and T3 regions). As long-term continuous epidural block to relieve pain was no longer practical, SGB was used in place of continuous epidural block. On the 20th SGB procedure, she complained of throbbing headaches with SDBP elevated to near 200/100 mm Hg. Subsequent procedures increased SDBP, and prophylactic nifedipine was prescribed thereafter to prevent this.

Discussion

Successful SGB is believed to decrease efferent cervical sympathetic outflows. The current report, however, presents a series of cases mostly where systolic arterial pressure paradoxically increased to levels of more than 200 mm Hg after SGB. Elevated SDBP appeared a few minutes after induction of SGB, and persisted for more than 2 h until disappearance of local anaesthetic effects in cases where hypertension was not actively treated. We encountered seven cases in a 14-yr period, indicating an estimated rate of occurrence of 1/2000–1/3000 SGBs.

Local anaesthetic application for SGB affords dissemination of the anaesthetic into adjacent tissues. As such, local anaesthetics might infiltrate the carotid sheath thus causing partial block of the vagus nerve.⁷ Furthermore, as local anaesthetics disseminate in a cephalic direction, blockades of baroreflex-sensitive carotid sinus at the bifurcation of the carotid artery as well as the sinus and glossopharyngeal nerves may eventually be induced. In addition, we used the transverse process of C6 as a landmark for SGB puncture, which is closer to the carotid sinus than the anterolateral border of C7 transverse process. As a result, afferent neural inputs associated with baroreflex might be attenuated enhancing efferent sympathetic output leading to hypertension.^{8,9}

Studies that have examined the spread of local anaesthetic agents (using a higher volume than in our report), have documented several patterns of spread as well as frequent contact with the carotid sheath; however, no hypertension was reported.^{10,11} We need, therefore, an alternate explanation for the hypertension in addition to the anaesthetic effect of carotid sinus reflex. A possibility is that the local anaesthetic spread to the vagus nerve thus causing unopposed cardiac sympathetic stimulation.⁸

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