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No Prophylactic Effect of Early Sympathetic Blockade on Postherpetic Neuralgia

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We propose to determine whether sympathetic blockade given during an acute herpes zoster infection could prevent the development of postherpetic neuralgia. There is no reliable treatment available for postherpetic neuralgia; however, various treatments[1,2] have been used

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TABLE 1. Rating of Pain Relief from Prophylactic Sympathetic Blockade

	Group 1 n (%)	Group 2 n (%)
Good	30 (66.7)	157 (71.8)
Improvement	9 (20.0)	37 (17.1)
PHN	6 (13.3)	24 (11.1)
Total	45	218

PHN = postherpetic neuralgia.

during the acute phase of the infection to prevent its subsequent development. Sympathetic blockade is said to be one of the more effective measures.[3,4] However, as patients may visit pain clinics week, months, or even years following the rash, it is difficult to ascertain whether the postherpetic neuralgia could have been prevented by a sympathetic blockade during the acute phase of the disease. In this study, we analyzed retrospectively the results of 45 patients with herpes zoster for whom sympathetic blockade therapy was initiated prior to the manifestation of cutaneous herpetic eruption and compared them with the results of 218 patients for whom sympathetic blockade therapy was initiated within 10 days after the manifestation of cutaneous herpetic eruption.

PATIENTS AND METHODS

Between 1977 and 1984, 49 patients (21 men/28 women; age range 38–88 yr) received sympathetic blockade before the skin eruption appeared and the diagnosis of herpes zoster was not established (Group 1). Two hundred and sixty-two patients (120 men/142 women; age range 35–86 yr) received a sympathetic blockade treatment within 10 days after manifestation of herpetic cutaneous eruption (Group 2). In Group 1, patients complained of severe continuous paresthesia, shooting, pricking, burning, or formicating pain without any herpetic eruption in the pain area. The prerash pain due to herpes zoster might have been suspected at their first visit, but it was not established until the skin eruption appeared. Herpes zoster was subsequently diagnosed in all these patients. All were treated on an outpatient basis. Sympathetic blockades were performed at least once a day for 4 weeks; no antibiotics, analgesics, or steroid hormones were prescribed, and no ointments were applied to the cutaneous lesion. Cutaneous lesions were cleaned with alcohol swabs, taking care not to break the vesicles. Regardless of the degree of pain, patients were told that sympathetic blockade was absolutely necessary to prevent progression to postherpetic neuralgia. These "rules" were applied to all patients included in this study. A stellate ganglion block (8 ml of 1.0% mepivacaine) was offered to patients suffering pain in the trigeminal nerve or cervical nerve distribution. Epidural sympathetic block (6 ml of 1.0% mepivacaine) was performed in patients suffering pain in the thoracic or lumbar areas, and caudal epidural block was performed for pain in the caudal distribution. In this study, postherpetic neuralgia was categorized by the presence of severe pain or dysesthesia lasting more than 1 yr after the scars became pale and formed the typical pockmarks. In some cases the skin eruption was minimal, with very few vesicles in the acute phase; thus, few scars were observed in the developed condition of postherpetic neuralgia. All data were recorded using a punched-card system. On April 1, 1985, questionnaires were sent to all patients who received sympathetic blockade therapy before March 30, 1984. The questionnaire requested the patient to check the following three points: 1) complete pain relief; 2) persistent discomfort with minimal disturbance of daily activity; and 3) severe pain or dysesthesia. According to their answers, the response to sympathetic blockade therapy is expressed as good (answer 1), improvement (answer 2), and postherpetic neuralgia (answer 3). The chi-square tests were used to assess for statistical significance of the response to sympathetic blockade therapy.

TABLE 2. Age Distribution/Effect of Sympathetic Blockades

	Group 1				Group 2			
Age	No. Patients	Good	Improvement	PHN (%)*	No. Patients	Good	Improvement	PHN (%)*
>80	5	3	1	1 (20)†	24	13	6	5 (20)†
70–79	16	10	4	2 (12.5)	57	37	14	6 (10)
60–69	13	10	1	2 (15.4)	64	50	6	8 (12)
50–59	8	5	2	1 (12.5)	41	32	6	3 (7)
40–49	2	1	1	0 (0)‡	24	18	4	2 (8)
<39	1	1	0	0 (0)‡	8	7	1	0 (0)‡
Total	45	30	9	6 (13.3)	218	157	37	24 (11)

PHN = postherpetic neuralgia.
* Absolute number; % of total patients who had PHN in parentheses.
† Significantly higher compared with the total; $P < 0.01$.
‡ Significantly lower compared with the total; $P < 0.05$.

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TABLE 3. Pain Distribution/Effect of Sympathetic Blockade

Dermatomes	Group 1				Group 2			
	No. Patients	Good	Improvement	PHN	No. Patients	Good	Improvement	PHN
Trigeminal	7	3	2	2	33	18	8	7
Cervical	8	5	2	1	39	26	7	6
Thoracic	17	12	3	2	82	67	11	4
Lumbar	9	7	2	0	44	32	8	4
Caudal	4	3	0	1	20	14	3	3
Total	45	30	9	6	218	157	37	24

PHN = postherpetic neuralgia.

RESULTS

Of the 49 patients in Group 1, 45 patients (91.8%) responded to the questionnaire. Of the 262 patients in Group 2, 218 patients (83.2%) responded. As shown in table 1, there is no significant difference in the results of "good," "improvement," and "postherpetic neuralgia" between Groups 1 and 2. Table 2 shows that the occurrence of postherpetic neuralgia is higher in patients older than 80 yr of age and lower in patients younger than 39 yr in both groups. There appeared to be no correlation between the development of postherpetic neuralgia and other factors, including: pain distribution (table 3); stage of initiation (table 4); or number of sympathetic blockades administered during the 4-week period (table 5).

DISCUSSION

Postherpetic neuralgia occurs in older patients, predominantly in the first branch of the trigeminal area, and is accompanied by severe cutaneous lesions in the early stage.[1,2] Of all patients with herpes zoster, approximately 10% will develop postherpetic neuralgia.[1] Prevention of postherpetic neuralgia may be achieved by different types of treatment, including antiviral agents with continuous compresses of 40% idoxuridine in dimethyl sulfoxide (DMSCO),[5] cytosine arabinoside (ARA-C),[6] steroids,[7] and nerve blocks.[3,4] The relative value of steroids, chemotherapy, and psychotropic drugs is still under discussion,[1,2] but undoubtedly there is basic logic in treating the acute herpetic condition with sympathetic blockade.[2] The primary lesion in herpes zoster is an acute inflammatory state of the posterior root ganglion, and this may cause increased sympathetic vasoconstriction in the affected segments. Blocking this ganglion can interrupt sensory afferent impulses through the sympathetic fibres and ganglia proceeding to the dorsal roots, as well as producing vasodilation. Dan *et al.*[3] reported that 49 out of 67 herpetic patients (73%) in whom sympathetic blockade was initiated between 2 weeks and 3 months after manifestation of skin eruption obtained almost total relief. Milligan *et al.*[4] achieved complete pain relief in six of eight herpetic patients (75%) to whom sympathetic blockades were carried out at an interval of 1 month more than 1 year after the onset of symptoms. In both groups of our present study, sympathetic blockade therapy was performed at even earlier stages than in the studies of Dan *et al.*[3] and Milligan *et al.*[4] Complete pain relief was observed in 66.7% in Group 1 and 71.8% in Group 2. Our results shows a lower percentage than that reported by Dan *et al.*[3] or Milligan *et al.*,[4] although the difference may be insignificant. These facts strongly suggest that the subsequent outcome of herpes zoster is not dependent on whether sympathetic blockade therapy is performed prior to eruption, immediately after eruption, 3 months after eruption, or 1 yr

TABLE 4. Stage of Initiation of Sympathetic Blockade

Period (day)	No. Patients	Good	Improvement	PHN
Group 1				
−6	1	1	0	0
−5	2	0	1	1
−4	8	6	1	1
−3	16	10	4	2
−2	12	9	2	1
−1	6	4	1	1
Group 2				
+1	6	1	2	3
+2	9	2	3	4
+3	8	8	0	0
+4	11	5	4	2
+5	22	16	4	2
+6	30	20	9	1
+7	38	29	2	7
+8	30	22	4	4
+9	22	18	4	0
+10	42	36	5	1

Numbers in "Period" column indicate that the first block was performed that number of dates before (negative) or after (positive) the appearance of skin eruption. The date of appearance of skin eruption is day 1.

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TABLE 5. Total Number of Blocks/Effect of Sympathetic Blockade (over 4-week period)

No. Blocks	Group 1				Group 2			
	No. Patients	Good	Improvement	PHN	No. Patients	Good	Improvement	PHN
>31	1	0	0	1	5	0	1	4
26–30	2	0	1	1	8	0	3	5
21–25	18	12	4	2	87	68	10	9
16–20	11	8	2	1	53	42	7	4
11–15	4	4	0	0	20	15	4	1
6–10	6	3	2	1	22	12	9	1
1–5	3	3	0	0	23	20	3	0
Total	45	30	9	6	218	157	37	24

PHN = postherpetic neuralgia.

after eruption. In each series of patients suffering from postherpetic neuralgia, it is difficult to ascertain whether improvements achieved are a result of treatment received or a natural resolution of the disease process. We conclude that sympathetic blockade, even when performed in the extremely early stage, cannot always prevent the development of postherpetic neuralgia. This does not mean that sympathetic blockade therapy for postherpetic pain should be abandoned. However, we believe that the prophylactic effect of sympathetic blockade therapy in preventing postherpetic neuralgia is overemphasized. Whether it is of any prophylactic value remains to be determined in a large and/or well-controlled series. We suspect that the development of postherpetic neuralgia may be due to a yet-unknown factor, such as a special pain-provoking subtype of the virus.

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Intrathecal Baclofen for Treatment of Tetanus-induced Spasticity

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Clinically, tetanus can be viewed as an intoxication of the central nervous system with a long-acting, strychnine-like poison. The symptoms of tetanus depend on the pharmacodynamics and pharmacokinetics of the tetanus toxin. Spinal convulsions, often impairing respiration, are the immediate threat of tetanus. Complications and death are almost exclusively due to respiratory embarrassment and its consequent cardiovascular depression. Despite apparent progress in the intensive-care therapy of tetanus (*e.g.* artificial respiration, medication with suppressive and neuromuscular blocking drugs), the mortality rate of tetanus has improved very little and still ranges from 40 to 60%.[1] Complications from tetanus include those caused by the disease and those resulting from therapy such as prolonged mechanical ventilation.[2] Although the pathophysiologic problems from tetanus (*i.e.*, spasms and con-

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