

Phenoxybenzamine in the treatment of causalgia

Report of 40 cases

SALIM Y. GHOSTINE, M.D., YOUSSEF G. COMAIR, M.D., DONN M. TURNER, M.D.,
NEAL F. KASSELL, M.D., AND CAMILLE G. AZAR, M.D.

*Central Military Hospital and Berbir Medical Center, Beirut, Lebanon, and Division of Neurosurgery,
Department of Surgery, The University of Iowa Hospitals, Iowa City, Iowa*

✓ Forty consecutive cases of causalgia treated during a 7-year period are presented. The patients ranged in age between 17 and 55 years, and all patients were males who received their nerve injuries from missile or shrapnel wounds. The greater occipital nerve was involved in two cases, median nerve in 10, sciatic nerve in 12, brachial plexus in seven, cauda equina in five, and multiple nerves in four cases. Each patient was treated with phenoxybenzamine, a postsynaptic α_1 -blocker and presynaptic α_2 -blocking agent. The drug was given orally in gradually increasing increments until a maximum daily dose of 40 to 120 mg was reached. Duration of treatment was usually 6 to 8 weeks. Total resolution of pain was achieved in all cases. The follow-up period ranged between 6 months and 6 years. Side effects of phenoxybenzamine were minimal and transient, consisting primarily of mild orthostatic hypotension and ejaculatory problems. We conclude that oral phenoxybenzamine is a simple, safe, and effective treatment of causalgia.

KEY WORDS • causalgia • guanethidine • phenoxybenzamine • sympathectomy • nerve injury

CAUSALGIA refers to a symptom complex observed after partial nerve injury, and consists primarily of severe burning pain associated with sensory, vasomotor, and trophic phenomena, often exacerbated by emotional factors.^{23,27} Effective treatment for this condition has been surgical sympathectomy.^{21,27} This report presents a series of 40 consecutive cases of causalgia secondary to missile or shrapnel wounds. These patients were all treated with phenoxybenzamine, a postsynaptic α_1 -blocker and presynaptic α_2 -blocking agent.¹⁰

Summary of Cases

Patient Population

During the 7-year period, 1975 to 1981, 40 male patients were admitted to the Central Military Hospital and Berbir Medical Center with the diagnosis of causalgia. The criteria used to diagnose this condition were deep burning pain, exacerbated by physical or emotional stimuli, associated with partial nerve injury to a major peripheral nerve. In most cases the distribution of pain followed the cutaneous distribution of the injured peripheral nerve but frequently spread to in-

volve the whole limb. Evaluation of the extent of motor and sensory involvement was almost always difficult to ascertain exactly, since the severe pain, which was markedly aggravated by mere touch, precluded extensive strength testing and careful sensory evaluation. After pain was controlled, however, partial motor paralysis was observed in 70% of cases. These motor deficits had usually resolved by the time of long-term follow-up examination, although some patients had residual motor paresis.

Vasomotor changes, consisting of severe vasodilatation, sweating, and less often vasoconstriction, were present in nearly all cases. Patients with vasodilatory changes occasionally achieved some relief from wrapping the involved limb with a cold wet cloth. Trophic changes were present in only a few patients seen late after their initial injury, and these changes were not considered necessary for the diagnosis of causalgia.

The patients ranged in age between 17 and 55 years (Table 1). All patients received their nerve injuries secondary to high-velocity missile or shrapnel wounds. The greater occipital nerve was affected in two cases, median nerve in 10, sciatic nerve in 12, brachial plexus

TABLE 1
Clinical summary of cases treated with oral phenoxybenzamine

Case No.	Age (Yrs)	Onset of Pain After Injury	Time to Treatment (days)	Time to Complete Relief (days)	Duration of Treatment (wks)	Maximum Daily Dose (mg)	Follow-Up Period	Complications & Treatment
greater occipital nerve injury								
1	40	immediate	3	10	8	80-60	6 yrs	orthostatic hypotension: stockinettes, abdominal girdle
2	55	immediate	5	18	6	80	36 mos	orthostatic hypotension: stockinettes
brachial plexus injury								
3	36	hours	10	2	16	60-80	6 yrs	—
4	24	hours	14	7	6	60	4 yrs	—
5	23	hours	21*	6	7	60	6 mos	—
6	38	3 days	7	10	8†	90	2 yrs	orthostatic hypotension
7	18	12 hrs	14	10	6	70	2 yrs	—
8	22	48 hrs	6	4	6	70	6 mos	—
9	24	hours	7	12	6	90	4 yrs	orthostatic hypotension
median nerve injury								
10	35	1 day	5	7	13	90-120	6 yrs	orthostatic hypotension: stockinettes, abdominal girdle
11	18	24 hrs	7	5	6	60	2 yrs	—
12	35	24 hrs	4	18	8	100	3 yrs	orthostatic hypotension, seminal fluid reduction
13	18	48 hrs	70	6	6	70-60	18 mos	orthostatic hypotension
14	17	immediate	6	4	6	60	6 mos	—
15	41	3 days	14	10	6	90	2 yrs	—
16	28	12 hrs	15	10	7	80	30 mos	—
17	22	12 hrs	14	14	6	70	18 mos	—
18	17	immediate	14	12	10†	90-60	2 yrs	orthostatic hypotension
19	26	2 days	21	10	6	90	3 yrs	—
associated nerve injury								
20‡	37	immediate	14	10	6	90	36 mos	orthostatic hypotension
21§	19	1 day	6	16	8†	120	4 yrs	orthostatic hypotension: stockinettes
22§	23	12 hrs	8	12	6	40	1 yr	—
23§	31	48 hrs	14	9	6	80-60	6 yrs	orthostatic hypotension
cauda equina injury								
24	25	12 hrs	14	6	6	80	18 mos	—
25	35	12 hrs	5	10	8	90	2 yrs	orthostatic hypotension
26	28	48 hrs	14	14	7	120	4 yrs	—
27	18	2 days	15	5	6	70	6 mos	—
28	24	2 hrs	13	7	6	80	2 yrs	orthostatic hypotension
sciatic nerve injury								
29	30	immediate	2	14	6	100	2 yrs	orthostatic hypotension: stockinettes
30	22	12 hrs	7	6	6	60	4 yrs	—
31	26	12 hrs	7	8	6	70	2 yrs	—
32	21	hours	6	12	6	90	5 yrs	—
33	24	12 hrs	7	9	8†	80-60	18 mos	orthostatic hypotension
34	28	immediate	6	7	6	60	8 mos	—
35	22	12 hrs	30*	10	8	90	3 yrs	orthostatic hypotension: stockinettes; seminal fluid reduction
36	43	48 hrs	13	10	6	80	18 mos	seminal fluid reduction
37	19	12 hrs	2	7	6	70	6 mos	—
38	32	2 weeks	60*	14	9	120	2 yrs	orthostatic hypotension: stockinettes, abdominal girdle
39	26	3 days	68*	3	8	100	6 yrs	—
40	38	immediate	6	4	6	60	18 mos	—

* Evidence of trophic changes.

† Recurrence after 6 weeks of uninterrupted therapy.

‡ Median, ulnar, and radial nerve injuries.

§ Median and ulnar nerve injuries.

in seven, cauda equina in five, and associated nerves in four cases (Table 2).

Phenoxybenzamine Treatment

The onset of causalgia most often began several hours

after injury and was progressive in nature (Table 1). Phenoxybenzamine treatment was begun usually within 2 weeks after the onset of causalgia (Table 3). Patients with pain of longer duration were also acceptable candidates for treatment, five of these patients having

Phenoxybenzamine therapy for causalgia

TABLE 2
Distribution of nerve injuries

Nerves Injured	No. of Cases
greater occipital nerve	2
brachial plexus	7
median nerve	10
associated nerves	
median & ulnar	3
median, ulnar, & radial	1
sciatic nerve	12
cauda equina	5
total cases	40

TABLE 3
Duration of pain before institution of phenoxybenzamine therapy

Duration of Pain	Percent of Cases
less than 6 days	20
6 to 13 days	40
2 weeks to 4 weeks	33
more than 4 weeks	8

evidence of trophic changes (Table 1). The starting dose of phenoxybenzamine was 10 mg, orally, every 8 hours (30 mg/day), and the dosage was gradually increased by 10 mg/day every 2 days until there was complete resolution of pain or the occurrence of postural hypotension. Since absorption of phenoxybenzamine from the gastrointestinal tract is incomplete and unpredictable, slight postural hypotension was considered evidence of effective penetration of the drug. Duration of treatment was usually 6 weeks, after which the drug was slowly tapered and then discontinued. Causalgic pain recurred during the tapering schedule in four patients, and the dose was increased to the prior "relief" dose for an additional 2 weeks, after which tapering was again attempted. The follow-up period ranged from 6 months to 6 years (Table 1).

Results of Treatment

Before phenoxybenzamine treatment, all patients had only incomplete relief of causalgic pain at best, with the help of various pain medications including narcotics. Total resolution of causalgic pain was achieved in all cases within 18 days of phenoxybenzamine treatment (Table 1). In some patients, however, relief of the severe burning pain preceded complete relief, leaving a lesser superficial pain. The average total daily dosage necessary for relief was 80 mg, although the maximum dosage required was 120 mg (Table 1). Most patients were treated for 6 weeks, but some received treatment for as long as 3 months (Table 1). At follow-up review, all patients continued to have total resolution of their causalgia, despite previous termination of phenoxybenzamine treatment.

TABLE 4
Complications of oral phenoxybenzamine treatment in 40 cases

Complications	No. of Cases	No. Requiring Treatment
orthostatic hypotension	17	8
seminal fluid reduction	3	0

Complications

Minor complications of phenoxybenzamine were observed which required neither interruption nor cessation of drug treatment. The most frequent complication was orthostatic hypotension as manifested by subjective light-headedness, which occurred in 17 cases. This symptom usually resolved spontaneously at the end of the 1st week of treatment. Persistent orthostatic hypotension necessitated wearing either leg stockinettes or an abdominal girdle for the duration of treatment (Table 4). Ejaculatory problems characterized by reduction of seminal fluid were observed in three patients. These problems also resolved spontaneously following the conclusion of treatment.

Representative Cases

Case 10

This 35-year-old orthopedic surgeon received bullet wounds to the right arm and right knee in 1975. Surgery was performed immediately, revealing a lacerated right humeral artery which required end-to-end anastomosis. At surgery the peripheral nerves of the arm were noted to be intact. One day following surgery, the patient developed progressive deep burning pain in the right forearm and hand in the distribution of the median nerve. The pain became excruciating when the affected area was touched even lightly by cloth, and emotional stress also aggravated his symptoms. Narcotics provided only incomplete relief.

Oral phenoxybenzamine was begun 5 days after the initial injury, and total resolution of pain was achieved 7 days after onset of treatment at a daily dose of 90 to 120 mg. On three occasions prior to conclusion of treatment the drug was unavailable, and the patient's pain promptly returned. In each instance, reinstitution of drug treatment abolished the pain within 24 hours. Phenoxybenzamine caused complaints of light-headedness, and this orthostatic hypotension was relieved by application of stockinettes and an abdominal girdle. Treatment was discontinued permanently after a 3-month course, and there has been no recurrence of symptoms. Orthostatic hypotension resolved spontaneously after termination of phenoxybenzamine treatment. Presently, the patient is continuing his practice of orthopedic surgery free of pain and with full use of the right arm and hand.

Case 3

This 36-year-old security officer sustained a bullet injury to the right supraclavicular area in 1977. Within a few hours after injury, he developed burning and throbbing pain of the right hand in a distribution indicating involvement of all three peripheral nerves. This pain was markedly increased by any sudden noise or emotional upset, and was incompletely relieved by narcotics. Phenoxybenzamine was begun 10 days after injury and prompt relief was obtained in 48 hours at a daily dose of 60 to 80 mg. Again, interruption of treatment secondary to unavailability of the drug immediately produced recurrence of pain, which promptly resolved within 24 hours after reinstatement of treatment. Treatment was stopped permanently after 4 months and the patient has been pain-free since that time. No complications of treatment were reported by this patient.

Case 32

This 21-year-old soldier sustained a bullet injury to the right buttock with damage to the right sciatic nerve. A few hours later he developed severe burning pain in the right foot. An unsuccessful attempt was made to control the pain with wet cloths and narcotics.

Six days after injury, he was begun on progressively increasing doses of phenoxybenzamine. Complete relief of pain was obtained in 12 days at a daily dosage of 90 mg. Duration of treatment was only 6 weeks, since when the patient has been completely pain-free for 5 years.

Discussion

Causalgia-like pain was first reported by Denmark⁴ in 1813, and no additional cases were described until 1864.²³ The term "causalgia" was coined in 1867 by Mitchell²² to describe a symptom complex observed after partial nerve injury characterized by subjective severe burning pain and associated with sensory, vasomotor, and trophic phenomena. Additionally, the pain is aggravated by emotional upset. Its incidence after peripheral nerve injury is estimated at 2% to 8%,^{9,16,30,33,40} and the duration of pain without treatment is variable.^{27,32} A genetic susceptibility is possible,^{35,41} which is supported by evidence that experimental autotomy can be produced only in specific strains of cats and rats.³⁷

It has been suggested that causalgia results when nerve injury promotes the production of an ephapse or short circuitry between somatic afferent fibers and adjacent sympathetic efferent nerve fibers.⁸ The occurrence of such electrical "cross-talk" is supported by evidence from experimentally produced neuromas of ephapses occurring both acutely^{11,12} and chronically^{3,28} between adjacent myelinated fibers. Morphological evidence of axonal apposition between adjacent unmyelinated fibers in neuromas has also been demonstrated.^{2,3} These unmyelinated fibers are usually

separated by Schwann cell cytoplasm or basal cell membrane.²⁵ Because of the conflicting data concerning the recording of ephapses from intermediate-term neuromas (1 to 30 days)^{6,36} and other factors,^{3,7,15} the role of ephapses as the pathophysiological etiology of causalgia has recently been challenged by most investigators.^{5,20,21,26}

An alternative theory explaining the etiology of causalgia suggests a primary role of nerve sprouts which arise from the damaged nerve. These sprouts have been shown to be highly excitable to noradrenaline, an effect reversed by the α -blocking agent, phentolamine, and unaffected by β -blocking agents.^{3,36} The axonal sprouting has been demonstrated to occur early and at high frequency²⁴ following reversible nerve injury characterized by myelin sheath disruption and axonal swelling.²⁴ Total axonal disruption was not a prerequisite for sprouting to occur.^{5,24} Demonstration of axonal uptake and transport of intravenous horseradish peroxidase by injured nerve³⁵ indicates breakdown of the blood-nerve barrier which may favor the penetration of chemical mediators to the axonal sprouts.

It is well established that the sympathetic nervous system is involved in the pathophysiology of causalgia,³⁴ as demonstrated by the success of chemical^{13,14} or surgical sympathectomy^{21,27} in the treatment of this condition. Experimentally, it has been shown that afferent fibers from a neuroma are activated by repetitive stimulation of the paravertebral sympathetic trunk or by intravenous injection of noradrenaline. Such activation is blocked by phentolamine, an α -adrenergic antagonist.⁷ Furthermore, autotomy in rats after experimental neuroma production is prevented by prior administration of guanethidine,³⁷ a ganglionic blocking agent which initially discharges noradrenaline before occupying its storage sites.¹⁰ Guanethidine injected intravenously to perform regional sympathetic blockade has also been shown to relieve causalgia.^{13,14,20}

A more controversial theory regarding the etiology of causalgia involves a possible peripheral alteration of sensory nerves distal to the site of nerve injury.²⁶ It has been reported that regional perfusion of guanethidine below the level of a tourniquet placed distal to a partial lesion of the brachial plexus can relieve causalgic pain.^{14,20} Hypersensitivity to noradrenaline of the peripheral sensory receptors after nerve injury is possible.³⁸

Treatment of causalgia has traditionally consisted of surgical sympathectomy. Leriche^{18,19} first performed sympathectomy for this condition in 1914, and it was later popularized by Spurling in 1930.³¹ Since then many authors have reported high cure rates^{21,29,39} and suggested early operative intervention in order to prevent the occurrence of trophic and vasomotor changes, although such changes are cured even by late sympathectomy. Morbidity of the procedure is low,¹ but it does entail permanent disruption of sympathetic flow to a given limb.

Phenoxybenzamine therapy for causalgia

We elected to treat 40 cases of causalgia with phenoxybenzamine, a postsynaptic α_1 -blocker and presynaptic α_2 -blocker. This drug was chosen as a medical alternative to surgical sympathectomy in an effort to further reduce the minimal morbidity associated with a surgical procedure and particularly to avoid the permanence of this surgical therapy. In addition, the drug has been shown to be effective in the treatment of Sudeck's atrophy,¹⁷ a condition that may occur usually 1 month after the onset of causalgia and which is characterized by severe trophic changes, skin changes, and osteoporosis. In our series, all cases of causalgia were cured by treatment with phenoxybenzamine, regardless of their time of presentation, location of injury, or presence of trophic changes. The success of phenoxybenzamine treatment again suggests an active role of the postganglionic sympathetic nervous system in the pathogenesis of causalgia, but fails to elucidate the exact mechanism. We realize that the lack of a controlled trial is a criticism of our series, but such a trial was not feasible due to the existing war conditions. Three patients, however, served as their own internal controls: when the drug was unavailable, all three had recurrence of pain which was abolished with reinstitution of phenoxybenzamine. The reason for lack of recurrence of pain after termination of the complete phenoxybenzamine regimen is not presently known. Side effects of the drug were minimal, consisting mainly of mild orthostatic hypotension and transient ejaculatory problems. None of the side effects required interruption of treatment or persisted after termination of treatment.

Conclusions

Forty consecutive cases of causalgia were successfully treated with oral phenoxybenzamine. Maximum daily doses ranged between 40 and 120 mg. and duration of treatment averaged 6 to 8 weeks. All patients achieved permanent cure and only mild transient complications of treatment were observed. Although the exact mechanism of action of phenoxybenzamine in the treatment of causalgia is not known with certainty, we feel that phenoxybenzamine treatment is simple, safe, and effective with less morbidity than surgical sympathectomy. In addition, we recommend that treatment should be instituted directly following diagnosis in order to prevent the development of trophic changes.

Acknowledgments

The authors would like to thank Dr. Robert King and Dr. Peter Bosch for their critical review of the manuscript, and Mrs. Helen Memh for secretarial assistance.

References

1. Baker AG, Winegarner FG: Causalgia. A review of twenty-eight treated cases. *Am J Surg* 117:690-694, 1969
2. Bernstein JJ, Pagnanelli D: Long-term axonal apposition

- in rat sciatic nerve neuroma. *J Neurosurg* 57:682-684, 1982
3. Blumberg H, Jänig W: Neurophysiological analysis of efferent sympathetic and afferent fibers in skin nerves with experimentally produced neuromata, in Siegfried J, Zimmermann M (eds): *Phantom and Stump Pain*. Berlin/Heidelberg/New York, 1981, pp 15-31
4. Denmark A: An example of symptoms resembling tic douloureux produced by a wound in the radial nerve. *Med Chir Trans* 4:48-52, 1813
5. Devor M: Nerve pathophysiology and mechanisms of pain in causalgia. *J Auton Nerv Syst* 7:371-384, 1983
6. Devor M, Bernstein JJ: Abnormal impulse generation in neuromas: electrophysiology and ultrastructure, in Culp WJ, Ochoa J (eds): *Abnormal Nerves and Muscles as Impulse Generators*. New York: Oxford University Press, 1982, pp 363-379
7. Devor M, Jänig W: Activation of myelinated afferents ending in a neuroma by stimulation of the sympathetic supply in the rat. *Neurosci Lett* 24:43-47, 1981
8. Doupe J, Cullen CH, Chance GQ: Post-traumatic pain and causalgic syndrome. *J Neurol Neurosurg Psychiatry* 7:33-48, 1944
9. Freeman NE: The treatment of causalgia arising from gunshot wounds of the peripheral nerves. *Surgery* 22: 68-82, 1947
10. Gilman AG, Goodman LS, Gilman A: *The Pharmacological Basis of Therapeutics*, ed 6. New York: Macmillan, 1980
11. Granit R, Leksell L, Skoglund CR: Fibre interaction in injured or compressed region of nerve. *Brain* 67: 125-140, 1944
12. Granit R, Skoglund CR: Facilitation, inhibition and depression at the "artificial synapse" formed by the cut end of a mammalian nerve. *J Physiol (Lond)* 103:435-448, 1945
13. Hannington-Kiff JG: Intravenous regional sympathetic block with guanethidine. *Lancet* 1:1019-1020, 1974
14. Hannington-Kiff JG: Relief of causalgia in limbs by regional intravenous guanethidine. *Br Med J* 2:367-368, 1979
15. Hannington-Kiff JG: Relief of Sudeck's atrophy by regional intravenous guanethidine. *Lancet* 1:1132-1133, 1977
16. Kirklin JW, Chenoweth AI, Murphey F: Causalgia. A review of its characteristics, diagnosis, and treatment. *Surgery* 21:321-342, 1947
17. Lankford LL: Reflex sympathetic dystrophy, in Omer GE Jr, Spinner M (eds): *Management of Peripheral Nerve Problems*. Philadelphia: WB Saunders, 1980, pp 216-244
18. Leriche R: De la causalgie envisagée comme une névrite du sympathique et son traitement par la dénudation et l'excision des plexus nerveux péri-artériels. *Presse Med* 24:178-180, 1916
19. Leriche R: *La Chirurgie de la Douleur*. Paris: Masson et Cie, 1940
20. Loh L, Nathan PW: Painful peripheral states and sympathetic blocks. *J Neurol Neurosurg Psychiatry* 41: 664-671, 1978
21. Mayfield FH: *Causalgia*. Springfield, Ill: Charles C Thomas, 1951
22. Mitchell SW: On the diseases of nerves, resulting from injuries, in Flint A (ed): *Contributions Relating to the Causation and Prevention of Disease, and to Camp Diseases*. New York: U. S. Sanitary Commission Memoirs, 1867
23. Mitchell SW, Morehouse GR, Keen WW: *Gunshot*

- Wounds and Other Injuries of Nerves.** Philadelphia: JB Lippincott, 1864
24. Nitz AJ, Matulionis DH: Ultrastructural changes in rat peripheral nerve following pneumatic tourniquet compression. *J Neurosurg* 57:660-666, 1982
 25. Peters A, Palay SL, Webster H deF: **The Fine Structure of the Nervous System: the Neurons and Supporting Cells.** Philadelphia: WB Saunders, 1976, pp 186-190
 26. The retracted ephapse. *Lancet* 2:462, 1978 (Annotation)
 27. Richards RL: Causalgia. A centennial review. *Arch Neurol* 16:339-350, 1967
 28. Seltzer Z, Devor M: Ephaptic transmission in chronically damaged peripheral nerves. *Neurology* 29:1061-1064, 1979
 29. Shumacker HB Jr: Causalgia. III. A general discussion. *Surgery* 24:485-504, 1948
 30. Speigel IJ, Milowsky JL: Causalgia. A preliminary report of nine cases successfully treated by surgical and chemical interruption of the sympathetic pathways. *JAMA* 127:9-15, 1945
 31. Spurling RG: Causalgia of the upper extremity. Treatment by dorsal sympathetic ganglionectomy. *Arch Neurol Psychiatry* 23:784-788, 1930
 32. Sunderland S: **Nerves and Nerve Injuries.** London: Churchill Livingstone, 1978, pp 381-420
 33. Ulmer JL, Mayfield FH: Causalgia. A study of 75 cases. *Surg Gynecol Obstet* 83:789-796, 1946
 34. Walker AE, Nulson F: Electrical stimulation of the upper thoracic portion of the sympathetic chain in man. *Arch Neurol Psychiatry* 59:559-560, 1948
 35. Wall PD: On the origin of pain associated with amputation, in Siegfried J, Zimmermann M (eds): **Phantom and Stump Pain.** Berlin/Heidelberg/New York: Springer-Verlag, 1981, pp 2-14
 36. Wall PD, Gutnick M: Ongoing activity in peripheral nerves: the physiology and pharmacology of impulses originating from a neuroma. *Exp Neurol* 43:580-593, 1974
 37. Wall PD, Scadding JW, Tomkiewicz MM: The production and prevention of experimental anesthesia dolorosa. *Pain* 6:175-182, 1979
 38. Wallin G, Torebjörk E, Hallin R: Preliminary observations on the pathophysiology of hyperalgesia in the causalgic pain syndrome, in Zotterman Y (ed): **Sensory Functions of the Skin with Special Reference to Man.** Oxford: Pergamon Press, 1976, pp 489-499
 39. White JC: Sympathectomy for relief of pain, in Bonica JJ (ed): **International Symposium on Pain. Advances in Neurology, Vol 4.** New York: Raven Press, 1974, pp 629-638
 40. White JC, Heroy WW, Goodman EN: Causalgia following gunshot injuries of nerves. Role of emotional stimuli and surgical cure through interruption of diencephalic efferent discharge by sympathectomy. *Ann Surg* 128:161-183, 1948
 41. Wiesenfeld-Hallin Z, Hallin RG: Possible role of sympathetic activity in abnormal behavior of rats induced by lesion of the sciatic nerve. *J Auton Nerv Syst* 7:385-390, 1983

Manuscript received August 23, 1983.

Address for Drs. Ghostine and Azar: Central Military Hospital and Berbir Medical Center, Beirut, Lebanon.

Address reprint requests to: Youssef G. Comair, M.D., Division of Neurosurgery, Notre Dame Hospital, P. O. Box 1560, Montreal, Quebec H2L 4K8, Canada.