
Four patients with presumed radiation-induced optic neuropathy who were treated with hyperbaric oxygen are presented. Radiation therapy had been given previously for a pituitary tumor in two patients and for reticulum cell sarcoma and meningioma in one patient each. One patient with unilateral decreased vision had a virtual “cure” after 14 treatments; another had marked improvement after 14 treatments but relapsed and did not respond to further sessions. The other two patients were probably treated too late after onset of optic neuropathy for good effect. These authors acknowledge that spontaneous remission can occur in this disease but believe that the two patients who were quickly recognized and treated had improvement that can be attributed to the hyperbaric oxygen treatment itself. They recommend hyperbaric oxygen emergently if visual loss has occurred within 2 weeks and continued therapy of 14 sessions after visual acuity has improved and stabilized.

*Lyn A. Sedwick, M.D.*


A 76-year-old woman with a sedimentation rate of 115 mm/hr (Westergren) had transient loss of vision in her right eye and headaches. A temporal artery biopsy disclosed a subacute granulomatous reaction without giant cells, some active inflammation, and fibrinoid necrosis. This biopsy was thought to be consistent with the diagnosis of polyarteritis nodosa, and further pathologic specimens from the patient’s bowel likewise showed evidence of this disease. The authors underscore that, although the literature does not stress the use of temporal artery biopsy in diagnosing this disorder, such a biopsy is often helpful in diagnosing polyarteritis nodosa as giant cell arteritis.

*Lyn A. Sedwick, M.D.*


These papers each describe one patient with a traumatic third nerve lesion who exhibited segmental contraction of the iris sphincter, in one with down-gaze and in the others spontaneously. Neither pupil reacted to light and, in the first case, classic signs of aberrant regeneration developed after the pupil abnormality was documented. In both cases, Dr. Cox implicates neurons in the ciliary ganglion as generating the impulse to contraction, although the exact mechanism postulated differs in each case.

*Lyn A. Sedwick, M.D.*


This is one of many recently published articles discussing the merits of botulinum toxin in the treatment of facial spasm. Unfortunately, at the time this is written, botulinum toxin is no longer available for clinical use. The company that commercially prepares the toxin is unable to obtain adequate liability insurance. For many patients incapacitated by facial spasm, botulinum toxin is clearly the treatment of choice. Let us hope that this undesirable situation is quickly resolved so that our patients may benefit from this valuable new treatment.

*Walter M. Jay, M.D.*