ACUPUNCTURE & ELECTRO-THERAPEUT. RES., INT. J., Vol. 5, pp. 83-91, 1980

0360-1293/80/010083-09$02.00/0

MALE-PATTERN HAIR LOSS -- A SUPRAORBITAL NERVE ENTRAPMENT SYNDROME?

C.C. Gunn, M.A., M.B., B. Chir.
Rehabilitation Clinic
Workers' Compensation Board of British Columbia
6951 Westminster Highway
Richmond, British Columbia V7C 1C6

Mathew H.M. Lee, M.D., M.P.H., F.A.C.P.
Professor & Director of Rehabilitation Medicine
Goldwater Memorial Hospital
New York University Medical Center, New York, N.Y.

(Received 10 August 1979; Accepted with Revision 15 December 1979)

ABSTRACT

The cause of male-pattern hair loss remains obscure. It is noted to occur in the geographic distribution of the supraorbital and sometimes the great occipital nerves. It is suggested that these nerves are susceptible to entrapment and subsequent neuropathy since signs of the latter precede and accompany hair loss. Male-pattern alopecia is uncommon in women, yet neuropathy and deprivation of the trophic factor can lead to hair loss in any part of the body in both men and women. It seems therefore that scalp hair loss, more common in the male, occurs because higher levels of testosterone create a situation in which scalp nerves become vulnerable to neuropathy. Testosterone greatly increases muscle and skeletal bulk, thickens skin and reduces subcutaneous fat, most especially in the head. These factors may well cause increased tension to scalp nerves. Recent animal experiments have demonstrated that early neuropathy may be amenable to electrical stimulation and the author has personally found that hair loss may thus be temporarily retarded. Further study by those working in this field is recommended. Perhaps surgical release of the entrapped nerves (as in a carpal tunnel syndrome) may be the final answer.

INTRODUCTION

Male-pattern hair loss in the scalp (premature or senile alopecia), although benign, is a common but important complaint since no man wishes to lose his crowning glory. The waste of vast sums of money for medicines promising in vain a head of thick luxuriant hair proves the point.

Scalp hair loss may be patchy, diffuse or "geographic." Patchy alopecia, whether scarring or non-scarring, usually follows the local destruction of hair follicles by a disease agent -- infective, traumatic or "idiopathic." Diffuse hair loss which is usually the result of a generalized disease (pneumonia, typhoid, hypothyroid and hypopituitary states, systemic

lupus erythematosus or ingested poisons -- thallium, heparin or even excessive Vitamin A and others) may be transient or permanent depending on the underlying pathology. Discussed here is the very common condition of premature or senile alopecia which is far more common in men than women. Clinically, the earliest hair loss extends back on both sides of the forehead in an M-shape (Figure 1) to meet a slowly enlarging area of similar hair loss on the vertex of the scalp (Figure 2). The degree of hair loss varies with the individual as does the age at which it begins. The dominant factors are stated in textbooks to be hereditary, age of the individual and hormones. Castrated males are reported not to have this male-pattern hair loss.

Contrary to the generally-accepted statement that there is no known cause for this "hereditary" disease, this paper suggests that it is probably an entrapment syndrome [1, 2], the result of peripheral neuropathy and entrapment of the supraorbital and sometimes the great occipital nerves. It may be explained by the physiology of peripheral neuropathy [2] which is well known to physiologists and clinicians involved in peripheral nerve disease but has not been applied to alopecia. Early hair loss may possibly respond to treatment of the neuropathy and entrapment.

CLINICAL OBSERVATIONS

Male-pattern hair loss is "geographic" in that it corresponds to the distribution of some peripheral nerves. Anteriorly, and as far back as the lambdoid suture, it coincides with the supraorbital nerves (Figures 1 and 2) though it tends to be less in the midline or "widow's peak." Initially, hair growth is retarded in the affected area; the hair becomes brittle, less resilient and liable to fracture. Subsequently as the follicles lose their vitality, hair loss is complete and the skin becomes tight and shiny. These changes, it is suggested, may be attributed to the loss of the trophic or nutritional factor in hair follicles as a sequence of peripheral neuropathy.

DISCUSSION

Peripheral neuropathy is distinct from denervation and has its own pathological significance. Its symptoms and signs are also different from those of total denervation. Although the causes of peripheral neuropathy are myriad, the peripheral nerve has but a limited repertoire of pathological reactions (axonal degeneration, segmental demyelination or neuronal disease)[2,3]. An early and important function of the peripheral nerve to be attenuated in the peripheral neuropathy is that related to the autonomic system.

The autonomic system is a division of the peripheral nervous system and is, by definition, entirely motor (except for vascular and visceral afferents). Autonomic efferent fibres supply the smooth muscles of the blood vessels. In neuropathy, when denervation supersensitivity [4,5,6,7] develops, vasoconstrictor disturbances occur affecting the trophic or nutritional state of all the nerve's target tissues including skin, hair and subcutaneous tissues. In autonomic dysfunction, pilomotor, sudomotor as well as vasomotor disturbances occur. Unlike the complete division of a peripheral nerve, which causes the denervated region of the skin to have a pink or rosy appearance because of vasodilitation brought about by interruption of sympathetic fibres to that part, neuropathy and denervation supersensitivity generally cause mottling of the skin, i.e., combined pallor and cyanosis

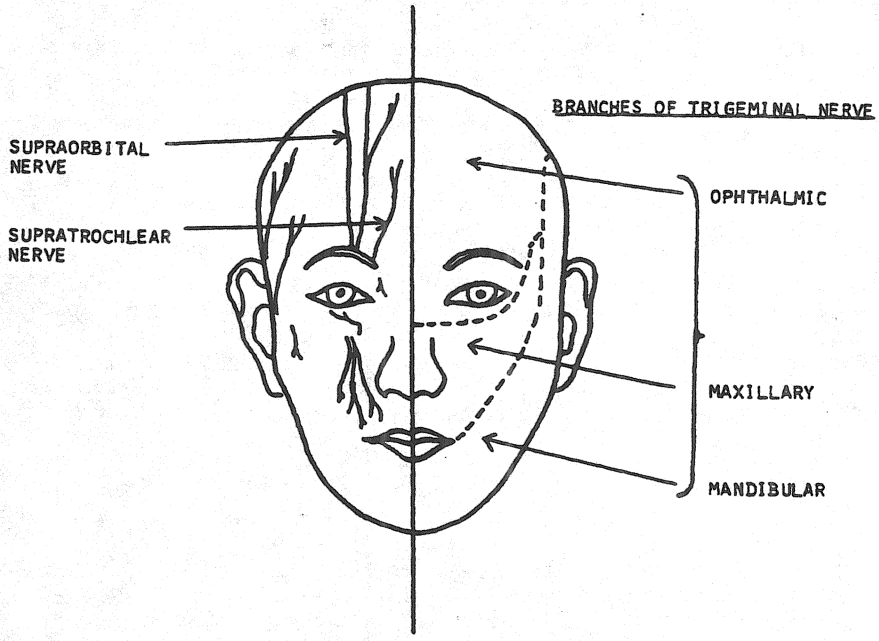


Fig. 1. Male pattern hair loss always occurs in the distribution of the ophthalmic branch sparing the maxillary and mandibular branches.

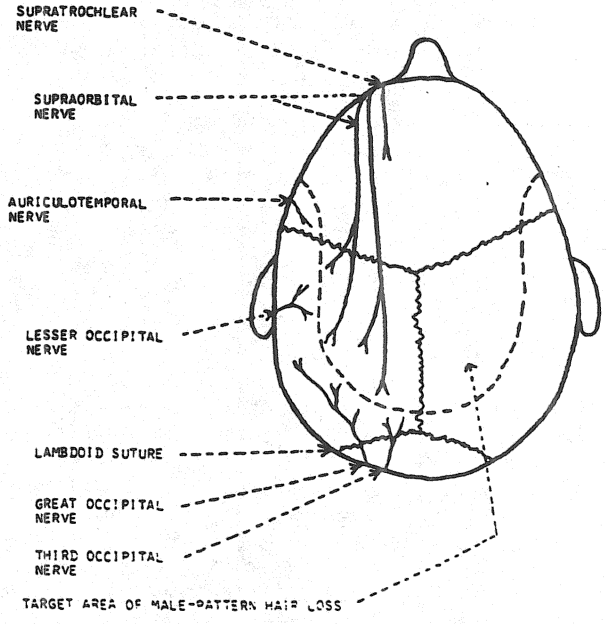


Fig. 2. The area supplied by the supraorbital nerves reaches as far back as the lamboid suture.

(although this is not noticeable in the scalp). The skin under such conditions has a lower temperature. Omura,[8], using an electronic thermometer with ultra-fine needle-shaped blunt-tip heat sensor probe and thermalcouple has been able to accurately measure the supraorbital temperature of the forehead. In skin, an early sign of neuropathy is trophedema [9]. There is gradual fibrosis of the subcutaneous tissue which develops a boggy, inelastic texture and when rolled between thumb and finger is easily distinguishable from subcutaneous fat (Figures 3,4 and 5). Unlike edema, trophedema is not gravity-dependent and is non-pitting to digital pressure, but when a small blunt instrument, e.g., the end of a matchstick is used, the indentation produced is clear-cut and persists for many minutes, distinctly longer than that over normal skin (Figure 6). Hair growth is retarded in neuropathy and hair texture in trophedema is brittle. Early hair loss begins as a result of attrition and fracturing occurs at the proximal end where the structural integrity of recent hair growth is impaired. Eventually the skin loses its delicate indentations, becomes inelastic, smooth and shiny and hair loss is irreversible when follicles perish. The undernourished skin is then only able to support the growth of fine, short and immature hair. Male-pattern hair loss occurs in the distribution of the supraorbital nerves which are particularly vulnerable to entrapment at the supraorbital notches or foramina. Entrapment in the male may also be accentuated by a more prominent supraorbital margin. Hair loss tends to be less at the midline or the "widow's peak" probably because there is some cross-innervation and overlapping of the nerves from both sides; furthermore, the medial branch of the nerve has only to pass through the muscle belly of the frontalis whereas the lateral branch has to perforate the unyielding epicranial aponeurosis.

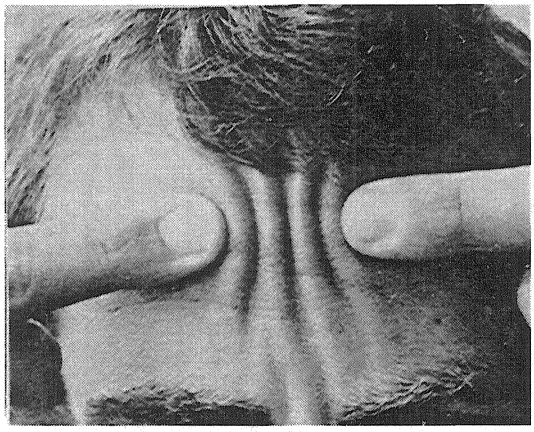
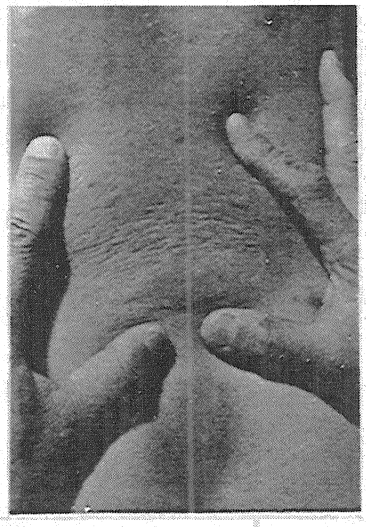


Fig. 3. Trophedema with boggy inelastic texture when skin is "rolled."



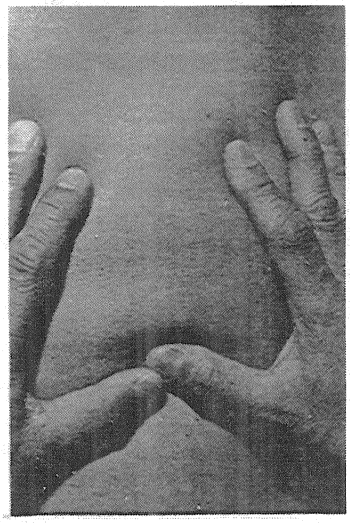


Fig. 4. Trophedema occurs on any part of the body with neuropathy -- here shown in the low back.

Fig. 5. A normal low back without trophedema -- shown for comparison with Fig. 4.

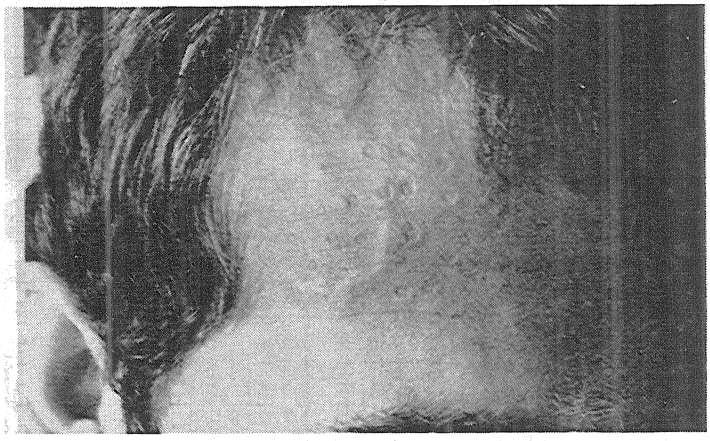


Fig. 6. The "matchstick test" for trophedema (positive).

Another early clinical manifestation of neuropathy which leads to denervation supersensitivity is the development of hyperalgesia [9,10,11,12]. The affected nerves as they cross the orbital margin or at the occiput become tender to digital pressure with the tenderness paralleling the neuropathy and the period of greatest hair loss.

Hair loss results from the deprivation of the trophic or nutritional factor of hair follicles and may occur anywhere on the body. It may be

generalized following a severe constitutional upset or acute illness, which, if of short duration and without irreversible follicular destruction, is followed by hair growth several weeks later. The trophic factor, however, may be locally jeopardized by destructive agents or in the distribution of a peripheral nerve following peripheral neuropathy. Chronic, low-grade, peripheral neuropathy can not only cause hair loss on the scalp, but also at any other part of the body where the trophic function of the nerve is impaired. For example, in both men and women with chronic low back neuropathy, when L5 or S1 nerves are involved, there often is hair loss in the legs within their "geographic" dermatomes (Figures 7 and 8), associated with other clinical signs of neuropathy [9], although pain may or may not be an accompanying factor. Male-pattern hair loss belongs to this latter category and is confined in the scalp to the geographic distribution of the above-mentioned superficial nerves.

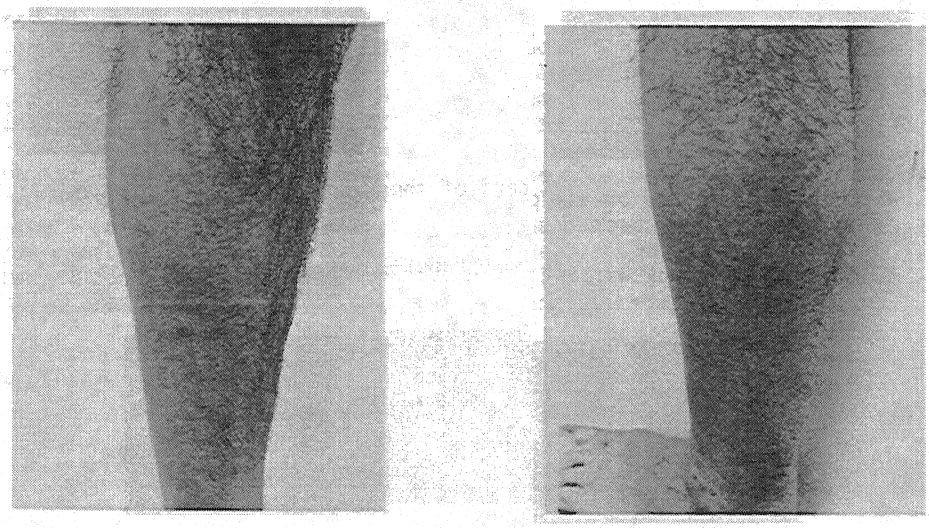


Fig. 7. Hair loss in geographic distribution of L5 dermatome (right side).

Fig. 8. Hair loss in S1 dermatome.

It is important to realize that actual physical interruption of the nerve is not necessary for neuropathy to develop -- even minor degrees of damage can destroy the microtubules within the nerve axons. Such a nerve still conducts impulses, evokes muscle action potentials and contraction, but its trophic function is impaired. Furthermore, in early neuropathy, electrical studies are generally unrevealing. Nerve conduction velocities generally remain within normal limits and electromyography may only show a slight loss of the recruitment pattern. Later, insertional activity may be increased and polyphasic wave forms appear in greater numbers. Electromyographic evidence of denervation such as abnormal potentials at rest are late signs and denote denervation rather than neuropathy [3].

Male-pattern hair loss is not common in women and eunuchs because of lower circulating levels of testosterone. Since women, despite their lower levels of androgen, can commonly have hair loss in the legs from lower spinal neuropathy but not in the scalp, it is reasonable to surmise that hair loss

is not the primary consequence of high testosterone levels but of neuropathy. In males, it is probable that high male hormone levels create a situation in which scalp nerve neuropathy is more likely to occur. Testosterone has remarkable growth-promoting properties leading to great development of skeletal musculature and thickening of skeleton. Skin also becomes thicker with a proliferation of sebaceous glands and a reduction of subcutaneous fatty tissue. The region of most spectacular change is in the head -- the occipito-frontalis (temporal, masseter and corrugator supercilii) muscles show a prolific increase in strength and bulk [13] with accentuation of the supraorbital margins and superciliary arches (most obviously seen in the skull of the male ape). These changes are likely to subject the involved superficial nerves to greater stress and spatial compromise from traction and pressure. The "hereditary" factor in hair loss is probably related to this skull/musculature formation and structure.

Is geographic hair loss reversible? Obviously, when all hair follicles have been destroyed, alopecia is irrevocable. However, in the early stages before total follicular damage, when hair growth is merely retarded and hair loss is due to hair strand friability, restoration of the trophic factor may help. Lomo, in animal experiments, has shown that denervation supersensitivity, a product of neuropathy, may be reduced or abolished by electrical stimulation [13]. Hypersensitivity (as assayed by the sensitivity of muscle extrajunctional membrane to acetylcholine) was shown to diminish at a rate depending upon the amount and pattern of the electrical stimuli. Even very low levels of electrical stimulation were shown to strongly suppress hypersensitivity. Applying this concept, one of the authors, (C.C.G.) and three personal friends who were having early hair loss, tried transcutaneous electrical neural stimulation (TENS) administered twice a day to the affected nerves and were able to reduce trophedema, tightness of skin and nerve tenderness. (Using a commercially available, portable, single-channel, battery-operated unit with biphasic asymmetrical square wave form -- output 5-50 milliamps, pulse rate 15-150 pulses per second; nominal values when measured into 510 ohm load. Exact values are probably not critical.) Within two weeks of treatment, the daily count of fallen hair strands when combing diminished from about ten strands per day to about two or three. This was followed by an improvement of hair texture and a gradual resumption of hair growth rate. Some immature hair actually progressed to maturity with the receding hair line postponed (Figure 9) and maintained (at the time of writing for over a year.)

Stimulation may also possibly be induced by acupuncture. Omura, using an ultra-miniature reflection-type photoelectric plethysmographic sensor has demonstrated that acupuncture induced changes in capillaries and arterioles -- initial vasoconstriction is followed by vasodilation. The beneficial effects of acupuncture are probably associated with significant vasodilation of the micro-circulatory system, not only of local areas but distal parts of the body. These are accompanied by characteristic blood chemistry changes, including the production of ACTH, morphine-like substances and prostaglandin. (His study indicated that not only is the temperature of the supraorbital region increased but also that of the underlying brain and even the extremities.)

Obviously, with the underlying pathology of entrapment unrelieved, the continuing daily application of electrical stimulation remains necessary. As control, periods when stimulation were discontinued were followed within a few days by the resumption of high daily counts. Nevertheless, it was found that once trophedema and skin tightness were reduced, less frequent stimula-

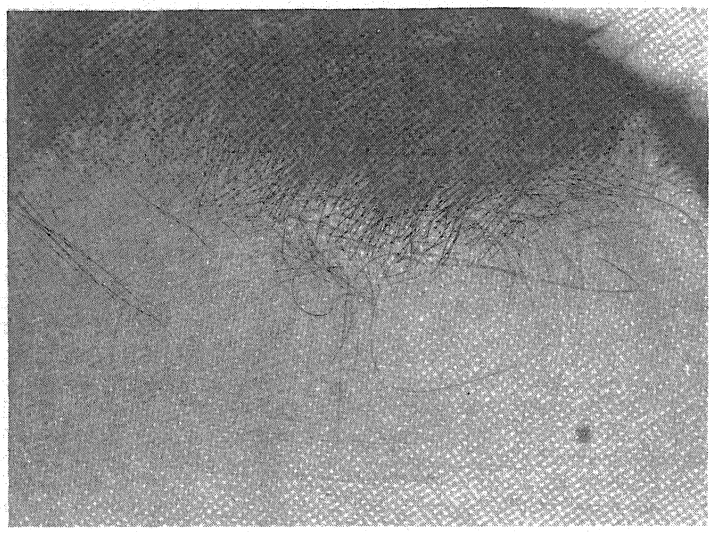


Fig. 9. Some immature hair progressed to maturity following TENS.

tions became necessary. Stimulation presently given once a day once or twice a week is all that is required to maintain a reduced daily hair fall count, although a degree of stubborn trophedema persists because of the underlying entrapment. Also as control, during periods when only one side of the head was stimulated, ignoring the other, hair loss was more on the untreated side.

The clinical findings accompanying early hair loss presented in this paper have been repeatedly confirmed in a large number of random subjects. The authors put forward the proposal to dermatologists and others that further investigations based on this preliminary and admittedly personal report may be worthwhile. Some suggestions are: randomized clinical trials employing electrical stimulation; animal experiments with neuropathy induced by trauma or the application of local neurotoxic agents (e.g., colchicine or vinblastin) unilaterally and using the contralateral side as control; anatomical studies to determine if the tendency to hair loss is related to the presence of a supraorbital foramen rather than a notch and therapeutic-wise. Would surgical decompression, which would appear to be a relatively simple procedure as for a carpal tunnel syndrome, prove the answer? As a "new operation and not a new drug," it should be possible for the value of decompression to be soon assessed without the need of randomized studies [15].

REFERENCES

1. Kopell, H.P., Thompson, W.A.L.: Peripheral Entrapment Neuropathies. Williams & Wilkins, Baltimore, Maryland, 1963.

2. Bradley, W.C.: Disorders of Peripheral Nerves. Blackwell, Oxford, pp. 152-155, 1974.

3. Goodgold, J., Eberstein, A.: Electrodiagnosis of Neuromuscular Diseases. Williams & Wilkins, Baltimore, Maryland, pp. 157-180, 1972.

4. Cannon, W.B., Rosenblueth, A.: The Supersensitivity of Denervated Structures. New York, MacMillan, pp. 1-22, 185, 1949.

5. Axelsson, J., Thesleff, S.: A study of supersensitivity in denervated mammalian skeletal muscle. J Physiol 147:178, 1958.

6. Katz, B., Miledi, R.: The development of acetylocholine sensitivity in nerve-free segments of skeletal muscle. J Physiol 170: 380-396, 1964.

7. Tower, S.S.: The reaction of muscle to denervation. Physiological Review 19 (1): 1-48, 1939.

8. Omura, Y.: Normal and abnormal relationship between brain circulation estimated by supraorbital temperature measurements and grasping force of the corresponding hands: Their clinical application for diagnosis and evaluation of various modes of treatment. Acupuncture & Electro-Therapeutics Research 3 (1-2): 49-96, 1978.

9. Gunn, C.C., Milbrandt, W.E.: Early and subtle signs in low back sprain. Spine 3: 267-281, 1977.

10. Gunn, C.C., Milbrandt, W.E.: Tenderness at motor points -- A diagnostic and prognostic aid for low-back injury. Bone and Joint Surg 58A (6): 815-825, 1976.

11. Gunn, C.C., Milbrandt, W.E.: Tenderness at motor points -- an aid in the diagnosis of pain in the shoulder referred from the cervical spine. JAOA 77: 196/75-212/91, 1977.

12. Gunn, C.C.: "Pre-spondylosis" and some pain syndromes following denervation supersensitivity. In press, Spine.

13. Goodman, L.S., Gilman, A.: The Pharmacological Basis of Therapeutics. Macmillan, New York, Fifth Ed., pp. 1451-1471, 1975.

14. Lomo, T.: The role of activity in the control of membrane and contractile properties of skeletal muscle. Chap 10. Motor Innervation of Muscle. Edited by S. Thesleff. Academic Press, London, New York, San Francisco, 1976.

15. Bonchek, L.I.: Are randomized trials appropriate for evaluating new operations? N Engl J Med 301 (1): 44-45, 1979.