

OFFICE SURGERY: ELECTRODESSICATION IN SYRINGOMAS AND TRICHOEPITHELIOMA

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SYNOPSIS

Electrodesiccation with a hyfrecator, is a simple surgical procedure that is performed frequently in many dermatologists' offices. I have used this as the main treatment for syringomas and trichoepitheliomas, for a few years. Some of the problems encountered, include the selection of cooperative and motivated patients, the most appropriate local anaesthesia and the adequate amount of desiccation. The post-desiccation complications include peri-orbital oedema, scarring and hyperpigmentation. So far, local anaesthesia via a dental syringe and repeated sessions have proven to give acceptable results.

INTRODUCTION

Most dermatologists use the hyfrecator to destroy a wide range of benign skin tumours, whether these be verruca vulgaris or plana, seborrhoeic keratoses, naevi; often with good results. A hyfrecator is a simple compact device that generates a high frequency damped current of relatively high voltage and low amperage by means of spark-gaps. The spark-gap currents generated are for fulgration, desiccation and coagulation. The hyfrecator delivers a high alternating current to tissue producing an electric field about the tip of the electrode. The high resistance of this electric current causes mechanical disruption and death to the cells (1).

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Fulguration is a mono-terminal technique with the electrode held just above the lesion and the sparks generated destroys the lesion. Electrodesiccation, also mono-terminal, destroys by dehydration from the heat generated by the electrode placed within the lesion (2,3,4,5). The patient is not incorporated into the circuit. This procedure is fast, convenient and has produced good cosmetic results. Electrocoagulation is seldom used, is bi-terminal, using low voltage, low frequency and high amperage. Of these, we found electrodesiccation most useful for treating syringomas and trichoepitheliomas.

METHODS AND MATERIALS

Syringomas present as skin coloured papules, predominantly periorbital, in females and lesser in males, from the second decade. These are eccrine gland hamartomas and are sited in the upper to mid dermis. They consist of two cell layered cysts filled with an amorphous material, or as aggregates of eccrine cells looking like "tadpoles with a tail".

Trichoepitheliomas present as papular, sometimes cystic lesions, more prominently on the paranasal and cheek area. These are autosomal dominant in inheritance and may appear at an earlier age group, in children. These are hair follicle hamartomas. On histology, they can be mistaken for basal cell epithelioma.

The lesions are preferably cleaned with a non-inflammatory solution, normal saline. If alcohol or spirit is used, it should be allowed to dry completely first. Various forms of anaesthetic procedures were tried: from local infiltration of the lesions with 1% lignocaine using a fine needle, using a dental syringe and needle: this is the best; to facial and supra orbital nerve blocks, and general anaesthesia. Facial blocks with 1 ml of 1% lignocaine, are given at the infra orbital foramen, on the lower rim of the orbital ring, against the bone.

The patient's hair is wrapped in a dressing towel. The eyelid is protected by a piece of gauze, and the electrode is pointed at the peri-orbital lesions, away from the cornea. The lesions are electrodesiccated with a low power setting. The procedure was repeated in stages. Both types of lesions were desiccated till they were flat. They may be curetted and redessicated to achieve further depth of destruction. Tetracycline ointment is applied. These lesions heal by re-epithelisation. A scab forms and drops by the sixth to twelfth day. A thinner, tenacious scab forms and when this drops, a soft, pink flat scar forms.

From June 1984 to May 1985, 36 cases of syringomas were done, 32 females to 4 males. Of these, 12 cases required repeat cauteries. There were 5 cases of trichoepithelioma, 3 males and 2 females. 3 cases required repeat cauteries. Patients noted a cosmetic improvement of 70% in that the lesions flattened. Two hyfrecators were used, the Birtcher (6) and the Skypak.

DISCUSSION

Some of the problems encountered with the technique and the post cautery complication are as follows:

1. The eyelid and peri-orbital area is a sensitive area and patients tend to blink frequently, screw up the

eye muscles and shift about. To overcome this, I have tended to select patients who are very keen on some form of cosmetic surgery and they are advised to relax. Anxious patients and youngsters are offered general anaesthesia.

2. Anaesthesia: Local anaesthesia with multiple injections of Lignocaine around the periorbital area causes pain. At the same time, it produces oedema and the syringomas/trichoepitheliomas are submerged and difficult to identify. Excess lignocaine produces post-operative oedema and erythema, which last 3—4 days. This is overcome by using the dental syringe with a very fine and very long needle, allowing the lignocaine to be given in smaller doses. A facial nerve block is the other alternative. This can be performed by the trained doctor but this may not give adequate anaesthesia, at times.

General anaesthesia is necessary for extensive trichoepitheliomas of the paranasal and cheek area and is useful in the apprehensive young patients. These patients are observed for 3—4 hours post operatively and discharged home. Thus it can be performed in day surgery.

3. Scars: To minimise excessive scarring, electrodesiccation is carried out in 3—4 stages using low power. In electrocautery, the amount of destruction increased linearly with increases of power and the length of application; for both the surface damage and the depth of damage (4). It would be easier if the operator knows the pathology of the lesions so that he can apply an appropriate power and length of time. The operator can use the more expensive machines in which the depth of destruction is calibrated. Otherwise he has to experiment on other skin to estimate the depth of destruction to power and time. Hypertrophic scars and ectropion have not occurred.
4. Post cautery hyperpigmentation: this cannot be avoided but is uncommon and fades by a few months. This occurs in the pigmented race.

The best cosmetic results is usually produced by one with clinical and histologic knowledge of the lesion, working knowledge of electrocautery and experience.

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POSTURAL HYPOTENSION IN THE ELDERLY

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SYNOPSIS

Postural hypotension occurred in 22% of elderly patients seen in a general medical unit. There was an increased incidence of the condition with advancing age. Though the majority of patients were asymptomatic, postural hypotension was found to be related with morbid falls, transient cerebral attacks and cerebral infarction. Multiple precipitating factors were common in these patients but the underlying basic mechanism is often related to the aging autonomic nervous system and peripheral arterial rigidity associated with supine hypertension. The general principles of management and the associated therapeutic problems are discussed.

INTRODUCTION

In the elderly, the aging process is often associated with impaired homeostatic mechanisms. Under normal circumstances, the elderly may function adequately but the ability to compensate is easily compromised when rapidly changing circumstances occur. Postural hypotension is a common clinical disorder in the elderly which reflects impaired homeostasis. This condition is defined as a drop in systolic blood pressure of more than 20mmHg and an associated drop in diastolic blood pressure of at least 10mmHg (1). The following study examines the clinical patterns of postural hypotension in elderly patients seen in the outpatient clinic.

MATERIALS AND METHODS

200 elderly outpatients over 65 years were screened for the presence of postural hypotension. There were 80 females and 120 males and their ages range from 65 years to 98 years. These patients were mostly ambulatory or could stand with minimal support; ill patients were excluded from the study. A standardised measurement technique was used for measuring both lying and standing blood pressures. Each patient was rested for ten minutes in the supine position and after this rest period, three consecutive readings were taken at one minute intervals. Having stood erect for two minutes, another three consecutive one-minute interval readings were taken. The average of the three

readings was taken for either supine or erect blood pressure. The patients were enquired for symptoms related to postural hypotension. In addition, the possible precipitating factors and the resulting morbidity from postural hypotension were evaluated.

RESULTS

In this study systolic blood pressures showed a rising trend between the 6th and 9th decades; in contrast there was a plateauing of diastolic blood pressures with increasing age (Fig. 1). Postural hypotension occurred in 22% of elderly outpatients. There was increased incidence of postural hypotension in the older age groups (Table 1). The majority of

Figure 1

Distribution of systolic and diastolic blood pressures according to age. White columns: Average systolic BP, dark column: Average diastolic BP

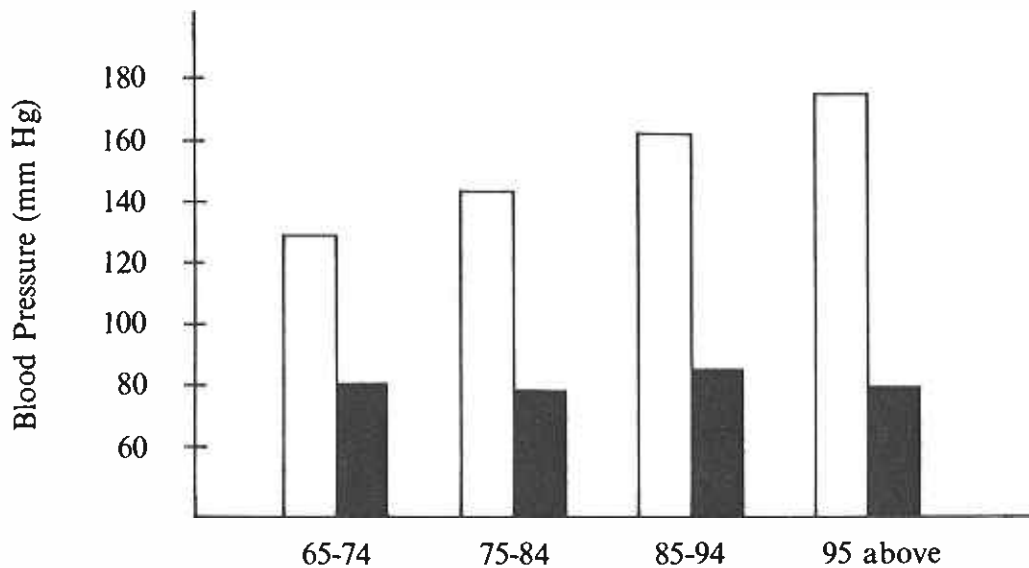


Table 1

Prevalence of postural hypotension

<u>Age</u>	<u>Percentage with postural drop</u>
65 and above	22% (44 out of 200 patients)
65-74	18%
75-84	21%
85 above	26%

patients were asymptomatic (28 out of 44 patients). Of the remaining 16 symptomatic patients, the most common complaint was that of dizziness (Table 2).

TABLE 2: CLINICAL PRESENTATION

Nos of patients	
A	Asymptomatic 28
	Symptomatic 16
Types of symptoms	
	Dizziness or lightheadedness 7
	loss of balance and fall 4
B	vertigo 2
	syncope 3

Postural hypotension was noted to be associated with morbid falls, transient ischemic attacks and cerebral infarction (Table 3). There was no obvious precipita-

**TABLE 3
MORBIDITY ASSOCIATED WITH
POSTURAL HYPOTENSION**

Morbidity	Nos of patients
Falls	4
Transient ischemic attacks	2
cerebral infarct	2

ting factor in one-third of the cases. In the remaining two-thirds multiple factors were often present. The commonest precipitating cause was related to drug therapy. Multiple drug therapy was a common feature; methyldopa, adalat and isordil were the common offending agents (Table 4).

DISCUSSION

Postural hypotension is a common disorder in the elderly. In four major studies overseas, the prevalence of this condition ranges from 11 to 30% (1). The figure of 22% in this study could have been higher if hospitalised patients were included as acute illnesses may contribute to deconditioning and further postural drop.

There was an increased incidence of postural hypotension with age in this study. This can be explained by decreased baroreceptor sensitivity associated with ageing; cardiac beat-to-beat variation and heart-rate response to standing have been demonstrated to decline with age (2). Postural hypotension exemplifies the concept of impaired homeostasis due to impairment of the autonomic nervous system.

The trend of increasing systolic pressures and plateauing of diastolic pressures with age is consistent with the large-scale results of the Community

**TABLE 4
PRECIPITATING FACTORS IN
POSTURAL HYPOTENSION**

Precipitating factors	Nos of patients
Drugs	14
methyldopa	8
isordil	6
adalat	5
diuretics	4
antidepressants	2
largactil	2
madopar	1
Dehydration	2
Anaemia	3
hypokalemia	5
cerebrovascular disease (strokes)	4
Parkinson's disease	1
varicose veins	5
diabetes with peripheral neuropathy	3
No obvious factors	15

Hypertension Evaluation Clinic Program in the States (3). Systolic hypertension has to be considered as important as the diastolic component as systolic pressures are shown to have a stronger correlation to the risk of stroke and cardiac failure in the elderly (4). In relation to postural hypotension two recent studies have shown that there is a positive correlation between postural hypotension and the lying systolic blood pressure (5,6). The old concept of impaired autonomic nervous system as the cause of postural hypotension, may now be supplemented by the recent evidence that the loss of arterial rigidity associated with systolic hypertension can also contribute to the postural drop. Blood vessels show local regulation to changes in intramural pressures. The loss of elasticity of peripheral arteries will lead to rapid fall of blood pressure with gravity before baroreceptor reflex activities come into play (7). Blood vessel rigidity may also interfere with the function of the central baroreceptors (8). Thus aging of the autonomic nervous system and arterial rigidity secondary to systolic hypertension are both important contributory factors to postural hypotension in the elderly. As the two factors deteriorate with age, it is expected that postural hypotension worsens with aging.

The majority of old people with postural hypotension are asymptomatic (9). If symptom do occur, the most common complaint is that of dizziness or loss of balance. In the normal person the cerebral circulation has a substantial ability for autoregulation; decreased cerebral blood flow does not occur till the systolic blood pressure drops to less than 70 mmHg (10). This impaired autoregulation occurs at a higher blood pressure threshold in the elderly. However, due to variations in cerebral autoregulation, elderly patients may remain asymptomatic unless the postural drop in blood pressure is significant (10).

Neurosurgical reviews from the Mayo Clinic revealed the interesting syndrome of primary orthostatic cerebral ischemia in the elderly (11). These patients were noted to have (a) vague symptoms of dizziness on standing up (b) no significant drop in blood pressure to

account for the symptoms (c) marked drop of cerebral circulation pressure on standing as shown by ophthalmodynamometry (d) widespread arteriosclerotic disease in the carotid circulation and (e) dramatic improvement in symptoms after carotid artery bypass. This syndrome may reflect impaired cerebral autoregulation associated with selective aging of the autonomic nervous system in the brain.

The management of postural hypotension in the elderly may be frustrating at times because treatable specific disease modalities may not be present. This is expected as the majority of elderly develop postural hypotension due to aging of their baroreceptor reflex mechanism or peripheral arterial rigidity. Though no specific treatment can be instituted, general supportive measures to reeducate the aged baroreceptor reflex may be started. These include sleeping with the bed elevated, keeping the patient semi-upright on the tilting bed and slow changes of posture on standing up.

Multiple precipitating factors may contribute to further drop in blood pressure in addition to the already compromised baroreceptor reflex mechanism. These must be actively sought as removal of such treatable factors will at least help to alleviate the severity of the condition. Polypharmacy is an important factor to consider. The most common drugs in this study comprise the antihypertensives, calcium channel blockers and coronary vasodilators. Two separate clinical studies have shown that diuretics do not affect the frequency of postural hypotension in the elderly (12). Judicious use of diuretics can lower the blood pressure without producing unwanted postural effects. However, diuretic-induced hypokalemia can be a precipitating cause for postural hypotension (13). In this study there were five patients with hypokalemia associated with postural hypotension; correction of hypokalemia improved the postural drop in three out of the five patients. Apart from hypokalemia, other reversible factors such as anaemia, hyponatremia and dehydration should be actively sought.

Drug therapy for postural hypotension may be worth a trial but as expected in the elderly, addition of new drugs may produce other unwanted side-effects. Fluorocortisone can lead to sodium and fluid retention resulting in cardiac failure. Ergotamine can improve postural hypotension by its vasoconstrictive effect but due to its low bioavailability higher oral doses at 15 mg bd may be required to produce the desired effect (14). Unfortunately this can lead to unwanted vasoconstrictive effects on the coronary and peripheral circulation leading to supine hypertension in the long term. Kochar et al reported a significant effect of indocid on improving postural hypotension (15). Excess prostaglandins in the peripheral circulation has been postulated to cause vasodilatation resulting in pooling of blood. Indocid inhibits pro-

staglandin synthetase and thus reduces the peripheral pool of circulating prostaglandins. Metoclopramide and clonidine have been tried with variable results (16,17). All these drugs are still experimental and have a high tendency to produce unwanted side-effects in the elderly. The final mainstay of management should be based on general supportive measures and the elimination of treatable factors.

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