The Journal of Laryngology and Otology
January 1997, Vol. **111**, pp. 34–37

A retrospective study of the role of the argon laser in the management of epistaxis secondary to hereditary haemorrhagic telangiectasia

P. A. LENNOX, M. HARRIES, V. J. LUND, D. J. HOWARD

Abstract
Epistaxis is a common symptom in patients with hereditary haemorrhagic telangiectasia. It may vary in severity from infrequent mild bleeds to regular severe bleeding, the latter requiring multiple blood transfusions. Laser, which can be used to coagulate the telangiectasia without destroying the overlying nasal mucosa, would theoretically seem to be the ideal mode of treatment. In this pilot study, an argon laser has been used on nineteen patients with hereditary haemorrhagic telangiectasia and is shown to be beneficial in patients with mild or moderate epistaxis. Patients with severe epistaxis, or who fail argon laser treatment, are candidates for alternative therapeutic strategies such as a modified Young's procedure.

Key words: Laser surgery, argon; Epistaxis; Telangectasia, hereditary haemorrhagic

Introduction

Hereditary haemorrhagic telangiectasia is characterized by an autosomal dominant inheritance, multiple telangiectasia, involving skin and mucous membranes, and haemorrhage. The vascular malformations bleed spontaneously or following minimal trauma. The condition is more common in women with a ratio of 5:1 and patients usually present with significant bleeding by the age of 30 years (Harrison, 1956). Epistaxis occurs in 90 per cent of patients (Harrison and Lund, 1993), which may vary from severe bleeds, necessitating frequent blood transfusions to mild occasional bleeds, which can nevertheless affect the patient's quality of life.

The argon laser produces a beam with wavelengths at 488 nm and 514 nm that are selectively absorbed by haemoglobin. It can, therefore, be used to coagulate the nasal telangiectasia without destroying the overlying mucosa. This cannot be achieved by hot wire or chemical cautery. The aim of this study is to determine the role and efficiency of argon laser coagulation in the management of epistaxis due to hereditary haemorrhagic telangiectasia.

Method

A retrospective series of 19 patients, aged between 15 and 68 years, who had undergone argon laser treatment to the nose for recurrent epistaxis due to hereditary haemorrhagic telangiectasia from December 1991 to January 1995 was investigated. Eighteen

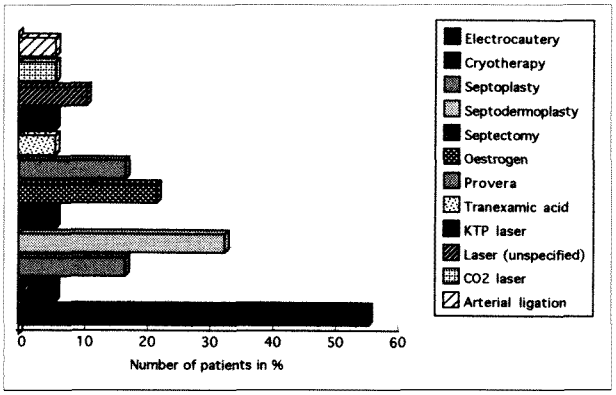

FIG. 1
Diagram showing previous treatments undergone by patients.

of these patients had tried more than one other medical and/or surgical treatment (Figure 1). These had either been unsuccessful or the side effects had been intolerable. Argon laser coagulation under general anaesthesia was performed by one of three consultants. The patients were interviewed in person or by telephone and were asked a number of questions to establish the following points:

(1) The severity of the epistaxis. This was determined by the patient. All patients had been asked to keep a diary of frequency and severity of their epistaxis prior to their first admission and between successive laser

From The Institute of Laryngology and Otology, 330 Grays Inn Road, London, UK.
Accepted for publication: 14 November 1996.

treatments. Half the patients were able to refer back to this.

(2) The number and frequency of laser treatments for each patient.

(3) Whether there was a decrease in the severity and frequency of the epistaxis after the laser, and if so, how long did this improvement last?

(4) Overall, was there an improvement in their quality of life, with particular reference to the effect on their work, social life and emotional status, as a result of the laser treatments?

The patients were interviewed between six months and two years, seven months after their last treatment.

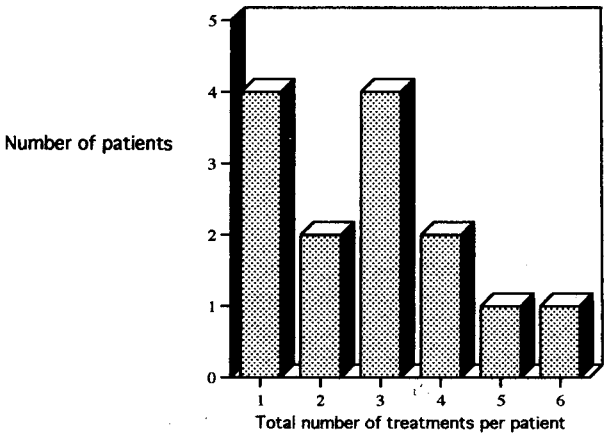

FIG. 2
Number of treatments with argon laser per patient.

Results

One patient (five per cent) stated that his epistaxis was mild and not affect his quality of life. He was the only patient who had not undergone any other treatment. Thirteen patients (68 per cent) rated their epistaxis as moderately severe, being sufficient in amount and frequency to affect their quality of life. One patient who rated his epistaxis as moderate had required two blood transfusions in the preceding 10 months. Four patients (21 per cent) were bleeding heavily every day and rated their symptoms as severe. This correlated well with a history of multiple blood transfusions. All had required one or more blood transfusions in a six month period.

Fourteen patients (74 per cent) noticed an immediate improvement in their symptoms after the first argon laser treatment. The duration of improvement determined the interval between treatments as well as the total number of treatments that the patient required, but this length of time was highly variable. The treatments were discontinued when the patient felt that the severity of their epistaxis no longer justified the inconvenience of repeated hospital admissions. Follow-up ranged from six to 31 months (mean 13.5 months). In four of these patients (21 per cent of the total of 19 patients) the improvement in their epistaxis persisted after only one argon laser treatment and no further laser treatments were required. However, one individual underwent a unilateral Young's procedure for unilateral epistaxis six months later. Follow-up for the remaining three patients is 15, 27 and 31 months respectively. Ten patients (53 per cent) required an average of three treatments before they perceived a persistent improvement in their epistaxis (Figure 2). In all except one case, the duration of improvement increased with successive laser treatments although none of the patients reported that the epistaxis had completely stopped. All of these patients had initially described their bleeding as mild or moderate and none had undergone any blood transfusions.

Thirteen of the 14 (69 per cent of the total of 19 patients) also noted a global improvement in their quality of life. The exception was one individual who after three laser treatments felt that the improvement in epistaxis was insufficient to impact on the quality of life. No complications either early or late were reported by the patients in relation to the argon therapy.

It should be noted that two out of the 14 patients were started on medroxyprogesterone acetate (Provera) following their second laser treatment. As they both underwent further argon laser treatment in addition to the Provera, the reduction in frequency and severity of their epistaxis may not be attributable to the laser alone. One of the patients on Provera has now undergone a unilateral Young's procedure three years after his last argon laser treatment that has completely stopped the nose bleeds.

Five patients (26 per cent) derived no improvement from argon laser treatments. These patients included those who rated their epistaxis as severe and the patient who had described his symptoms as moderate but who had received blood transfusions in the previous 10 months. One patient required bilateral nasal packing after both argon laser treatments and continued to bleed so heavily that he ultimately required ligation of both external carotid arteries. A second patient derived no improvement following two laser treatments and underwent ligation of the anterior and posterior ethmoidal and maxillary arteries. A third patient underwent bilateral septodermoplasty and remained on Provera. In these three patients none of the interventions stopped the epistaxis. All three individuals rated their epistaxis as moderate and one has recently undergone bilateral modified Young's procedures. A fourth patient continues to undergo regular laser treatment, although at another hospital. These provide only a temporary improvement and she continues to require frequent blood transfusions. The fifth patient has had a unilateral modified Young's procedure which stopped bleeding from that side and is now considering a septodermoplasty on the opposite side as he feels unable to tolerate bilateral nasal obstruction.

Discussion

The underlying abnormality in herediatry haemorrhagic telangiectasia is a weakened vascular wall. Hanes (1909) originally described superficial vessels lined by a single layer of endothelium, but deficient in muscular or elastic tissue that electron microscopic studies have confirmed. These venules have a single layer of endothelium on a basement membrane. Smooth muscle cells are visible, but do not form a continuous coat and there are no elastic fibres present. In addition, there are also dilatations of arterioles and capillaries forming arterio-venous fistulae. The telangiectasia can be found anywhere in the skin or mucous membranes, but it is those in the most exposed sites that tend to bleed. Another factor that determines whether these lesions bleed is the overlying epithelium (Harrison, 1964). The telangiectasia in the nose do not have a protective coat of squamous epithelium and are therefore vulnerable to minor trauma. Treatment has therefore been aimed at either improving the protective function of the overlying epithelium or reducing the trauma. Several different types of laser have been used to treat the telangiectasia. A series of 19 patients were successfully treated by the neodymium-yttrium-aluminium-Garnet laser photocoagulation (Kluger *et al.*, 1987). Although better than the CO_2 laser at coagulating blood vessels, it does cause significant tissue destruction. A second study found that the argon laser was more effective than the CO_2 (Illum and Bjering, 1988). The argon laser produces continuous coherent light with wavelengths at 488 nm and 514 nm. The beam passes through water and clear colourless structures without thermal damage and is absorbed by the complementary colour red. It is therefore possible to coagulate the telangiectasia without destroying the epithelium. The KTP/532 which has a similar wavelength (532 nm) to the argon has also been used with some success in hereditary haemorrhagic telangiectasia (Levine, 1989).

This study is clearly subjective, depending on the individual patient's assessment of the severity of their epistaxis. This, however, provided a relevant assessment as, in the non-life threatening situation, it is the patient's perception of the level of the problem and their ability to cope with it that causes them to seek help. Some objectivity was introduced when the patient was able to refer back to a diary, and by noting whether the patient had ever required a blood transfusion. The latter correlated well with the patient's own assessment. All the patients who rated their epistaxis as severe had required at least one blood transfusion in six months. Only one patient regarded his epistaxis as moderate despite requiring two blood transfusions in the preceding ten months.

Twenty-six per cent of the patients were not helped by the argon laser treatment. These are the same patients who rated their epistaxis as severe

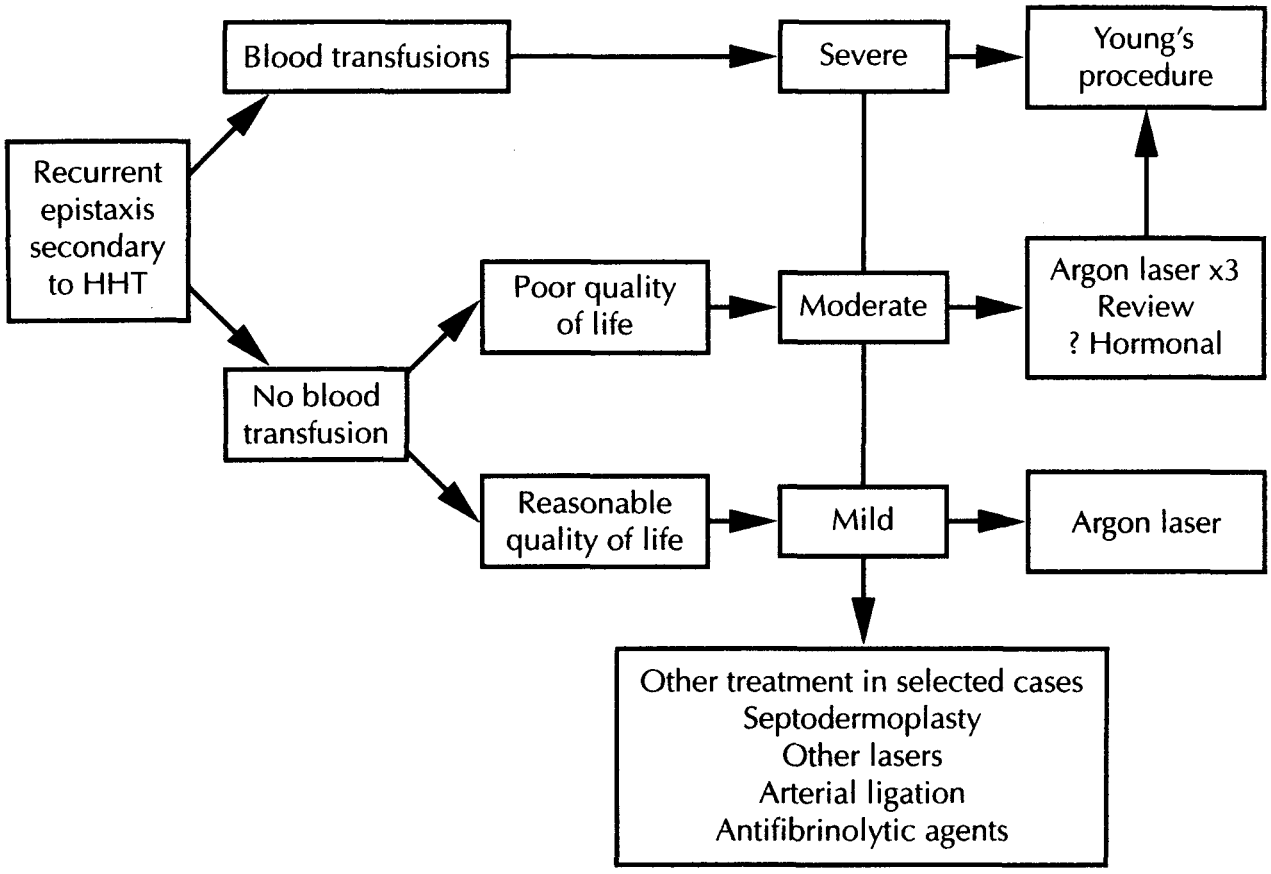

FIG. 3
Algorithm for management of hereditary haemorrhagic telangiectasia.

https://doi.org/10.1017/S0022215100136370 Published online by Cambridge University Press

and/or required at least one blood transfusion every six months. The study by Kluger *et al.* (1987) also found that those patients with severe epistaxis did not benefit from laser photocoagulation. It is suggested that those patients requiring blood transfusions are unlikely to do well and should therefore not be offered argon laser treatment.

Seventy-four per cent of patients reported an immediate improvement in their epistaxis after argon laser treatment. The length of time that this improvement lasted was, however, very variable but it appeared to increase after each session. Mean follow-up after the last argon laser treatment for this group of patients was 13.5 months and four patients required only one treatment. The other 10 patients required an average of three treatments before a persistent improvement was perceived which suggests some cumulative effect. All these patients had rated their epistaxis as mild or moderate and had not required any blood transfusions prior to treatment.

Two patients, both of whom rated their symptoms as moderate were commenced on Provera during the course of argon laser treatment, as they initially derived limited benefit from the laser treatment. The drug has been shown to be of benefit in the reduction of epistaxis in hereditary haemorrhagic telangiectasia (Van Cutsem *et al.*, 1988). In this study both patients on combined treatment felt that their quality of life improved but it is impossible to define the contribution made by each individual treatment. The rationale for Provera is the same as for systemic oestrogens, which are thought to cause squamous metaplasia (Harrison, 1982) thereby protecting the overlying mucosa. However, Provera is not without side-effects and both individuals were considering discontinuing the drug because of water retention.

This study relies on a retrospective classification of severity of epistaxis by the patient. However, the identification of two groups of patients – those with mild or moderate bleeding and those with severe bleeding requiring blood transfusions does provide an indication of therapeutic response. A more systematic approach has now been adopted based on these preliminary findings and a prospective study using a quality of life inventory modified and validated for use in rhinological patients is being utilised (Piccirillo *et al.*, 1995).

We consider that argon laser therapy has a significant role to play in the treatment of mild and moderate epistaxis caused by hereditary haemorrhagic telangiectasia. Based on these results we now offer mild to moderate cases a series of three argon laser treatments, usually at three monthly intervals (Figure 3). However, careful review is needed and some patients require more frequent treatments. It is possible that a combination of argon laser and hormone therapy (if it can be tolerated) may be complementary to one another. However, in severe cases complete closure of the nostril (modified Young's procedure) has been shown to offer optimal management (Gluckman and Portugal, 1994; Lund and Howard, 1996).

References

Gluckman, J. L., Portugal, L. G. (1994) Modified Young's procedure for refractory epistaxis due to hereditary haemorrhagic telangiectasia. *Laryngoscope* **104:** 1174–1177.

Hanes, F. M. (1909) Multiple haemorrhagic telangiectasia causing epistaxis. *Johns Hopkins Medical Bulletin* **20:** 63-67.

Harrison, D. F. N. (1956) Babington's disease, hereditary haemorrhagic telangiectasia. *Guy's Hospital Reports* **104:** 246–266.

Harrison, D. F. N. (1964) Familial haemorrhagic telangiectasia: 20 cases treated with systemic oestrogen. *Quarterly Journal of Medicine New Series* **129:** 25–38.

Harrison, D. F. N. (1982) Use of oestrogen in treatment of familial haemorrhagic telangiectasia. *Laryngoscope* **92:** 314–320.

Harrison, D. F. N., Lund, V. J. (1993) *Tumours of the Upper Jaw*. 1st Edition, Churchill Livingstone, Edinburgh.

Illum, P., Bjerring, P. (1988) Hereditary haemorrhagic telangiectasia treated by laser surgery. *Rhinology* **26:** 19–24.

Kluger, P. B., Shapsay, S. M., Hybels, R. L., Bohigian, R. K. (1987) Neodymium-Yag laser intranasal photocoagulation in hereditary haemorrhagic telangiectasis: an update report. *Laryngoscope* **97:** 1397–1401.

Levine, H. L. (1989) Endoscopy and KTP532 laser. *Annals of Otology, Rhinology and Laryngology* **98:** 46–51.

Lund, V. J., Howard, D. J. (1996) Closure of the nasal cavities in the treatment of refractory hereditary haemorrhagic telangiectasia. *Journal of Laryngology and Otology* **111:** 30–33.

Piccirillo, J. F., Edwards, D., Haiduk, A., Yonan, B. S., Thawley, J. F. (1995) Psychometric and clinimetric validity of the 31-Item rhinosinusitis outcome measure (RSOM-31). *American Journal of Rhinology* **9:** 297–306.

Van-Cutsem, E., Rutgeerts, P., Geboes, K., Van Gompel, F., Vantrappen, G. (1988) Oestrogen-progesterone treatment of Osler-Weber-Rendu disease. *Journal of Clinical Gastroenterology* **10:** 676–679.

Address for correspondence:
Professor Valerie J. Lund, M.S. F.R.C.S., F.R.C.S. (Ed.)
Professorial Unit,
Royal National Throat, Nose and Ear Hospital,
330/332 Gray's Inn Road,
London WC1X 8DA.