Can altering the pattern of laser photocoagulation for proliferative diabetic retinopathy help retain visual fields for driving?

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Laser panretinal photocoagulation has long been established as the treatment of choice for proliferative diabetic retinopathy. Meyer Swickerath[1,2] described it as a mode of treatment in the 1960s using xenon arc. Initially treatment was applied directly to areas of neovascularisation, but with increasing knowledge of the eye's response to photocoagulation and the use of ruby and argon lasers, indirect scatter patterns were also employed.

The Diabetic Retinopathy Study, which was commenced in 1971 and sponsored by the National Eye Institute in North America, looked at several aspects of photocoagulation including whether it prevented severe vision loss (<5/200) from proliferative diabetic retinopathy; whether there was a difference in efficacy and safety between argon laser and xenon arc in both focal and extensive scatter; and which stages of retinopathy benefited most from treatment (or at which stages treatment may be of no benefit or even harmful). Side-effects of photocoagulation were also investigated by this large study, which showed that a decrease in visual acuity and constricted visual field allied to the treatment were more frequently encountered in eyes treated with xenon arc than with argon laser.[3]

Since that groundbreaking study other side-effects and complications including decreased night vision,[4] colour vision,[5] glare,[6] temporary loss of accommodation,[4] choroidal neovascularisation[7] and macular oedema[8] have been described in relation to both treatments with xenon arc and argon laser photocoagulation.

Techniques and parameters in treatment have altered somewhat since the 1970s. Heavy burns often associated with earlier treatments are more likely to reduce the visual field, especially with the xenon arc photocoagulator.[3,9] These tended to destroy all the retinal layers whereas the effects of lighter xenon, argon laser and more recently diode laser tend to be limited to the retinal pigment epithelium, thus reducing the risk of constricted visual fields and decreased night vision.

Confluent burns or retreatment over previously treated areas are more likely to have a detrimental effect on peripheral vision.

The constriction of visual fields attributable to laser alone that occurs as a complication of panretinal photocoagulation is relatively low at 19%[10] (diabetic patients can lose visual fields even without undergoing laser photocoagulation; the reason for this is unclear but may be related to subclinical microangiopathy).[11] The loss of visual fields can have catastrophic effects on the affected individual, with possible loss of independence and the ability to drive a motor vehicle.

The Driving and Vehicle Licensing Agency (DVLA) requirements for driving[11] include the ability to read a car registration plate at 67 feet (which has been shown under realistic conditions to equate to a visual acuity of 6/10),[12] along with minimum visual field of at least 120° along the horizontal and 20° above and below in the vertical meridian (as defined by the Esterman binocular visual field test).[13]

Most authorities would not initially take into consideration preservation of visual fields when undertaking panretinal laser photocoagulation, preferring to control the neovascularisation process as the main priority.

An adequate panretinal laser ablation should have a minimum of 2000–3000 burns applied with a spot size of 200–300 μm and placed at intervals of one spot apart.[14] (Closer placement of burns may eventually result in a confluent appearance with enlargement of the spots over time.[15]) Supplemental laser may be necessary to invoke regression. This may also result in a confluent appearance to the treatment.

With altering patterns in scatter laser photocoagulation over the years, some operators will avoid the temporal and nasal retina initially with a view to limiting the

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damage to the visual fields, only treating these areas if supplementary laser treatment is required.

Past experience suggests that the central versus peripheral patterns of laser photocoagulation are not significantly different in terms of the improvement in retinopathy status.[16] Mathematically calculating and tailoring the areas for panretinal photocoagulation in individual eyes with proliferative diabetic retinopathy is a novel and intriguing way of attempting to preserve the driving visual field (as suggested by N. Davies[17] in this issue of *Eye*). This does not, however, take into account areas of capillary non-perfusion as a possible stimulus for neovascularisation and assumes that it is the total number of burns that is of more importance than specifically treating areas of ischaemia.

To fully realise the possible benefits of altering the pattern of panretinal photocoagulation, a randomised controlled clinical trial would be appropriate. In practical terms it may well be difficult to control retinal neovascularisation due to diabetes in any particular pattern of laser photocoagulation. Any alteration in treatment pattern or parameters that potentially reduces side-effects in this chronic debilitating condition is worthy of consideration.

References

1. Meyer Swickerath G, Gerke E. Treatment of diabetic retinopathy with photocoagulation. Acta Ophthalmol (Copenh) 1983;61:756–68.
2. Meyer Swickerath G. Light coagulation. St Louis: CV Mosby, 1960.
3. DRS report no. 8. Photocoagulation of proliferative diabetic retinopathy: clinical applications of DRS findings. Ophthalmology 1981;88:583–600.
4. Hamilton AMP, Ulbig MW, Polkinghorne P. Management of diabetic retinopathy. London: BMJ Publishing, 1996.
5. Trick GL, Burde RM, Gorden MO, Santiago JV, Kilo C. The relationship between hue discrimination and contrast sensitivity deficits in patients with diabetes mellitus. Ophthalmology 1989;95:693–8.
6. Mackie SW, Barrie T, Elliot DB, MacCuish AC, Walsh G. Glare disability in diabetic patients before and after panretinal photocoagulation. Invest Ophthalmol Vis Sci 1994;35:1593.
7. Lewis H, Schachat AP, Haimann MH, *et al*. Choroidal neovascularisation after laser photocoagulation for diabetic macular oedema. Ophthalmology 1990;97:503–11.
8. DRS report no. 12. Macular oedema in diabetic retinopathy study patients. Ophthalmology 1987;94:754–60.
9. DRS report no. 4. Diabetes 1979. Proceedings of the 10th congress of the International Diabetes Federation, Vienna, Austria, September 1979:781–94.
10. Mackie SW, Webb LA, Hutchinson BM, Hammer HM, Bowie T, Walsh G. How much blame can be placed on laser photocoagulation for failure to attain driving standards? Eye 1995;9:517–25.
11. Trick GL, Trick LR, Kilo C. Visual field defects in patients with insulin-dependent and non-insulin-dependent diabetes. Ophthalmology 1990;97:475–82.
12. Road Traffic Act 1988. London: HMSO, 1988.
13. Drasdo N, Haggerty CM. A comparison of the British number plate and Snellen vision tests for car drivers. Ophthalmic Physiol Opt 1981;1:39–54.
14. Form CLE 1060. DLVC Drivers Medical Branch. Revised Sept 1992.
15. Schatz H, *et al*. Progressive enlargement of laser scars following grid laser photocoagulation for diffuse diabetic macular oedema. Arch Ophthalmol 1991;109:1549–51.
16. Blankenship GW. A clinical comparison of central and peripheral argon laser panretinal photocoagulation for proliferative diabetic retinopathy. Ophthalmology 1988;95:170–7.
17. Davies N. Altering the pattern of panretinal photocoagulation: could the visual field for driving be preserved? Eye 1999;13:531–6.