

Port-wine Stain Treatment is Wavelength Independent in the Range 488-620 nm using 200-ms Pulses

W. VERKRUYSSE^a, M.A. TRELLES^a, J.W. PICKERING^b, M. VÉLEZ^a, J. RIGAU^a

^a Instituto Médico Vilafortuny, E - 43850 Cambrils, Spain

^b Laser Centre, Academic Medical Centre, Amsterdam, The Netherlands

Correspondence to Dr M.A. Trelles MD PhD

Paper received 6 June 1993

Abstract. To investigate the possible wavelength dependency of vascular damage to port-wine stains, an argon pumped dye laser was used at wavelengths varying from 488 to 620 nm with a spot size of 1 mm diameter, power density of 127 W cm^{-2} and pulse length of 200 ms. Clear differences in tissue reactions for different wavelengths could not be observed, either clinically or histologically. During the relatively long exposures a considerable part of the heat produced in the vessel is conducted away to the surrounding tissue. Additionally, dermal damage results from heat conducted from the epidermis. These effects probably override the effect of changing the wavelength.

INTRODUCTION

Port-wine stains (PWS) are congenital vascular disorders of the skin, consisting of ectatic dermal blood vessels (1). Laser therapy of PWS is based upon selective heating and consequential damage to the ectatic blood vessels through selective absorption of light by oxyhaemoglobin, without heating other skin constituents to a degree in which scarring or other complications occur (2).

The argon laser has been used by many clinicians to successfully treat PWS (3-6). Although the principal wavelengths (488 and 514 nm) are selectively absorbed by oxyhaemoglobin and therefore may be expected to damage vessels selectively, these wavelength lines are not considered to be ideal. A competing absorber, the chromophore melanin in the epidermis, absorbs these lines relatively well compared to longer wavelengths. A considerable proportion of the incident light is absorbed by the melanin and produces heat which conducts to the superficial dermis causing non-selective damage, thus increasing the risk of hypertrophic scarring.

A combination of relatively low epidermal absorption and high absorption by oxyhaemoglobin is achieved at 577 nm (yellow light) and this is therefore expected to be the optimal wavelength (7, 8). As a consequence, many new

techniques have been developed at or near this wavelength (2, 8-12) and histological studies have shown it to produce more blood vessel selective damage than the argon laser, on condition that the yellow light is delivered by pulsed dye lasers (13, 14). The argon laser is essentially a continuous wave laser.

In 1990 Tan et al proposed 585 nm as the new wavelength of choice on the basis of experiments with a pulsed dye laser (pulse length 0.45 ms). These experiments showed that at 585 nm vascular damage was caused at greater depths compared to those achieved when using 577 nm (15). A scientific explanation, based on the difference in absorption by oxyhaemoglobin at those wavelengths, was recently given by Pickering & van Gemert (16) and Verkruysse et al (17). Clinically, treatment with this new choice of wavelength produced better PWS clearance in PWS that had been treated previously with 577 nm (15, 18).

The question arises as to whether a wavelength dependency for vascular injury depth, as shown with the pulsed dye laser, can also be found when using an argon pumped tunable dye laser. Such a laser has in the past been used to treat PWS (10, 11, 19, 20). The goal of this research was to provide the answer to this question by comparing histology from PWS, irradiated with laser light at various wavelengths, using an argon pumped dye laser.

MATERIALS AND METHODS

A Coherent argon laser (Supergraphite CR-18, Coherent Ltd, USA) was used to pump an Excell tunable dye laser (Excell 350, Excell Inc, CA, USA) equipped with rhodamine 6G dye and a quartz fused dye jet. The light was focused into a 200- μm fibre, to a spot size of 1 mm diameter on the PWS. The laser power was measured at the end of the fibre using a Lambda Coherent power meter (Labmaster LM-E, Coherent Ltd, USA). The dye laser was tuned to the following wavelengths: 575, 585, 590 and 620 nm, each with a 2-nm bandwidth.

The argon laser power was adjusted to give 1 W (± 0.05 W) at the output end of the fibre for each wavelength. This power corresponds to an irradiance of 127 W cm⁻². Combining the power density with the pulse length of 200 ms leads to a radiant dose per exposure of 25.4 J cm⁻². To be able to irradiate at the wavelengths 488 and 514 nm, the argon laser was used in mono-line mode. Test spots were also irradiated with the argon laser in multi-line mode (488–514 nm). An optical shutter was placed between the argon laser and the dye laser to produce 200-ms exposures.

Three adult patients were selected for the trials, with port-wine stains as macroscopically similar as possible. Each of these patients, having agreed on the conditions of the taking of biopsies, was locally injected with an anaesthetic [Mepivacaine (2%) without vasoconstrictor]. One cm² was irradiated in adjacent spots, using each wavelength mentioned above. All trials with different wavelengths were performed in the same lesion (obviously in different areas but with no significant macroscopical differences). Biopsies from the test spots were taken immediately, 1 or 2 weeks and 1 month after treatment. The biopsies were preserved in formol (40% formaldehyde) until investigated, and stained with haematoxylin. The pathologist interpreting the slides was not told which biopsy corresponded to which wavelength and he examined two samples from each biopsy.

RESULTS

The most important result is that no significant difference between the histology at each wavelength was observed. Therefore, for completeness, we give the following brief,

wavelength independent, description of the histology.

Biopsies, taken immediately after treatment [Fig. 1(a, b)] showed destruction of the epidermis. Vascular damage was non-specific and limited to the superficial dermis. Some vessels appeared smaller and the vessel wall was partially destroyed, especially those vessels located at depths varying from 0.4 to 0.8 mm.

One week after treatment [Fig. 2(a, b)] the epidermis had not recovered. Some vessels showed thrombosis and vessel wall destruction with extravasation of erythrocytes. Fibrotic tissue was formed in the superficial dermis.

Two weeks following treatment, the epidermis showed signs of recovery, the papillary dermis was dense and fibrotic. Around and within some blood vessels there was dense fibrotic tissue, showing the so called hyalinisation phenomenon (Fig. 1).

One month after treatment [Fig. 3(a, b)] it could be seen that the epidermis was well recovered, and had well-constituted tissue. Collagen was dense and fibrotic at the superficial dermis. However, some ectatic vessels still remained, particularly in the deeper dermis.

These histological results are consistent with those described for similar large pulse length argon laser treatment (13, 21).

The clinical appearance of a treated PWS after 1 month showed no significant difference between spots irradiated at the various wavelengths (Fig. 4).

DISCUSSION

The histology and clinical results show that PWS tissue reaction to laser light irradiation with a long exposure (200 ms), 1 mm spot diameter, and power density conducive to immediate 'minimal' whitening (127 W cm⁻²), is not wavelength dependent in the wavelength region from 488 to 620 nm. No difference in the depth of vascular injury could be observed. The histological results were comparable with those found by other authors using the argon laser (13, 21) or argon pumped dye laser at 577 nm with long pulse lengths (22). The mechanism that coagulates vessels at greater depths with 585 nm compared to 577 nm seems not to play a significant role in the experiments reported above.

Greater depths of vascular injury achieved at 585 nm compared to 577 nm using a 5 mm

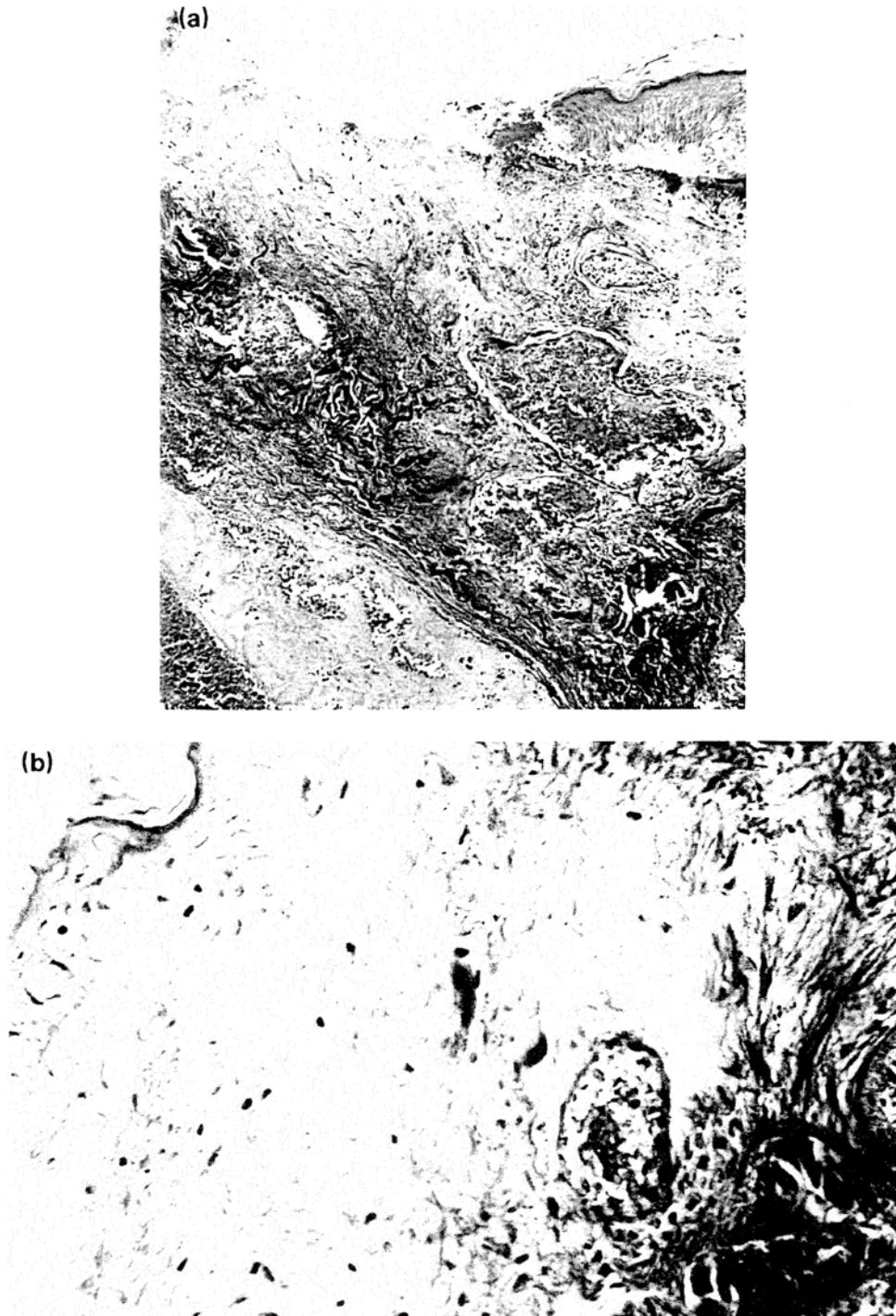


Fig. 1. Biopsies, taken immediately after (a) 575 nm and (b) 620 nm laser treatment, showed epidermis destruction and vascular damage limited to the superficial dermis which also appears destroyed. Some vessels appeared smaller with their walls partially damaged.

beam diameter are explained in terms of light absorption by oxyhaemoglobin as it is the only parameter differing significantly (7, 15). The spot size of short pulse dye lasers is usually 5 mm in diameter. Monte Carlo simulations showed the fluence rates at a certain depth in

the tissue increase when the beam diameter increases and the irradiance is kept equal (23). A large spot size (3–5 mm diameter) is thus preferred to a small spot size. The power produced by the argon pumped dye laser is too low to have spot sizes much larger than 1 mm

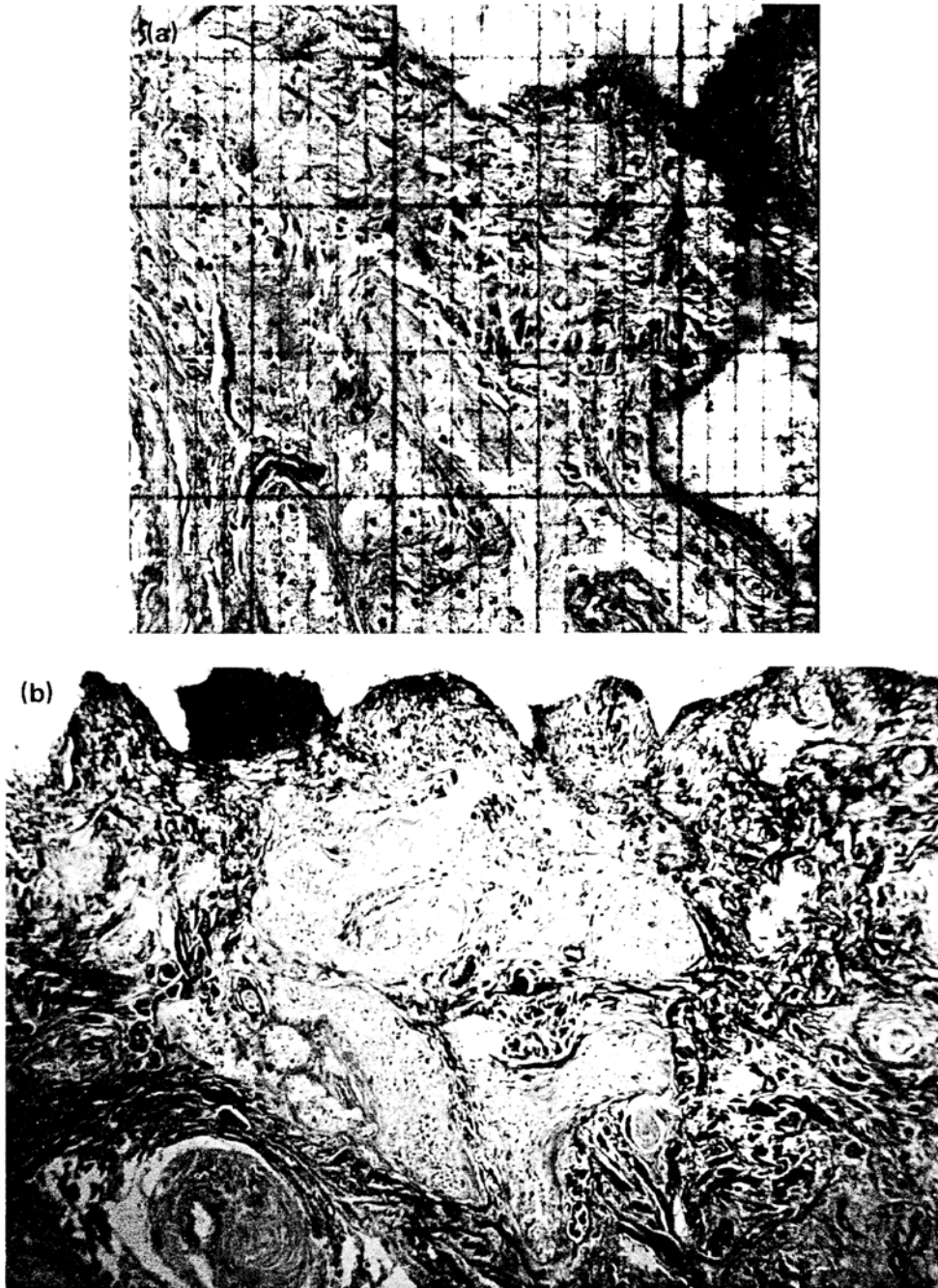


Fig. 2. Port-wine stain sample 1 week after (a) 575 nm and (b) 620 nm laser treatment showed tissue constitution not yet recovered. Some vessels present thrombosis phenomenon and vessel walls are destroyed. Erythrocytes are extravasated and images of fibrotic tissue are formed in the superficial dermis.

diameter while maintaining the desired irradiance.

Although the difference in wavelength between 577 and 585 nm is only 8 nm, the associated absorption coefficients of oxyhaemoglobin differ considerably. At 585 nm this coefficient is around 190 cm^{-1} and at 577 nm around 350 cm^{-1} (7). A considerable amount of light reaching a vessel is scattered light. At

585 nm, vessels around a 'target vessel' absorb less scattered light, compared to 577 nm. Therefore, more light reaches deeper vessels. The higher fluence rates that are achieved at the target vessel because of this effect appear to compensate for the fact that the target vessel itself absorbs the 585 nm light less efficiently too.

The important difference between irradiation

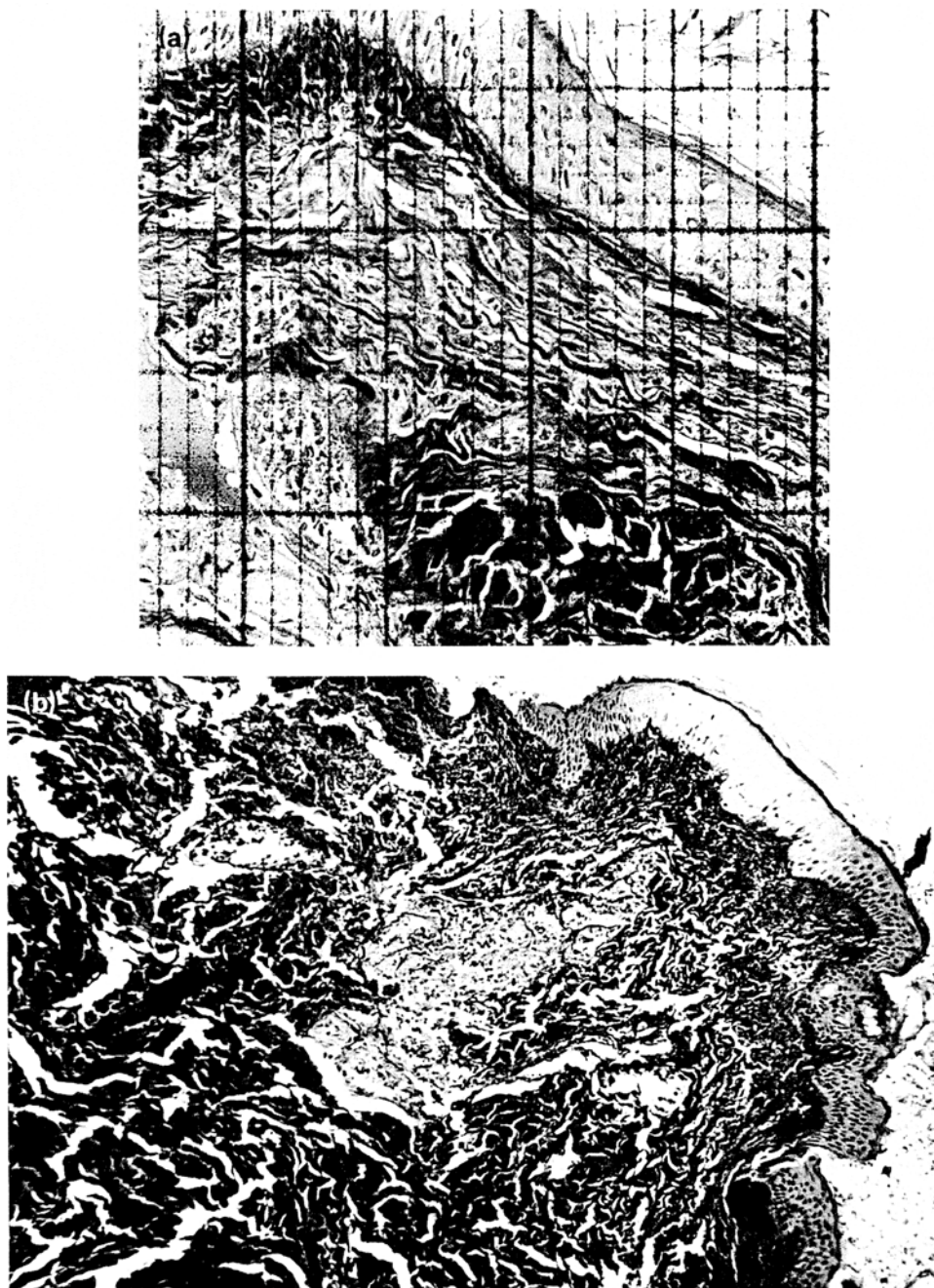


Fig. 3. One month after (a) 575 nm and (b) 620 nm laser treatment, the epidermis appears well recovered and reconstituted. Collagen is dense and fibrotic at superficial dermis level. Some ectatic vessels can be seen in the deeper dermis.

tion with a pulsed dye laser and irradiation from the argon pumped dye laser as described above is the pulse length. Pulsed dye lasers, available for medical use, produce very short pulses of 450 ms at a very high power (typically in excess of 1000 W), whenever the continuous wave delivery is by a short, low peak power exposure.

The limited power deliverable on the tissue per pulse by an argon pumped dye laser, which

is essentially a continuous wave system in combination with an optical shutter, restricts the pulse length to a minimum of around 100 ms. Recently, some systems have enabled a pulse as short as 30 ms with a hexascan hand-piece (24). Shorter pulses would be expected to give better results. Different theoretical models indicated optimal pulse lengths to be between 1 and 10 ms (2, 7, 25–27). These are optimal, because the pulse length is short

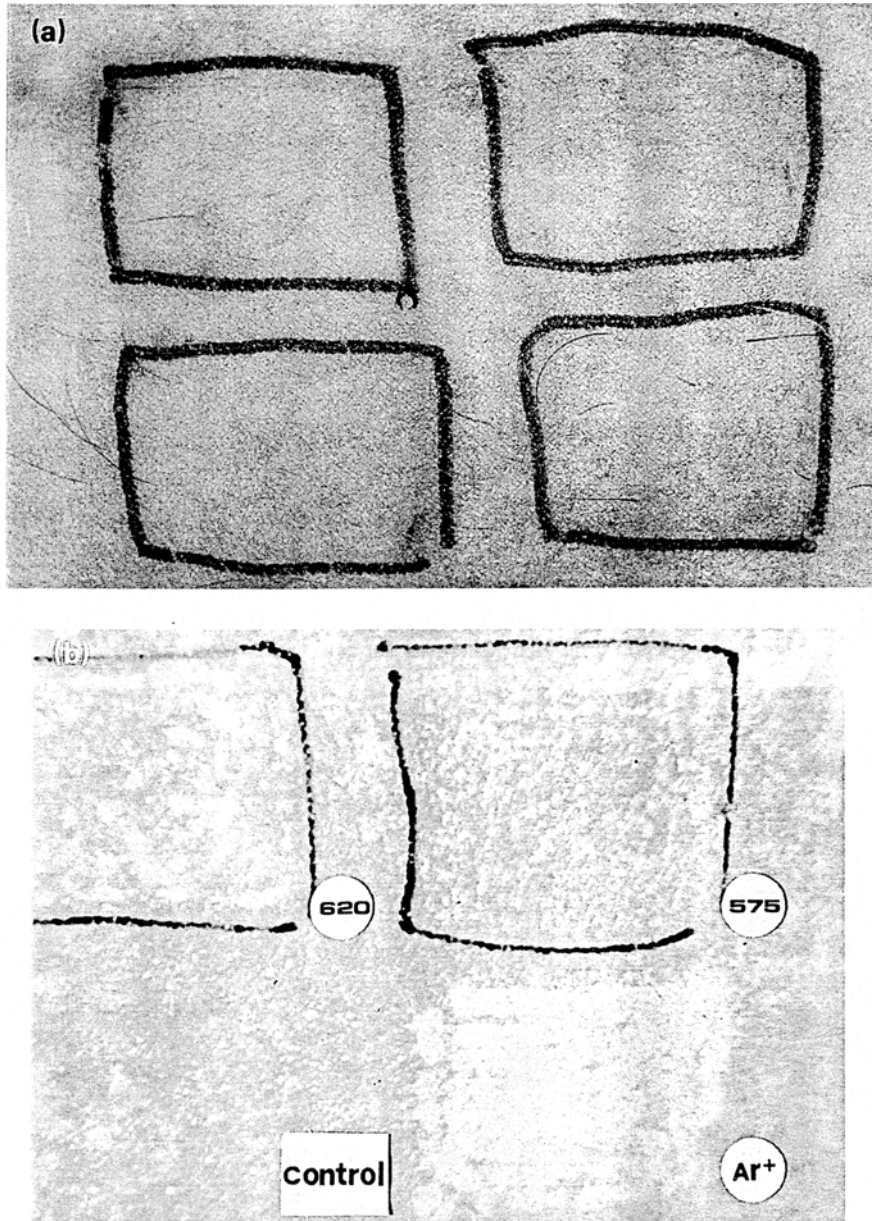


Fig. 4. (a) Port-wine stain before 620, 575 nm and argon multi-line test. The same protocol for every laser emission was tested. Similar characteristic conditions of the lesion were observed. (b) Aspect 1 week after treatment. Note the blanching at the area treated by the argon laser (Ar^+). Results here do not correspond to PWS blanching, but to epidermis destruction and necrosis. No particular differential reactions can be seen in the samples corresponding to 575 and 620 nm.

enough to confine enough generated heat to the vessel and its immediate surroundings to damage these structures. Other structures do not reach a high enough temperature to be irreversibly damaged.

The pulse length of 200 ms is 20–200 times longer than the optimal pulse length. A great deal of the heat produced in the vessels is conducted away to surrounding tissues. Therefore, changing the absorption coefficient of blood by changing the wavelength of the irradiated light does not alter the heat distribution

significantly. During a 200-ms pulse, more heat is produced in the vessel at 577 nm (absorption coefficient $\pm 354 \text{ cm}^{-1}$) than at 590 nm (absorption coefficient $\pm 69 \text{ cm}^{-1}$), for example. Light of longer wavelengths (585–620 nm) compared to 577 nm is less absorbed in the upper part of the dermis. Therefore the fluence rate is less attenuated through absorption and will diffuse through scattering to deeper parts of the dermis. Although the light is absorbed less at 620 nm, it will be absorbed eventually. Using the longer

wavelengths, therefore, it is expected that the 620 nm light is absorbed in relatively deeper blood vessels compared with 577 nm. But the loss of heat by conduction appears to override this effect, as the damage to the vessels does not show significant differences at different wavelengths.

When considering light absorption, melanin, a pigment in the epidermis, is the main competing absorber in the skin. Heat produced in the epidermis because of this chromophore will be conducted to the dermis and can cause thermal damage which may heal as a scar. Skin surface temperatures during argon laser therapy with pulse lengths of 200 ms and 1 W power (as in our experiments) were measured to be up to 50 °C (27). This so called 'iron heater effect' can be greatly reduced when using pulse lengths between 1 and 10 ms (7).

Another effect of the absorption of light in the epidermis is that the intensity of light penetrating into the dermis is decreased and therefore the desired effect, damaging the vessels through a photothermal effect, is also decreased.

Light absorption in the epidermis, and therefore the heat production, is approximately 30% greater at the shorter wavelengths (488–514 nm) than at the longer wavelengths (577–590 nm). This appears not to have resulted in significantly more damage in the epidermis or in the upper dermis when those shorter wavelengths are compared to the longer wavelengths. Part of the epidermal heat vaporized the epidermis and the other part of the heat was conducted into the dermis. A wavelength dependency is not apparent because of the same arguments as described above for heat distributions in and around the blood vessels. Heat conduction overrides wavelength dependency at these long pulse lengths.

It must be concluded that the new wavelength of choice, 585 nm, is only significantly increasing the depth of vascular injury when used in conjunction with a short pulse (around 0.45 ms). The argon pumped dye laser cannot produce short high power pulses. The argon pumped dye laser always produces less power (typically up to 3 W for yellow light) than the argon laser itself (with this laser a maximum of 27 W can be reached). The power 'lost' in the dye laser may be better used to decrease the pulse length whilst maintaining similar energies per pulse in argon laser treatment at 488 and 514 nm.

ACKNOWLEDGEMENTS

The financial support of the 'Fundacion Antoni de Gimbernat' E-43850, Cambrils/Tarragona, and the professional aid of the Anatomic-pathologist Dr E. Mayayo is greatly appreciated.

REFERENCES

- 1 Barsky SH, Rosen S, Geer DE, Noe JM. The nature and evolution of portwine stains: a computer-assisted study. *J Invest Dermatol* 1980, **74**:154–7
- 2 Anderson RR, Parrish JA. Microvasculature can be selectively damaged using dye lasers. *Lasers Surg Med* 1981, **1**:263–76
- 3 Goldman L, Dreffer R, Rockwell RJ, Perry E. Treatment of port wine marks by an argon laser. *J Dermatol Surg* 1976, **2**:385–8
- 4 Arndt KA. Treatment techniques in argon laser therapy. *J Am Acad Dermatol* 1984, **11**:90–7
- 5 Sheenan-Dare RA, Lanigan SW, Cotterill JA. Argon laser treatment of port wine stains: comparison of the effects of 0.2 s and 1 s pulse duration. *Lasers Med Sci* 1989, **5**:271–6
- 6 Noe JM, Barsky SH, Geer DE, Rosen S. Port wine stains and the response to argon laser therapy: successful treatment and the predictive role of color, age and biopsy. *Plast Reconstr Surg* 1980, **2**:130–6
- 7 van Gemert MJC, Welch AJ, Amin AP. Is there an optimal laser treatment for port wine stains? *Lasers Surg Med* 1986, **6**:76–83
- 8 Lahaye CTW, van Gemert MJC. Optimal laser parameters for port wine stain therapy: a theoretical approach. *Phys Med Biol* 1985, **30**:573–87
- 9 Tan OT, Stafford TJ. EMLA for laser treatment of port-wine stains in children. *Lasers Surg Med* 1992, **12**:543–8
- 10 Cotterill JA. Preliminary results following treatment of vascular lesions of the skin using a continuous wave tunable dye laser which emits at 577 nm. *Clin Exp Dermatol* 1986, **11**:628–35
- 11 Lanigan SW, Cartwright P, Cotterill JA. Continuous wave dye laser therapy of port wine stains. *Br J Dermatol* 1989, **21**:345–52
- 12 Pickering JW, Butler PH, Walker EP, van Halewyn CN. Copper vapour laser treatment of port wine stains and other vascular lesions. *Br J Plast Surg* 1991, **43**:273–82
- 13 Greenwald J, Rosen S, Anderson RR et al. Comparative histological studies of the tunable dye laser at 577 nm and argon laser: the specific vascular effects of the dye laser. *J Invest Dermatol* 1981, **77**:305–10
- 14 Walker EP, Butler PH, Pickering JW et al. Histology of port wine stains after copper vapour laser treatment. *Br J Dermatol* 1989, **121**:217–33
- 15 Tan OT, Morrisson P, Kurban AK. 585 nm for the treatment of port wine stains. *Plast Reconstr Surg* 1990, **86**:1112–7
- 16 Pickering JW, van Gemert MJC. 585 nm for the laser treatment of portwine stains: a possible mechanism. *Lasers Surg Med* 1991, **11**:616–8
- 17 Verkruysse W, Pickering JW, Keyzer et al. Modeling the effect of wavelength in pulsed dye laser treatment of portwine stains. *Appl Optics* (in press)

- 18 Hayashi H, Yasuda Y, Tsukada S. Flashlamp pumped dye laser treatment of port wine stains: the comparative effects of SPTL-1p (577 nm) and SPTL-1 (585 nm). *J Jpn Soc Laser Med* 1988, **9**:459-62
- 19 Scheibner A, Wheeland RG. Argon pumped tunable dye laser therapy for facial port wine stain hemangiomas in adults: a new technique using small spot size and minimal power. *J Dermatol Surg Oncol* 1989, **15**:277-81
- 20 Henning H, van Gemert MJC. Port wine stain coagulation experiment with a 540 nm continuous wave dye laser. *Lasers Surg Med* 1983, **2**:205-10
- 21 Tan OT, Carney JM, Margolis R et al. Histologic responses of port wine stains treated by argon, carbon dioxide, and tunable dye lasers. *Arch Dermatol* 1986, **122**:1016-22
- 22 Landthaler M, Haina D, Brunner R et al. Effects of argon, dye, and Nd:YAG lasers on epidermis, dermis, and venous vessels. *Lasers Surg Med* 1986, **6**:87-93
- 23 Keyzer M, Pickering JW, van Gemert MJC. Laser beam diameter for port wine stain treatment. *Lasers Surg Med* 1991, **11**:601-5
- 24 Rotteleur R, Mordon S, Buys B et al. Robotized scanning laser handpiece for the treatment of port-wine stains and other angiodyplasia. *Lasers Surg Med* 1988, **8**:283-7
- 25 Pickering JW, Butler PH, Ring BJ, Walker EP. Thermal profiles of blood vessels heated by a laser. *Australas Phys Eng Sci Med* 1989, **12**:11-5
- 26 Pickering JW, Butler PH, Ring BJ, Walker EP. Computed temperature distributions around ectatic capillaries exposed to yellow (578 nm) laser light. *Phys Med Biol* 1989, **34**:1247-58
- 27 Shakespeare PG, Hambleton J, Carruth JAS. Skin surface temperatures during argon and tunable dye laser therapy of port wine stains. *Lasers Med Sci* 1991, **6**:29-34

Key words: Laser; Port-wine stains; Pulse length; Wavelength; Heat conduction