Histologic Comparison of Argon and Tunable Dye Lasers in the Treatment of Tattoos

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Cosmetic benefit from laser therapy of tattoos may simply be the result of thermal injury and host reparative response which remove pigment by a "slough and bury" mechanism. Tattoo pigment of 4 colors (black, white, red, and blue) was introduced into the skin of guinea pigs and studied histologically at 48 h, 7 days, 4 and 6 weeks, and 3 months. Tattoos of each color were treated with argon laser (488 and 514 nm) and tunable dye laser at 3 different wavelengths (505, 577, and 690 nm). Treated tattoos were biopsied immediately and at 48 h, 7 days, and 3 months. Selective laser absorption by the tattoo pigment was suggested by pigment-related differences in threshold doses for histologic damage. Clinical clearing of tattoo pigment correlated well with the extent of immediate epidermal and dermal necrosis and was as well associated histologically with the deposition of parallel bands of collagen fibers (i.e., scar) between the residual pigment and the overlying epidermis. "Lightening" of tattoos probably depends more on widespread necrosis, subsequent tissue sloughing, and resultant dermal fibrosis than on specific changes in tattoo pigment chemistry, morphology, physical properties, or handling by macrophages.

Most methods of tattoo removal have the disadvantages of incomplete pigment removal, the need for repeated procedures, and the inability to control the depth of destruction, excessive postoperative pain, and unsatisfactory cosmetic results [1]. In 1967, Goldman et al [2] were the first to report the successful use of lasers in removal of tattoos. They used both neodynium (1060 nm) and pulsed ruby (694 nm) lasers with success. More recently, the argon laser has been used effectively for the removal of tattoos [3–5]. The argon laser is the most widely used laser for photocoagulation of port-wine stains [4,6,7], hemangiomas, and other vascular ectasias [8,9]. The relatively low-power continuous emission lines at 488 and 514 nm are well absorbed by hemoglobin but tissue injury is not confined to blood vessels or pigment [10–12]. It is likely that granulation tissue and fibrosis occurring during repair of extensive but superficial necrosis are involved in producing the final, usually cosmetically acceptable, result [11].

It has recently been shown [12–15] that highly specific damage localized to skin microvasculature can be achieved using a pulsed organic dye laser operating at 577 nm. If the goal in laser therapy of tattoos is to achieve tissue destruction at finely controlled depths under visual control, then nonspecific superficial necrosis caused by argon or CO_2 lasers would be most effective. If, on the other hand, specific thermal or physicochemical alteration of the specific pigment is possible, other laser wavelengths and pulse durations might be chosen to specifically heat the tattoo pigment granules. For example, laser-induced changes in the pigment granule surface characteristics or particle size might change the host response to the inorganic tattoo pigment. Thermal necrosis might also be confined to a zone immediately adjacent to a tattoo particle thereby minimizing the amount of tissue destruction. It is possible that the extreme temperatures (hundreds of degrees centigrade) needed to oxidize or fragment tattoo pigment granules can be achieved just at the sites of pigment deposition without widespread local destruction.

The following is a gross and microscopic study of the use of the argon laser and the tunable dye laser at 3 different wavelengths (505, 577, and 680 nm) in the treatment of experimental tattoos of 4 colors in guinea pigs. We attempted to histologically document selective absorption of laser energy by the pigmented chromophore and to determine whether we could achieve selective photothermolysis of pigment to facilitate clearing of the tattoo.

MATERIALS AND METHODS

Following ether anesthesia, the backs of 3 albino guinea pigs were shaved and epilated with hot wax. Twenty-four hours later each animal was anesthetized with ketamine, and a professional tattoo artist employed a standard reciprocating tattoo needle to produce annular tattoos 0.4–0.6 cm in diameter on the skin of the back. On each guinea pig, 8 longitudinal rows contained 12–15 annular tattoos: 2 rows for each of the 4 tattoo pigments employed—black, blue, red, and white.

The appearance of untreated (control) tattoos of at least one color was studied histologically at 48 h, 7 days, 4 weeks, 6 weeks, and 3 months. Ketamine (general) and 1% Xylocaine (local) anesthesia was used. Tattoos were biopsied with a 4-mm trephine punch and usually contained a small portion of adjacent clinically normal skin. Specimens were fixed in 10% formalin, processed in a routine manner, and stained with hematoxylin and eosin.

At least 1 month after the tattoo pigments were injected, individual tattoo sites of each color were treated with the lasers. The argon laser (Coherent Radiation Model No. 1000) emitting radiation at both 488 and 514 nm simultaneously was focused into a 1 mm-diameter spot. The tunable dye laser (Candela Corp. Model No. MDL-250) was used at 3 wavelengths, with coumarin 504 for 505 nm, rhodamine 575 for 577 nm, and oxazine 720 for 690 nm. Dyes lasing at 505 and 577 nm were focused with a planoconvex lens (focal length = 165 mm) to a spot size of 3 mm. To achieve the required energy per unit area for the less efficient radiation at 690 nm, a 1-mm spot was used. The laser exposure settings are given in Table I.

The immediate clinical effects of laser treatment were graded and recorded as follows: 0 = no epidermal change, 1+ = evidence of epidermal charring, 2+ = significant epidermal change with epidermal slough or blister formation.

Immediately following laser treatment, a number of the treated tattoos were biopsied with a 4-mm trephine punch. Both treated and untreated tattoo was included within the same specimen and the tissue was bisected immediately to allow proper orientation. In an identical manner, biopsies of treated tattoos were studied histologically at 48 h, 7 days, and 3 months following laser treatment.

Prior to the 3-month biopsy, the degree of clinical clearing or lightening of tattoo pigment was graded and recorded as follows: 0 = no change in pigment, 1+ = pigment lightening, 2+ = significant diminution of pigment.

RESULTS

The immediate clinical effects and final results of laser treatment are detailed in Table II and an example of a final

TABLE I. *Summary of laser exposure settings utilized to treat tattoos*

	Laser power (W)	Power density (W/cm^2)	Pulsewidth (s)	Spot size (diameter, mm)	Treatment dose (J/cm^2)
Argon laser (488, 514 nm)					
A	1	125	Continuous	1	—
B	1	125	0.05	1	6.25
C	3.5	438	0.05	1	21.9
D	2.5	312	0.2	1	62.5
Dye laser, wavelength (λ, nm)					
E	505	10^6	10^{-6}	3	1
F	505	3×10^6	10^{-6}	3	3
G	505	4.7×10^6	10^{-6}	3	4.7
H	577	10^6	10^{-6}	3	1
I	577	3×10^6	10^{-6}	3	3
J	577	5.2×10^6	10^{-6}	3	5.2
K	690	10^6	10^{-6}	1	1
L	690	4×10^6	10^{-6}	1	4

At the argon laser power density of 125 W/cm^2, all tattoos were treated with the continuous wave manually swept across the tattoo (A) and also with single pulses of 0.05 s (B, 6.25 J/cm^2). Condition C was used to treat blue tattoos only and condition D was used to treat black tattoos only; all other conditions were used to treat tattoos of all 4 colors.

TABLE II. *Immediate clinical results of exposure condition and final results of tattoo treatment*

Condition	Treatment dose J/cm^2	Immediate effect			Final result		
		Black	Blue	Red	Black	Blue	Red
Argon							
A	Continuous	++	++	++	++ (Fig 1)	+	++
B	6.25	+	0	+	+	0	+^{c}
C	22	ND	++	ND	ND	+	ND
D	62.5	+	ND	ND	+	ND	ND
Tunable dye: 505 nm							
E	1	0	0	0	0	0	0
F	3	0^b	0^b	0^b	+	0	+^{c}
G	4.7	++	0^b	0^b	++	0	+^{c}
577 nm							
H	1	0	0	0	0	0	0
I	3^a	++	+	0	0	0	0
J	5.2^a	++	++	+	0	0	0
690 nm							
K	1	0	0	0	0	0	0
L	4	++	0^b	0	++	0	+^{c}

^{a} Purpura noted at these doses of 577 nm.
^{b} Although undetected clinically, histologic damage was obvious.
^{c} Lightening of tattoo pigment was subtle with red tattoo and difficult to accurately assess.

Key: *Immediate effect*
0 = No epidermal change
+ = Epidermal charring
++ = Epidermal blister and slough
ND = Not done
   Final result
0 = No loss of pigment
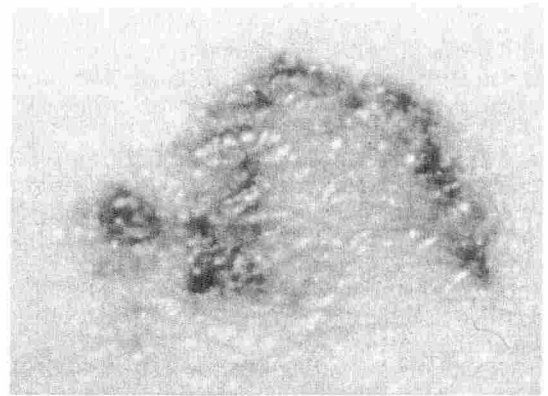+ = Slight pigment loss
++ = Significant pigment loss

FIG 1. Clinical photograph of black tattoo on guinea pig shows "lightening" of the central treated area 3 months after exposure to an argon laser operated at 1 W with continuous pulsewidth.

result is shown in Fig 1. In the white tattoos, it was not possible to clearly quantify epidermal necrosis or clearing (lightening) of tattoo pigment, and this pigment has been excluded from the table. Subtle lightening of the red tattoos was also difficult to assess due to the sparseness of the tattoo pigment (Table II).

The results of histologic studies of control tattoos and laser-treated tattoos are reviewed in detail below. Unless otherwise specified, all results are similar for the blue, black, and white pigments. Due to the sparseness of the pigment within the red tattoos, histologic evaluation was not possible.

Controls (Untreated Tattoos)

Forty-eight hours: Epidermal necrosis and ulceration with partial destruction of the underlying epidermal appendages was noted. Polymorphonuclear leukocytes were observed in the necrotic epidermis. Dermal hemorrhage and focal collagen necrosis were evident. Within the dermis, the finely granular

pigment was present primarily extracellularly. The heaviest concentration was present in numerous discrete foci running in a vertical direction ("needle tracks").

Seven days: The epidermis was completely reconstituted but showed evidence of regenerative "atypia." Individual pigment granules were dispersed at all levels of the regenerated epidermis. The epidermal appendages were also reconstituted. Within the papillary dermis, pigment was present extracellularly in bandlike array and, as well, in highly aggregated form apparently intracellularly.

Four weeks: The epidermis and epidermal appendages were normal in appearance. Vascular, myxoid-appearing granulation tissue was observed diffusely in the papillary dermis. At the lower border of the granulation tissue, pigment granules were present largely in bandlike array, and appeared to be present primarily intracellularly.

Six weeks (black only): The granulation tissue was less myxoid and less vascular in appearance. Early fibrogenesis with the appearance of plump fibroblasts and fine collagen bundles in parallel array to the overlying epidermis was present. Deep to this fibrosing process pigment granules, in largely aggregated form, were present in the upper reticular dermis.

Three months (blue only): The granulation tissue was almost entirely replaced by fibrosis; the latter appearing as fine collagen bundles in parallel array. Pigment was present in variable-sized aggregates intracellularly and extracellularly in the papillary and upper reticular dermis and clustered about vessels in the papillary and upper reticular dermis.

In absolute terms, quantitation of pigment was not possible on histologic evaluation of routinely prepared tissue sections.

However, there was an apparent diminution in the amount of pigment present between the 48-h and 7-day biopsies. As well, the pigment went from finely granular form in diffuse array at 48 h, to a more aggregated and focally concentrated appearance at 7 days. There was a slight further diminution in the amount of pigment over the ensuing 3 months. Throughout the entire process, inflammatory cell infiltration (both polymorphonuclear and mononuclear) was minimal.

Treatment with Laser

Immediate: With the tunable dye laser, a dose of 1 J/cm^2 (505, 577, 690 nm) appeared histologically to be subthreshold. Argon laser-treated tattoos were biopsied only if clinically above threshold. The histologic changes observed immediately following all laser treatments were qualitatively similar except as noted. Threshold exposures with all lasers produced subepidermal blister formation with more extensive epidermal necrosis seen following treatment with the argon laser (Fig 2) and the highest doses of the tunable dye laser. The papillary and superficial reticular dermis demonstrated focal homogenization and basophilia of collagen with necrosis of some blood vessel walls. Pigment was present in finely granular and dispersed form within areas of collagen necrosis (Fig 2) while pigment deep to the areas of dermal collagen necrosis was unaffected, being present in large and small aggregates primarily within macrophages.

The tunable dye laser at 577 nm (3 and 5.2 J/cm^2) produced more extensive vascular injury. Hemorrhage was prominent. Both within and at the periphery of areas of collagen necrosis, vessels contained numerous polymorphonuclear leukocytes within and surrounding vessel walls, but fibrinoid necrosis of vessel walls was not observed. Histopathologic changes with the tunable dye laser at 680 nm (4.0 J/cm^2) were seen only in the black and blue tattoos.

Forty-eight hours (excluding subthreshold doses): Tattoos treated by all lasers revealed subepidermal blisters overlying necrotic papillary dermal collagen. There was focal necrosis of the overlying epidermis, and some sections revealed "regeneration" of the epidermis by "ingrowth" from the surrounding undamaged epidermis and appendages (Fig 3A,B).

The histologic appearance of tattoos treated with the tunable dye laser at 577 nm revealed more prominent vascular injury including focal endothelial cell necrosis, and the presence of polymorphonuclear leukocytes within and around vessel walls.

Seven days (excluding subthreshold doses): There was complete or nearly complete regeneration of the epidermis and

FIG 2. Black tattoo biopsied immediately after treatment with argon laser (power = 1 W, pulsewidth = continuous) reveals epidermal and superficial dermal necrosis with subepidermal blister formation; upper portions of some hair follicles are also necrotic. Finely granular pigment is present in dispersed form in necrotic dermal collagen. Hematoxylin and eosin; × 100.
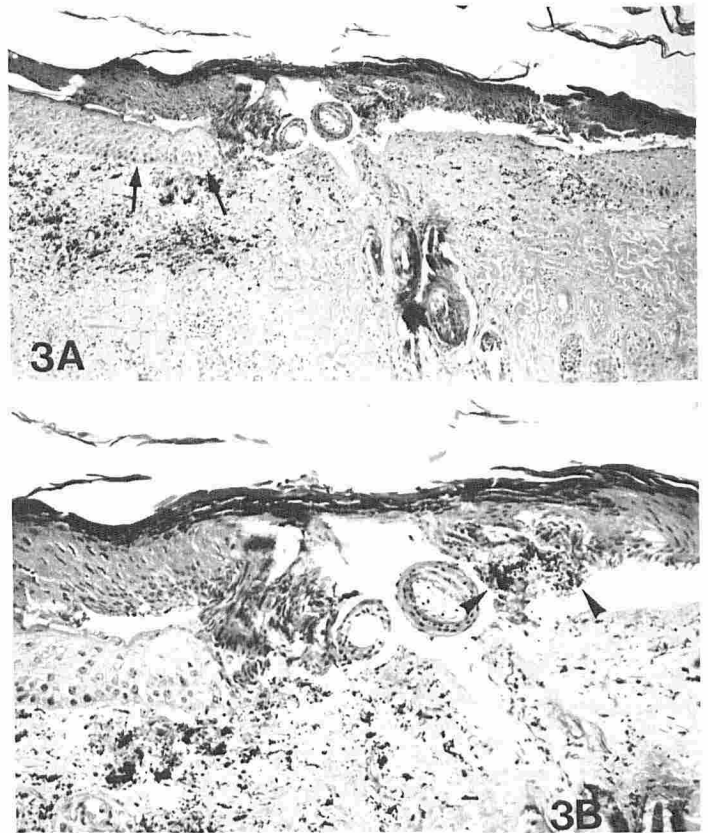

FIG 3. *A*, Black tattoo biopsied 48 h after treatment with argon laser (power = 1 W; pulsewidth = continuous) shows focal full-thickness epidermal necrosis, superficial dermal necrosis, necrosis of some appendages, and non-necrotic epidermis apparently reepithelializing the wound (*arrows*). Hematoxylin and eosin; × 100. *B*, Tattoo pigment aggregates are present in necrotic material in blister cavity (*arrowheads*). Dispersed pigment, present in necrotic collagen in blister floor, presumably will also be extruded in healing process. Hematoxylin and eosin; × 160.

cutaneous appendages. There were a few foci of residual collagen necrosis with hemorrhage. In areas of granulation tissue response, pigment was present in finely granular and dispersed array. Below these areas pigment was present primarily within macrophages in bandlike array.

Three months (includes tunable dye laser at highest doses only and argon settings which showed clearing of tattoo pigment) (see Table II and Fig 1): The granulation tissue response was no longer observed. The papillary dermis was widened by fibrosis which also involved the upper reticular dermis. Within these areas of fibrosis, there were numerous plump fibroblasts and thin collagen bundles running parallel to the overlying epidermis. Pigment was present primarily within macrophages in linear array at the junction of the fibrotic and nonfibrotic reticular dermis. In the untreated portions of the biopsies, the pigment was present primarily within macrophages surrounding vessels at the papillary-reticular dermal junction. Thus, there was a clear difference between the location of pigment in the treated and untreated portions of the biopsies; in the treated portions, the pigment was present at a deeper level within the dermis (Fig 4A,B).

Tattoos treated with the tunable dye laser at 577 nm showed much less prominent although qualitatively similar changes. At 505 nm only the black tattoo biopsy revealed the findings described above.

DISCUSSION

In 1927, Guillaume showed that insoluble tattoo pigment injected into the dermis gradually migrated to a perivascular

FIG 4. *A*, Biopsy of black tattoo 3 months after treatment with argon laser (power = 1 W; pulsewidth = continuous). Pigment is present primarily in macrophages in linear array at lower border of dermal fibrosis (scarring) (*arrowheads*). Hematoxylin and eosin; × 64. *B*, Higher-power view of right margin of fibrotic area in (*A*) shows pigment-laden macrophages arranged in linear array at junction of fibrotic and nonfibrotic dermis (*arrowheads*). Hematoxylin and eosin; × 160.

position and formed aggregates. Pigment is both engulfed by macrophages and lies free within the dermis [16,17]. Ultrastructural examination of rabbit skin injected with black tattoo pigment has shown that after 3–5 days the pigment begins to distribute throughout tissue and inflammation with polymorphonuclear leukocytes and monocytes occurs. After 6 days the pigment lies almost exclusively within macrophages. After 10 days phagocytosis of pigment by fibroblasts also occurs and after 13 days pigment begins to coalesce [18].

In the present study we have demonstrated that at 48 h, underlying a largely necrotic epidermis, finely granular pigment is present apparently both intracellularly and extracellularly within the dermis as well as in vertical array at sites of injection. At 7 days, the epidermis and epidermal appendages have largely reconstituted and within the papillary and upper reticular dermis pigment-laden macrophages are present primarily in bandlike array below the granulation tissue response. At 4 weeks, the granulation tissue response is diminished, and below this granulation tissue, pigment remains in bandlike array. At 6 weeks, the granulation tissue is actively being replaced by fibrosis and by 3 months, the granulation tissue has been entirely replaced by fibrosis. Beneath this fibrosis, and about vessels of the papillary and reticular dermis, pigment is present in variably sized aggregates intracellularly and extracellularly. As we shall discuss, qualitatively similar changes are observed in the lightening of tattoos during repair which followed laser treatment.

In 1967, Goldman et al [2] successfully used neodynium and pulsed ruby lasers for the treatment of tattoos. Biopsies of laser-treated tattoos showed nonspecific superficial and deep necrosis with retention of some of the tattoo material deep in the dermis. After healing, biopsies showed dermal fibrosis with retention of a few scattered fragments of tattoo particles. No morphologic differences were observed between the pigment masses found in the treated tattoo areas and untreated tattoo controls. Thus, the residual color of the remaining tattoo probably resulted from retention of pigment but with light transmission to and reflectance from the pigment modified by the fibrotic tissue [2].

Several later studies have investigated treatment of tattoos with the Q-switched ruby laser which provided a shorter pulse duration (10–20 ns) than the pulsed ruby laser (1.8 ms) [19–21]. Laub et al [20] showed that when low doses of the Q-switched ruby laser (<5.6 J/cm^2) were used to treat blue and black tattoos, biopsies at 7 and 12 days showed marked depigmentation. The epidermis was intact and the dermis was free of degenerative changes of the appendages, inflammation, and vascular injury. Biopsies performed 3 months later showed absence of pigment and no evidence of old thermal injury or radiodermatitis [20]. Complete eradication of tattoo pigment was seen at 3 months without the scarring seen with the longer pulses of ruby laser [20,21].

Laub et al [20] also showed that if higher doses (>5.6 J/cm^2) of Q-switched ruby laser were used, biopsies at 7 days showed a subepidermal blister similar to a second-degree burn. In addition, a "mild acute vasculitis" was noted in the dermis. In the epidermis, they noted swelling of epidermal nuclei, vacuolization of basal cell cytoplasm, and some areas of epidermal coagulation necrosis. Biopsies taken at 3 months revealed a thinned epidermis, moderate dermal fibrosis, and a mild "round cell" infiltrate [20].

Despite such optimistic preliminary reports [19–21] of the value of ruby laser in tattoo removal, subsequent research has shown it to be ineffective in practice due to the small target areas attainable and the risk of coagulation necrosis to the surrounding tissue [22,23]. More recently, the argon laser (488 and 514 nm) has been used effectively for the removal of tattoos [3–5]. The limitations of argon laser treatment of tattoos include 100% incidence of change in skin texture, 20% incidence of hypertrophic scar, and 75% incidence of "ghost" or residual pigmentation [24]. Histologic studies of tattoos following argon laser treatment have demonstrated the disappearance of pigment-laden macrophages from the upper dermis, regeneration of a normal epidermis, and the retention of undamaged epidermal appendages [24].

The precise mechanisms by which the repair of thermally necrosed tissue accounts for the removal of dermal tattoo pigment is poorly understood. Goldman et al [2] proposed that the mechanism which causes tattoo removal involves localized vaporization of the pigment particles which in a gaseous form are then expelled from the tissues as part of the "laser reaction plume." Secondary loss of tattoo pigment was then thought to occur by being "washed out" of the wound during the "serous phase." Finally, there was thought to be further dye removal during healing, possibly due to macrophage activity [2]. Beacon and Ellis [25] have shown that with CO_2 laser-treated tattoos, the early serous discharge and crusty scabs were heavily loaded with carbon dye. Skin appendages such as hair follicles and sweat glands are relatively spared and aid rapid epidermal regeneration [25].

It was initially thought that laser light might be specifically absorbed by the tattoo pigments, heating them and their immediate surroundings and eliciting an inflammatory response that in some way removed or obscured the pigment [21]. This may in part be the case; however, the large variety of lasers and exposure conditions which have been reported to successfully treat tattoos suggests that diffuse tissue necrosis rather than specific absorption by the tattoo pigment is the common event in successful treatments [26].

The clinical usefulness of selective laser photon absorption

in the treatment of tattoos may be limited by the fact that other pigmented tissue components such as hemoglobin, melanin, and cytochromes also absorb certain wavelength regions overlapping with those of tattoo pigments [27,28]. Furthermore, selective absorption within superficial pigment aggregations is not complete, leaving some radiation to be transmitted to and absorbed by deeper tissues. Upon photon absorption, energy is transferred to the pigmented sites with consequent heat denaturation of enzymatic and structural proteins. Within the pigment-laden macrophages of a tattoo, this may result in cell death and pigment release. Depending upon wavelength and pulse duration, simultaneous heating of erythrocytes may produce intraluminal thrombosis, vessel wall rupture, and coagulation necrosis of perivascular collagen and elastic fibers. A similar situation occurs for epidermal melanin pigment. The end result may be a diffuse fibrotic response and associated vascular sclerosis [10].

In 1968, Solomon et al [10] compared histologically the repair of argon laser-treated port-wine stains with those treated by neodynium and ruby laser and found them to be qualitatively similar, showing diffuse coagulation necrosis followed by a granulation tissue response and fibrosis. It has recently been shown that highly specific damage localized to skin microvasculature can be achieved using a pulsed organic dye laser operating at 577 nm [13–15]. The optical and thermal properties were modeled [13] and exposure conditions were then maximized for selective heating of blood vessels during the laser pulse. Radiation at the 577 nm oxyhemoglobin absorption band was chosen because this wavelength penetrates skin well [29], minimizes competing absorption by melanin in the overlying epidermis, and offers excellent selective absorption by blood vessels relative to the other constitutents of the dermis [12–15]. The pulse width of the dye laser (300 ns) was shorter than the calculated thermal relaxation times (about a millisecond) for structures the size of small vessels [13]. In contrast, argon laser exposures with a pulse duration of 50–200 ms allow time for extensive diffusion of heat generated in blood vessels to the entire exposed field, thereby resulting in nonselective thermal destruction [15]. By direct comparison, the damage to normal skin caused by a dye laser operating at 577 nm with a 300 ns pulsewidth is markedly distinct from the nonspecific thermal necrosis caused by the argon laser [15].

The present study investigated the use of the argon and tunable dye lasers at multiple exposure settings. Selective absorption by tattoo pigment was demonstrable. The threshold for injury to normal skin by the argon laser is approximately 20 J/cm^2 [15] while the threshold for black and red tattoos in our study was ≤ 6.25 J/cm^2. Blue tattoo pigment, which absorbs poorly at the argon wavelengths (488, 514 nm), was noted to have a higher threshold for signs of skin injury (Table II). Similarly with the tunable dye laser at 690 nm, histologic evidence of injury was seen for blue tattoo but was not seen for red or white tattoo even at the highest dose used (4 J/cm^2). This is consistent with the predicted better absorption by blue and black pigment at this wavelength. In general, the dose required with tunable dye lasers to induce the same histologic changes is much less than that required for argon, but this could be due to the shorter pulsewidth.

Regardless of power density, pulse duration, or total energy used (above a threshold dose) the results with the argon laser were histologically similar. Widespread coagulative necrosis of both the epidermis and upper dermis was produced. Tattoo pigment was "re-dispersed" in the areas of dermal necrosis. By 48 h, pigment aggregates, some apparently intracellular, were again observed. By 7 days, the papillary and upper reticular dermis was occupied by granulation tissue with the pigment present primarily in large aggregates, apparently both within macrophages and extracellularly, in linear array below this level. By this time, there was complete regeneration of the epidermis and cutaneous appendages.

Three months after laser treatment, some biopsies revealed widening of the papillary dermis by a fibrosing (scarring) process with collagen bundles running parallel to the overlying epidermis. The pigment-containing cells were present primarily in linear array at the lower border of the fibrotic dermis. In the unaffected portions of the biopsy, the pigment was present primarily in macrophages surrounding vessels of the superficial venular plexus. It was extremely difficult to quantitate any change in the absolute amount of pigment, but the pigment was definitely deeper within the dermis, separated from the epidermis by fibrosis. The cosmetically therapeutic result is thus probably, at least in part, due to the "burying" of tattoo pigment beneath this fibrosis. These findings are similar to those reported by Goldman et al in 1967 with neodynium and ruby lasers [2].

The tunable dye laser operated at 577 nm produced *qualitatively* different effects than the other lasers tested. Due to the preferential absorption by hemoglobin at this wavelength, vascular changes were more prominent. However, unlike the results reported by Greenwald et al [15] in normal human skin, there was no frank vasculitis (i.e., fibrinoid necrosis of vessel walls, leukocytoclasis) observed. Collagen necrosis and the subsequent granulation tissue and fibrosing responses were more superficial and less marked than with the other lasers. This probably is responsible for the lack of clearing (lightening) in any of the tattoos treated with 577 nm despite significant signs of epidermal damage.

In most cases, the absence of clinical signs of epidermal damage correlated with a lack of prominent epidermal and dermal damage histologically. Most doses that were clinically or histologically subthreshold at time 0 (i.e., producing no immediate epidermal or dermal necrosis) produced no changes in tattoo pigmentation. With the exception of 577 nm, any laser that produced significant dermal necrosis could produce significant "lightening" of the tattoo due to the deposition of parallel collagen fibers between the pigment and the overlying epidermis. Some pigment is apparently extruded with the necrotic epidermal and dermal debris eliminated in wound healing, but the significance of this process could not be consistently assessed.

Our data support the concept that selective absorption of energy by tattoo pigment is responsible to some degree for the localized necrosis observed. Despite the short dye-laser pulse duration of 1 μs, widespread tissue necrosis was observed at doses greater than threshold. Perhaps the even shorter pulse duration of the Q-switched ruby laser (10–20 ns) may account for the exceptional results described by Laub et al [20]. The question remains whether matching the laser wavelength precisely to the measured absorption characteristics of the tattoo and choosing a pulse duration based upon the particle size and thermal properties of the tattoo pigment might maximize selective absorption and modify the response both quantitatively and qualitatively.

In our study, "lightening" of tattoos depended upon widespread necrosis and resultant fibrosis rather than changes in the morphology of or macrophage response to the pigment granules. Clinical experience and expertise may, therefore, be more important in the final outcome than the actual laser utilized since all such lasers can be used to cause necrosis and fibrosis. After laser therapy of tattoos, some pigment seems to be removed during the acute sloughing of necrotic tissue; the remainder is partially "buried from sight" beneath a band of fibrosis. Our results seem to indicate that it is the host response to thermal injury and not a photophysical alteration of pigment which leads to improved cosmetic appearance.

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