

# Thermal conditions which cause skin burns

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The structure of human skin is illustrated diagrammatically in fig 1a. The thickness of the various components varies in different parts of the body; for the forearm the external horny layer (stratum corneum) is about 0.05mm thick, the underlying epidermis about 0.1mm and the dermis 0.5-0.7mm thick. Considerable variation occurs in the thickness of sub-dermal fat, often within the same individual at different times depending on the state of nutrition. Skin also contains hair shafts and sweat ducts, the deepest of these may just penetrate the sub-dermal fat. The walls of these structures are lined with epidermal cells. If skin is injured healing occurs by spread of epithelium from adjacent undamaged areas including epidermal cells from the hair shafts and sweat ducts. The blood supply (important for nutrition and heat regulation) lies mainly in two planes—a superficial plexus just below the epidermis and a deep dermal plexus situated just above the fat-dermis interface.

Clinically there are two important depths of burn, those that involve the partial thickness of the skin and those in which the entire depth of the skin (and possibly underlying tissues) has been destroyed. Partial skin thickness burns, although painful, will heal from surviving epithelial elements in skin deep to the burn (fig 1b). Full thickness burns can only heal by ingrowth of new epithelium from the wound edge (fig 1c); in man

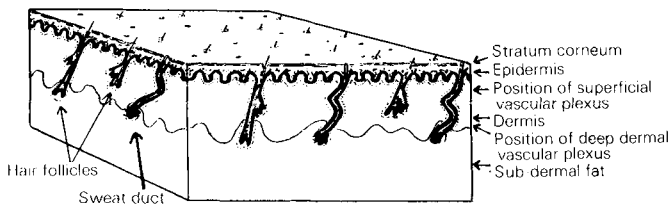


Fig 1a. Diagram illustrating the structure of normal skin. The vascular system has not been drawn but its positions are indicated

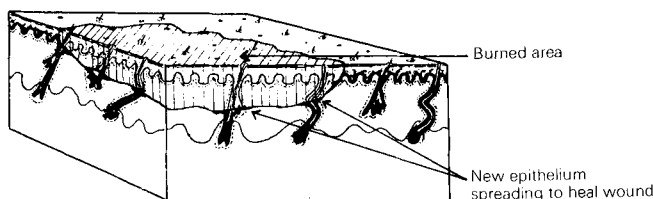


Fig 1b. Diagram illustrating a partial thickness burn of skin. Healing can occur from below the burn as well as the wound edge

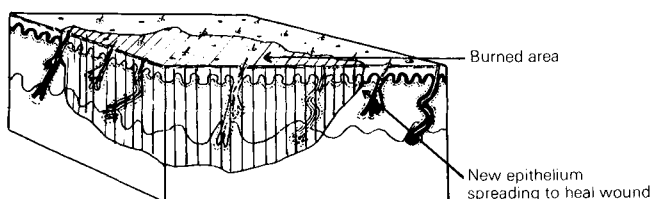


Fig 1c. Diagram illustrating a full thickness burn of the skin. Healing can only occur from the edge of the wound

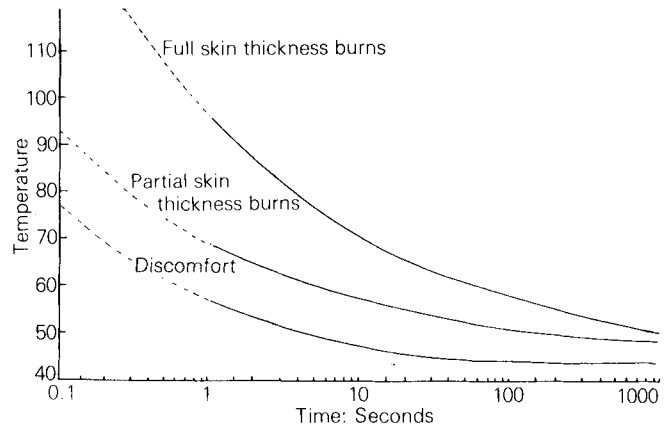


Fig 2. The relation of time and temperature to cause discomfort and thermal injury to skin

this process is slow and frequently unsatisfactory; consequently such burns, if more than a few square centimetres in area, are treated by skin grafting.

The combination of time and temperature to produce these types of burn has been thoroughly explored both in pigs and man by Moritz and Henriques<sup>1</sup> who found that the skin of both species exhibited a similar response to heat. Sevitt<sup>2</sup> investigated the pathological changes in guinea-pig skin and found that the response to burning was similar to that in pig and human skin. Standard curves showing minimum times and temperatures required to produce various types of burn are available<sup>3</sup>. Such curves can be used to determine safe surface temperatures of heated objects that might accidentally come into contact with skin. However, it is to be remembered that the limit of these curves is skin damage; in some situations—for example, where hot objects have to be handled—it might be wiser to choose conditions that do not cause discomfort. We investigated this by using human subjects<sup>4</sup>; from these experiments a further curve was produced (fig 2) which was found to parallel the curves of conditions causing actual skin damage. Small thermocouples were used to monitor the temperature between the skin and the hot handle (a copper pipe through which thermostatically controlled water was circulated); it was found that most subjects reported discomfort when the skin/handle interface reached 43°C. With the circulating water at 60°C 16 subjects (8 male, 8 female and both hands tested) reported discomfort at 43.5°C ± 0.13°C. Altering the water temperature did not significantly alter the temperature to cause discomfort although the time the apparatus could be held varied inversely with the temperature.

It seemed likely that preferred bath and shower temperatures might also provide information concerning the temperature that skin could tolerate without discomfort. The average bath temperature of a group of 20 subjects was 40.5°C; the range was from 36°C to 42.5°C. Average shower temperatures (7 subjects) were slightly lower than for baths, 40°C (range 38.5°C to 41.0°C).

These relatively low tolerable temperatures correlate with temperatures known to cause skin damage. Lawrence and Ricketts<sup>5</sup> studied the effect of exposing thin slices of isolated living skin to a range of temperatures (37°C to 50°C) for ½ hour. Respiration measurements showed that skin exhibited a graded response to heat (fig 3); these results have been evaluated by Probit Analysis<sup>6</sup>. The median effective temperature (ie, that causing a 50% reduction in the respiratory activity of skin, ET<sub>50</sub>) was 43.5°C. The response of human skin was similar to that of guinea-pig skin. Experiments have also been made in which the ear skin of living (but anaesthetized) animals was exposed to water at selected temperatures for ½ hour. After exposure the skin was excised and its respiratory activity measured. As with skin heated *in vitro* a graded response was obtained; the ET<sub>50</sub> being 43.1°C compared to 43.6°C for skin heated *in vitro*. It was perhaps surprising that skin appeared more temperature sensitive *in vivo* than *in vitro* especially at the higher temperatures; this may be due to water used to heat the animal's ears permeating the tissues to some extent.

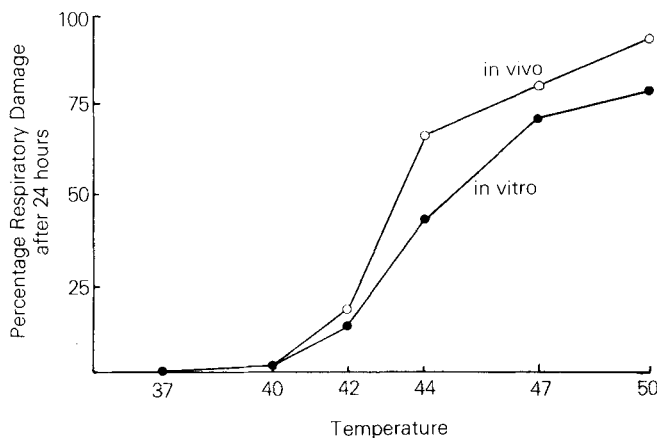


Fig 3. The effect of heat on the respiratory activity of isolated guinea-pig ear skin

These findings correlated with other observations; for instance, Moritz and Henriques<sup>1</sup> showed in man that prolonged exposure of skin to more than 43°C could cause blister formation (ie, a partial skin thickness burn); similar temperatures also produce permeability changes in the capillaries of guinea-pig skin<sup>7</sup>. From these observations it is reasonable to conclude that for an object to be handled or be in contact with skin for any appreciable length of time the temperature of the skin/handle interface should not exceed 42°C and that damage is to be anticipated with temperatures above 43.5°C.

Experiments have been made using guinea-pigs in which the temperature rise of the subcutaneous tissue was monitored with a fine thermocouple during and after the burning process. Burning was effected by means of a heated brass block applied to the epilated flank of the animal for ten seconds; the results are shown in Table 1. Experiments reported elsewhere<sup>8</sup> show that for exposures of ten seconds temperatures above 70°C are required to produce a full skin thickness in depth, at 60°C very superficial and at 50°C only erythematous. It is interesting to note that for relatively short applications of the heat source the skin takes a comparatively long time to cool. This is especially

marked at temperatures above 60°C. In this connection it is pertinent to note that little coagulation of tissue protein occurs at temperatures up to 60°C, thus the burns made at 50°C and 60°C cool by re-radiation and by conduction of heat by the vascular systems of the skin. At higher temperatures the vascular system is also impaired at an early stage thus reducing the efficiency of cooling. The efficiency of the blood supply in cooling these burns was demonstrated in a separate experiment where the 100°C burning iron was applied to the skin of a dead animal for ten seconds, in this circumstance the temperature rise was 31.2°C at a depth of 1.54mm below the skin surface.

It is these alterations to the local vascular supply of skin during heating that make calculations concerning heat flux difficult. If a linear relationship is assumed between burning temperature and the subcutaneous temperature recorded during burning (Table 1) the smallest temperature rise in the plane of the deepest epithelial elements consistent with production of a full skin thickness burn is about 45°C. This temperature might appear too low from evidence presented earlier (figs 2 and 3) particularly as relatively short times are involved.

Experiments reported by Cruickshank and Hershey<sup>9</sup> suggest that temperatures above 50°C for 60 seconds are needed to cause appreciable damage to isolated skin. Skin proteins vary in their sensitivity to heat, for example, collagen is not appreciably degraded at temperatures below 71°C whereas certain specific enzyme systems are sensitive to much lower temperatures<sup>10 11</sup>. The situation is further complicated by the fact that skin cells adjacent to the obviously heat coagulated tissue seen in many burns may not become completely necrotic until several days after injury<sup>12</sup>. Clearly we still need to do more work in order to understand the actual mechanism of thermal damage to living skin.

Obviously the curves shown in fig 2 are valuable for determining safe surface temperatures for objects that might come into contact with skin. Radiator panels are most efficient if run at a high temperature, however, if not guarded it is desirable that they should not produce a burn if touched accidentally. In determining a maximum temperature it may be necessary to allow for reaction time; in a normal person this is about 0.2 sec. (to electric shock) if the subject is unaware that the stimulus is likely to be painful<sup>13</sup>. It is possible that reaction time to a heat stimulus may be longer; it will also probably be longer in the elderly. Insulation, even in a thin layer (such as paint) is likely to modify the transfer of heat from the hot object to the skin but little

Applied temperature (10 secs)	50°	60°	70°	80°	100°
Thermocouple depth, mm	1.44	1.55	1.43	1.67	1.51
Maximum temperature rise recorded	2.70°	6.04°	11.01°	10.25°	11.56°
Time to return to original temperature (secs)	85	84	109	168	285
Number of observations	12	12	12	12	18

Table 1 Temperature measurements within the skin after application of a heated metal block

practical information is available concerning this. Experiments with the 'hot handle' apparatus in which the copper tube was replaced with a tube made of polythene showed that hand/handle interface temperatures to cause discomfort remained unchanged but the time taken to reach the discomfort point was prolonged.

In summary we have good clinical and laboratory methods for assessing burn damage to skin and we know reasonably thoroughly the conditions in terms of temperature and time of exposure to produce these effects. Measurement in terms of heat flux has proved more difficult; progress in this direction could help in better understanding of the events at a biochemical level which characterise burning injury.

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