

Investigating the depth of thermal burns in elephants

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ABSTRACT

Histological examination of burn injuries in elephants revealed that the depth was not as severe as expected from clinical observation. Although the actual burn depth was deep, the thickness of elephant skin, especially the dermis, resulted in the lesions being classified as less severe than expected. Examination of skin samples from selected areas showed that most lesions were either superficial (1st degree) or superficial partial-thickness (superficial 2nd degree) burns with the occasional deep partial thickness (deep 2nd degree) wound. These lesions however, resulted in severe complications that eventually led to the death of a number of the elephants.

Key words: depth, elephants, thermal burns, survivability.

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INTRODUCTION

A number of elephants were caught in a bush fire and sustained varying degrees of burn lesions. Seventeen young elephants were translocated to bomas and close clinical evaluation of large sections of their burn lesions appeared severe and were judged to be 3rd-degree burns or worse.

The understanding of the severity of a burn includes extent, depth and location of the lesion(s) and these factors are primary determinants of mortality following thermal injury³. Burn depth, in particular, is the primary determinant of the patient's long-term appearance and function³. Whereas the extent and location of the burn wounds suffered by elephants caught in bush fires can be determined from a distance, the depth is extremely difficult to gauge. 'Hands on' clinical assessment of the burn depth can be inaccurate and is dependent on the experience of the assessor, the length of time since the insult, and the difference and variation in skin physiology and anatomy between species and changes as the result of topical medications applied². Further

complicating this assessment is that burn wounds are dynamic and evolving, sometimes substantially, over the first few days following the insult, and that the injury is rarely of uniform depth throughout the field of damage^{2,4}.

In humans, early surgical intervention including excision, grafting and acute flaps individualised to each patient is usually performed between days 3 and 5 post-burn of all burns that will not heal within 3 weeks³. Burns that heal within 3 weeks generally do so without hypertrophic scarring or functional impairment, although long-term pigment changes are common³. Burns that take longer than 3 weeks to heal often produce unsightly hypertrophic scars and frequently lead to functional impairment as well as providing only a thin, fragile epithelial covering for many weeks or months³. The challenge is to determine which burns will heal within 3 weeks and this is generally related to the depth of the burn. Early surgical intervention is therefore the treatment of choice for deep dermal and full-thickness burns and should be done before spontaneous demarcation begins and while bacterial contamination remains minimal^{3,4}. Mortality rates have been lowered, with survival rate in young patients (17–30 years) with extensive burns [mean 73 % of total body surface area (% of TBSA)] improving from 9 % with delayed skin grafting to 45 % with early excision⁵. With this adoption of early surgery, classifying burns by their depth has assumed greater importance and although a number of techniques have

been described, clinical assessment is still the most commonly used method^{3,4}.

The diagnosis of burn injury depth refers to the burn wound by anatomical thickness of the skin involved based on certain clinical criteria, initially described by 'degrees' of damage from 1st to 4th degree. A preferred, more appropriate classification includes superficial, partial-thickness (superficial and deep), full-thickness and subdermal burns².

Superficial (1st degree, 1°) burns affect only the epidermis². Although the epidermis anatomically lacks a direct blood supply, the wound still becomes erythematous, possibly with a mild surrounding oedema, due to irritation of the vascular plexus that projects up into the epidermal-dermal junction². Blisters do not form and the affected surface is dry. Pain is in the form of tenderness to touch and may be delayed for a few hours. The area will heal itself within 3–5 days without evidence of scars through re-epithelialisation via proliferation of viable epithelial cells from surrounding areas and/or from hair follicles and sweat ducts situated within the dermis^{2,4}.

Partial-thickness (2nd degree, 2°) burns penetrate and destroy the epidermal layers and extend into the dermis. This category of burn can be subdivided further based on the dermal stratum affected and wound characteristics. A superficial partial-thickness (superficial 2°) burn extends down into the papillary, or superficial layer of the dermis and these wounds are red, moist (weepy) and painful. These lesions become erythematous due to dermal inflammation and a hallmark of this type of burn is that when pressure is applied to the reddened area it will blanch but with cessation of pressure will demonstrate immediate capillary refill and become red again². Intact blisters (fluid collection at the interface of the epidermis and dermis) will form which, when ruptured, will weep due to leakage of interstitial and intravascular fluid. Intense pain is experienced because of exposed nerve endings. Severe swelling and oedema are due to the involvement of the dermal vascular network. Healing usually takes a week or two and although scar formation is not a problem, pigmentation changes may occur due to the

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destruction of melanocytes².

Deep partial-thickness (deep 2°) burns extend deep down into the reticular layer of the dermis and present as a mixed ivory, pearl white or red colour. Although the reddish areas will blanch with pressure, the capillary refill will be sluggish when pressure is released². Blisters are usually absent but the exposed surface is wet or moist. Destruction of superficial nerve endings dramatically alters sensation so that appreciation of superficial pain by pinprick is absent yet pressure applied by the pin is still felt². This is due to the preservation of the Pacinian corpuscles, located deep in the reticular dermis, which are sensitive to pressure and vibration². Oedema is marked. Healing occurs spontaneously over several weeks through the process of granulation tissue formation and epithelialisation resulting in dense scar formation².

Full-thickness (3rd degree, 3°) burns destroy both the epidermal and dermal skin layers extending below the hair follicles and sweat glands to subcutaneous (fat) tissue. The colour of these wounds can vary from black (necrotic tissue), to shades of tan, to red (do not blanch as redness is from entrapment of haemoglobin liberated from destroyed red blood cells), to white (total ischaemia). Visible superficial blood vessels are commonly thrombosed². The surface appearance is of charred skin (eschar) that is dry, rigid, leathery and stiff and devoid of pain due to the destruction of all cutaneous nerves². The affected area may appear depressed or dehydrated due to tissue fluid evaporation whilst the surrounding tissue exhibits severe swelling. Scar formation and scar contractures are severe.

Subdermal (4th degree, 4°) burns extend into the subcutaneous levels of fat, muscle, tendons and bone. Areas of involvement appear charred and tissues are devitalised or mummified and pain sensation is again minimal².

Unfortunately, owing to the nature of a bush fire, which surges and subsides, many of the burn lesions would not fall exactly into the described categories and the depth of the burn would vary considerably over the burned area.

Biopsy and tissue histology in humans is considered the gold standard in determining the depth of skin burn; however, since this technique is invasive and time is required to fix the samples, prepare the slides and then to examine them, this method is mostly limited to cadavers⁴.

METHODS

Representative skin specimens were collected from selected areas of burn lesions from 2 elephants that died from



Fig. 1: Lower legs showing swelling, eschars and severe suppurative oozing.

complications associated with the severe and extensive burn injury sustained in a bush fire. Both elephants were euthanased, 1 within a month of the insult and the 2nd approximately a month later. Both had received topical and systemic treatment on a few occasions. However, these treatments were both erratic and inadequate since wild elephants are neither compliant nor easy to handle. It is unlikely that the treatments would have had a major impact on the samples taken.

Skin specimens ($\pm 1 \times 1$ cm) were cut from locations of macroscopically different depths of burn and fixed in 10% formalin. After inclusion in paraffin, serial sections were prepared perpendicular to the skin surface. The slides were stained with haematoxylin-eosin and then examined by optical microscope.

A normal skin control from a third elephant was taken from skin that covered the frontal area of the cranium.

RESULTS

Seventeen subadult or younger badly burnt elephants were translocated and isolated in groups in bomas. The severest burn damage was to the lower body and extremities (*i.e.* legs but often including the trunk and tail). The perineum, although difficult to view or examine (even when the animal was immobilised) appeared to be consistently and severely damaged; however this could have been exaggerated owing to the friction associated with this area.

Swelling was evident in the lower extremities of all the elephants, which severely affected mobility. Over time, eschars developed and scar contractures formed mostly over the joint areas of the

lower legs. The resulting fissures often exuded serum and or purulent exudate (Fig. 1). Mobility was affected and the animals exhibited varying degrees of stiffness and would regularly rock from side to side or shuffle slowly within the bomas.

The skin of a number of the elephants peeled away in the lower abdominal and proximal leg areas giving the animals a 'woolly' appearance (Fig. 2). The underlying skin varied in colour from red, to purple to a waxy white.

Histopathology of the various biopsy sites

- 1) Undamaged skin covering the frontal area of the cranium. This specimen exhibited characteristics commonly expected for skin and considering the species involved, the very thick *stratum corneum* and the exceptionally thick dermis were not surprising. Adnexal structures included sparse primary hair follicles without pilosebaceous units or sweat glands.
- 2) Skin covering the dorsum of the carpus (Fig. 3) (eschar excluded). The epidermis was thickened (acanthosis) and thrown into folds forming outward papillary projections and inward rete pegs reaching into the underlying dermis. The *stratum corneum* was markedly thickened exhibiting severe orthokeratotic hyperkeratosis. Approximately 10–15 cell layers into the *stratum corneum*, areas of serocellular crusts consisting of degenerated neutrophils, necrotic corneal debris and blood breakdown products were observed. No underlying tissue damage or active inflammatory

reaction was present.

- 3) Skin covering the medial fetlock region of the right hind limb (Fig. 4). There was severe, extensive, full thickness epidermal necrosis and superficial dermal collagenolysis extending to the level of the mid-dermis. Vascular thrombosis was prominent in the necrotic tissue and at the junction of the necrotic and viable dermis. Sloughing of a thick band of necrotic dermis-epidermal tissue resulted in mixed secondary bacterial (cocci to short rods) invasion with massive neutrophil influx to the mid-dermis. A sharp distinction could be made between viable and necrotic tissue, with the former showing extensive fibroplasia. No surviving residual basal epithelial cells could be found in the section, indicating that repair in this site would result in extensive scarring.
- 4) Peripheral skin of the ear margin (Fig. 5). The rostral planum showed marked acanthosis with large intermittent sections of full thickness epidermal and superficial dermal necrosis, complicated by secondary bacterial invasion and suppurative inflammation. In certain sections the inflammatory reaction extended to the underlying cartilage, accompanied by moderate oedema. In the areas where the epidermis was still intact, the *stratum corneum* exhibited marked degeneration and mild to moderate hyperkeratosis. The caudal planum of the ear showed similar damage but to a lesser degree.
- 5) Skin covering the frontal area of the cranium (Fig. 6). Multifocal areas of acanthosis, orthokeratotic hyperkeratosis and keratinocytic vacuolisation are separated by areas of ulceration ranging from superficial to full thickness epidermal and superficial dermis.



Fig. 2: The 'woolly' appearance of elephants from the sloughing skin.

- 6) Skin covering the dorsum of the trunk (Fig. 7). This section showed orthokeratotic hyperkeratosis and acanthosis with multifocal areas of epidermal ulceration. The superficial epidermal damage was associated with neutrophilic exocytosis resulting in serocellular crusting of degenerate neutrophils, necrotic debris (keratin and keratinocytes) and blood breakdown products. In the areas where the epidermal layer was damaged to the *stratum basale*, epithelialisation was

evident in the repair process. Mild to moderate subepidermal fibroplasia and mild perivascular lymphocytic infiltration was also noted.

- 7) Skin at the footpad junction of the medial aspect of the hind limb (Fig. 8). There was clefting and separation of the keratin layers in the *stratum corneum*. Some clefts were serum-filled while others were devoid of substance, presenting a vacuolated appearance to the mid-corneal area of the *stratum corneum*. At the junction of the skin and footpad the stratum corneum separated from the underlying epidermis. The latter was intermittently disrupted, exposing the underlying papillary dermis, which showed superficial neovascularisation and fibroplasia, indicating repair by granulation tissue formation with proliferation of basal keratinocytes

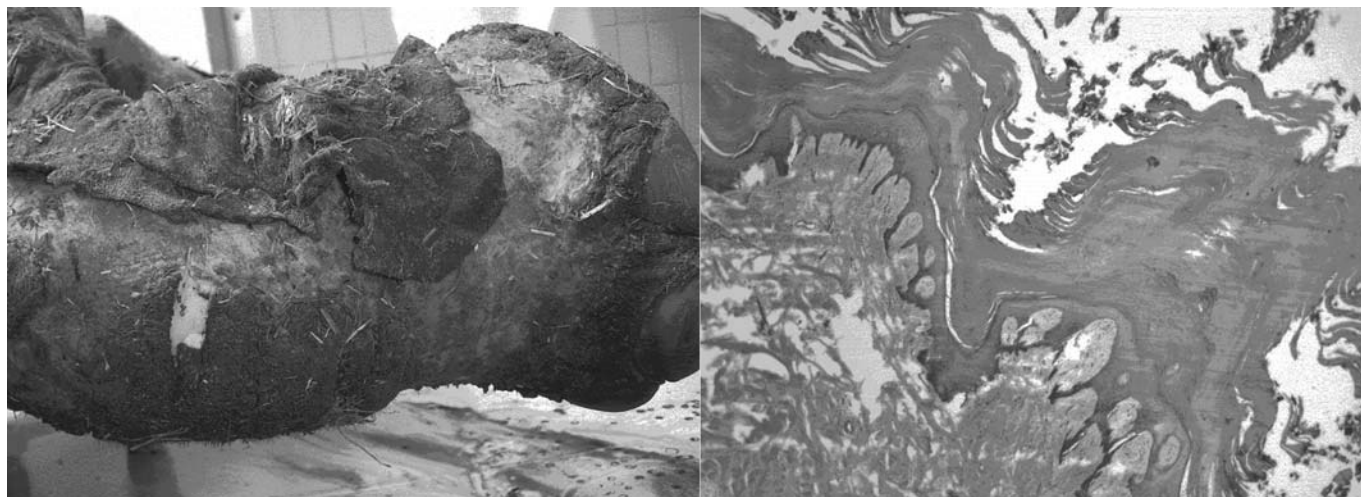


Fig. 3: Skin covering the dorsum of the carpus.

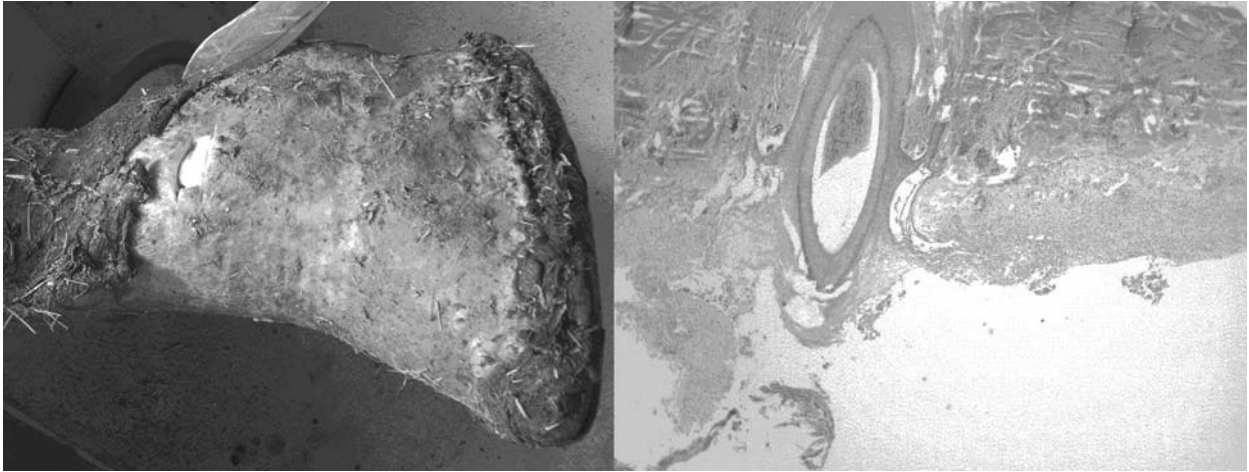


Fig. 4: Skin covering the medial aspect of the right hind limb.

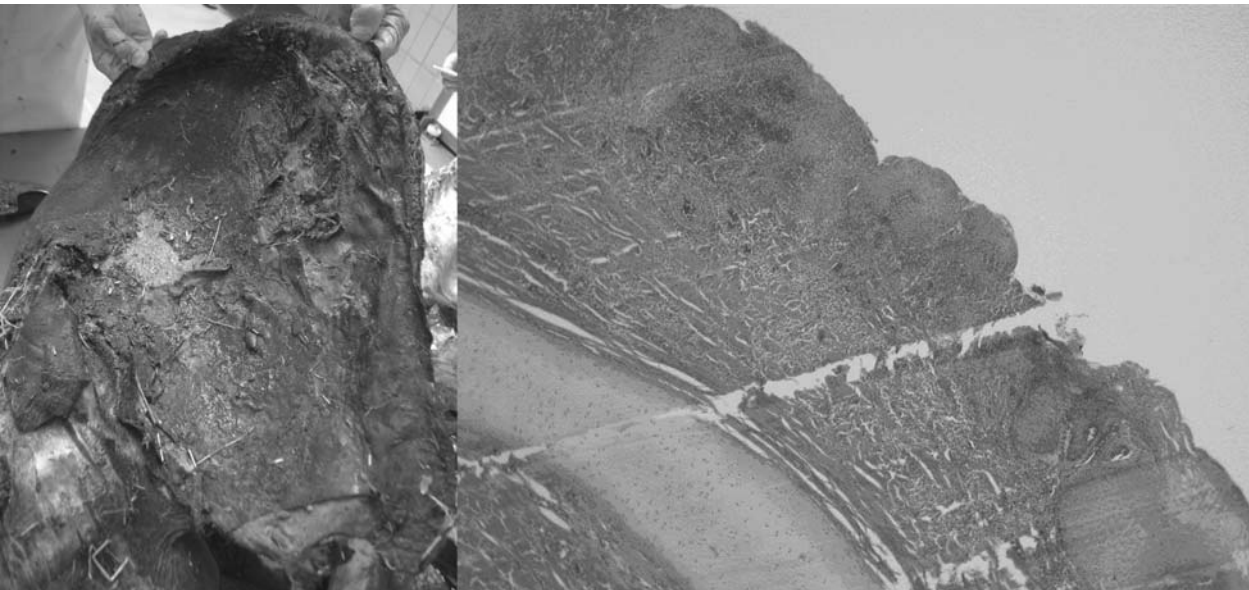


Fig. 5: Peripheral skin of the ear margin.

with epidermal bridging of exposed dermis.

- 8) Foot pads (Fig. 9). A very thick cornified wall is part of a modified epidermis characterised by primary epidermal lamellae forming tubular horn and interlamellar epidermis forming intertubular horn. The apex

of the primary lamellae are the most distal points of horn production and these areas, being closest to the external environment, when exposed to any heat source, will probably be most severely affected. Tubular horn being produced at the time of the insult will show the greatest abnormalities. The

interlamellar epidermis, positioned more proximally, will be affected less by the heat insult. The tubular and intertubular horn halfway into the footpad wall exhibited a layer distinctly different from normal horn and the tubular horn especially showed ballooning and filling with

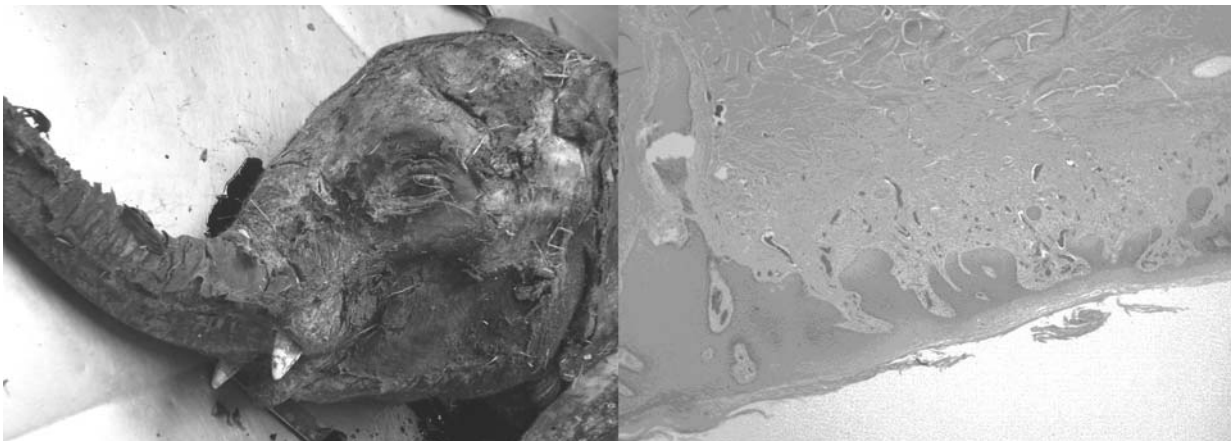


Fig. 6: Skin covering the frontal area of the cranium.

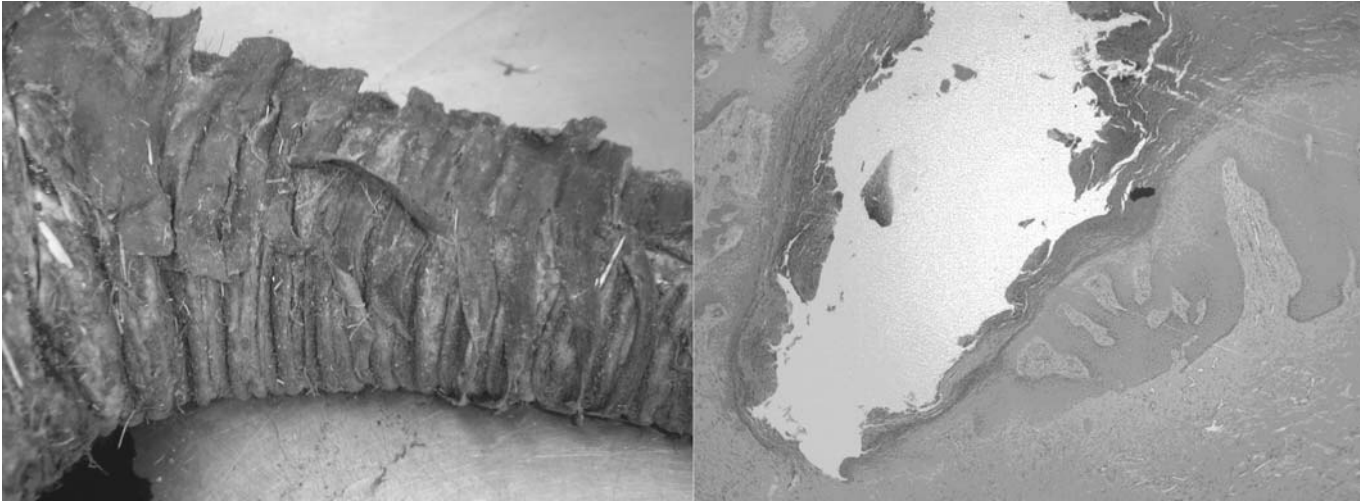


Fig. 7: Skin covering the dorsum of the trunk.

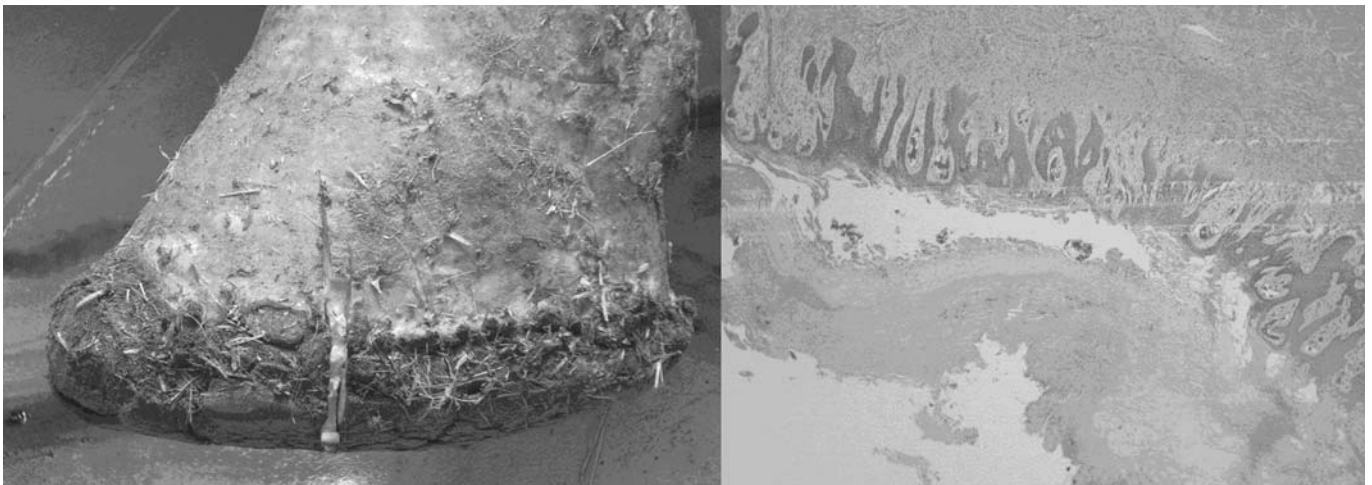


Fig. 8: Skin at the footpad junction.

pale eosinophilic substance (serum?). This layer could be the result of the burn damage to the horn cells forming new horn that has grown through with subsequent repair accompanied by normal production of horn.

DISCUSSION

Although the tissue samples examined were collected over a month after the fire, the degree of burn damage sustained at the time of the insult was readily determined and this time delay, although

unintentional, also provided an insight into the healing and repair process.

From the skin samples examined it appears that the burn damage to the elephants rarely reached full-thickness (3°) burns, although clinical observation

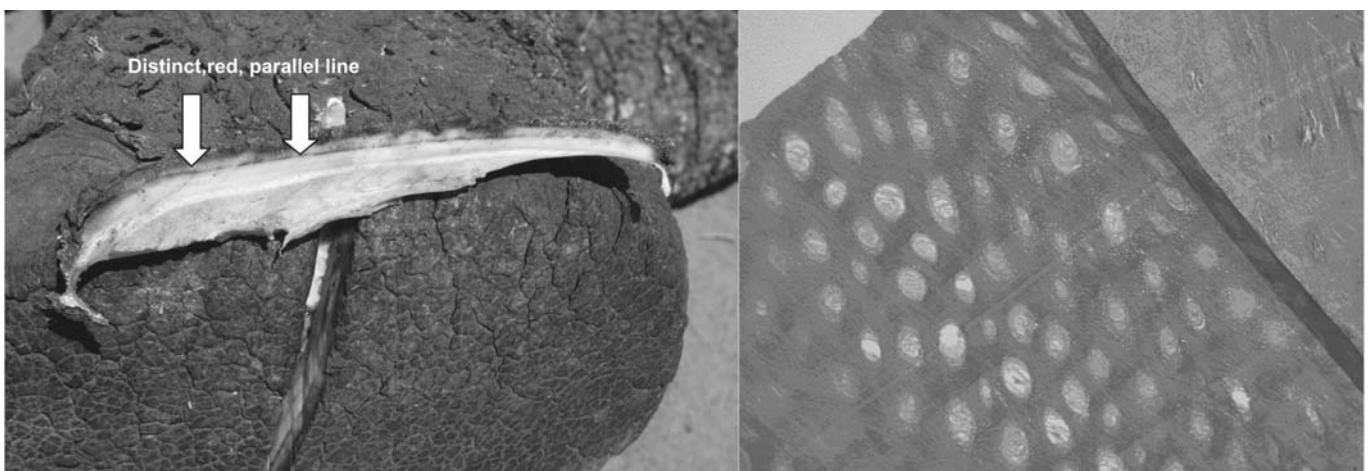


Fig. 9: Section through the footpad.

Table 1: Normal epidermal and dermal thickness: a comparison between the domestic dog, humans and the African elephant.

		Epidermis	Dermis	Total	
		(μm)	(μm)	(μm)	(mm)
Domestic canine ⁶	Foot pad*	1 800	1 150	2 950	3
	Nasal plane*	600	1 150	1 750	1.8
	Dorsal carpus*	75	1 150	1 225	1.23
African elephant [†]	Sole	12 950	9 000	21 950	21.95
	Trunk	900	6 900	7 800	7.8
	Dorsal carpus	900	9 000	9 900	9.9
Humans ¹	Sole	1 400	3 000	4 400	4.4
	Body average	70–120	1 000–2 000	1 070–2 120	1.1–2.1

*Dog skin measured at 6 months of age.

[†]Unpublished data: tissue for histological examination sampled during routine necropsy of a 2-year-old African elephant.

and examination suggested far worse. Most of the worst lesions were superficial partial-thickness (superficial 2°) burns that were probably due to the very thick dermis found in elephants. Table 1 compares the normal epidermal and dermal thickness between the domestic canine, humans, and the African elephant. Neither comments nor comparisons are made regarding differences between dermal connective tissue types, architecture and arrangement. These factors will definitely affect the skin's response to different types of insult. However, making a crude deduction from a comparison to dogs and humans, the elephant dermal collagen is much denser and coarser, whereas the epidermal differences are not as pronounced between the species.

Superficial partial-thickness burns in humans are associated with severe pain, loss of fluid, swelling and inflammation. Periodic and subjective evaluation of the elephants revealed a similar scenario as the majority of the lower legs were swollen, exuded fluid from fissures and exhibited severe pain. The presence of pain was gauged from the continuous stiff shuffling,

rocking, weight shifting and excessive rubbing of affected areas. Mobility was severely restricted and the refusal of the elephants to lie down during their stay in the bomas, despite repeatedly collapsing when falling asleep, also attested to the presence of pain.

The principal cause of death in humans and domestic animals following massive burn injury is wound sepsis². Bacterial toxins and inflammatory mediators released at the burn site contribute to increased localised microvascular permeability and disseminate intravascular coagulation, eventually leading to systemic inflammatory response syndrome and multiple organ failure. The lower legs of all the elephants were severely infected and inflamed so it is not surprising that all the *post mortem* examinations done on the elephants indicated that the cause of death was septicaemia and dehydration. The large eschars protected the underlying festering tissue from the occasional flushing by hosepipe and topical antibiotics.

Body fluids are lost from the surface of the burn primarily due to interstitial

leakage and capillary compromise, and in humans and domestic animals intravenous fluids are needed to prevent the development of clinical shock in major burns (*i.e.* >10 % burn extent of TBSA in children and >15 % extent of TBSA in adults²). Elephants would lose fluid by the same mechanism and to complicate matters, water intake would probably be severely curtailed owing to the burn injuries sustained to their trunks. Competition in the close confines of the bomas would exacerbate this problem and the practical impossibility of intravenous fluid administration inevitably results in dehydration being a major concern.

The trunks of a couple of elephants that appeared to be severely burnt simply hung limply with barely a twitch for days. The skin eventually sloughed and slowly the trunks became more mobile to such an extent that healing appeared complete.

The footpads appeared to be relatively unaffected macroscopically at the majority of *post mortem* examinations and subsequently attracted little attention. However, in the 2 cases in which these areas were more thoroughly examined, there was undermining of various lengths ($\pm 1\text{--}10$ cm by 2–3 cm) of the superficial footpad layer (Fig. 10), which, at first glance, did not appear to be problematic. It was assumed that thermal damage to this area would be minimal owing to the thickness of the footpad and because the fire exposure would have been doused quickly when trodden upon. Sectioning of all 8 footpads examined demonstrated a macroscopically distinct, parallel, red demarcation in the epidermis (Figs 9, 10). Despite thorough histological examination including various staining techniques, the lesion could not be identified nor determined. This

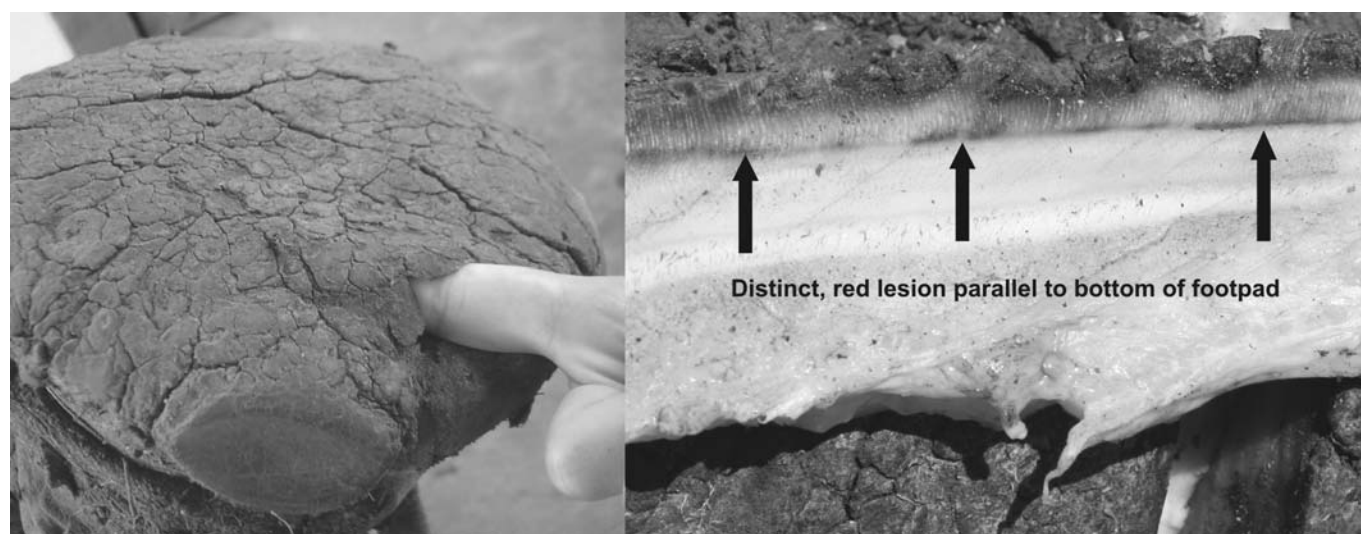


Fig. 10: Undermining of the footpad and the distinct lesion (arrows).

could be the response of horn growth damage at the time of the insult that had grown down by the time it was detected at *post mortem*. Again, this area could be a potentially weak spot in the footpad and could explain the sections of under running sole that were noted macroscopically.

Owing to the inaccessible terrain in certain areas of a local game reserve, it was estimated that as many as 50 elephants were caught in the bush fire. Seventeen subadult and juvenile elephants were translocated to a holding facility for treatment. Fifteen of the 17 eventually died over a 2½-month period. The fire burn location and pattern sustained by the elephants is almost identical in all cases with minor variations due to the size of the elephant or whether they were burnt from behind or facing the fire. The depth of the severest burn lesions appeared macroscopically far worse than the superficial to deep partial-thickness burns obtained. However, the classification used for histopathology is specific to humans. The lack of full-thickness burns and the occasional deep partial-thickness burns would probably result in very little, if any, scar formation and subsequent complications usually associated with this healing process. Nevertheless, the depth and extent was obviously severe enough in

these elephants to eventually cause death in most of these cases.

CONCLUSION

If the human classification for the depth of burn wound is used in elephants it appears that damage sustained is not as severe as initially thought when evaluated from close quarters. This is probably due to the increased thickness of the dermal layer of the elephant's skin, which is probably a protective mechanism. However, closer examination of the anatomy and structure of the elephant skin needs to be done to try to determine whether similar and/or other unique functions can be attributed to this organ.

In future, should elephants be burned in a fire, it will be essential to have a coordinated plan of action in place. A suggestion is that as soon as possible after the burn, the elephants should each be mapped for the extent of their burn injury⁷. If this value exceeds 40 % of TBSA then immediate euthanasia would be advisable⁷. If treatment is decided upon, in the light of the over-population and culling controversy, the depth of burn should be determined in various areas using the clinical parameters discussed. This could be done as each elephant is tranquillised for translocation. These results can help determine the type and intensity of treat-

ment. Three to 5 days later, each elephant should be anaesthetised again and, depending on the depth and location of the burn wounds, excision and debridement could form part of the treatment protocol.

Although bush fires can be a natural phenomenon, the restriction of wild animals within certain locations is not, so if and when elephants are burnt in bush fires, it is our responsibility to intervene to prevent undue pain and suffering.

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