Bodies associated with fires

W Lawler

Introduction

For the pathologist providing a routine necropsy service to the local coroner, examination of bodies associated with fires can generate difficult interpretational problems. Appropriate historical and circumstantial evidence may be vital to overall conclusions,¹ although, as with bodies recovered from water,² such collateral information should always be available before any coroner's necropsy is undertaken.³

Not all bodies recovered after a fire will have died from its effects, although they may show features indicative of having been in it. Such bodies, therefore, require particularly careful examination, both external and internal, to catalogue (and subsequently to explain satisfactorily) all injuries present; to determine whether death indeed followed the effects of the fire; and to see whether any natural disease (such as ischaemic heart, cerebrovascular disease, and hypertension) may have contributed to, precipitated, or even caused death. It is also important to determine whether the deceased was under the influence of alcohol or other drugs at the time of death.

Finally, the pathologist has a vital role in determining, from all pathological and circumstantial evidence available, whether the overall findings are consistent with, or even point directly towards, accident, suicide, or homicide.

Unfortunately, some bodies recovered after fires are extensively burned and therefore difficult to examine; nevertheless, careful examination should elicit sufficient findings to allow reasonable conclusions to be drawn.

For the pathologist to interpret accurately the necropsy findings, it is necessary briefly to consider and to appreciate the classification, appearances, and prognosis of burns, the possible causes of death for burned bodies, and the spectrum of post mortem and artefactual injuries.

Classification and appearances

Burns represent local tissue destruction by dry heat on or near the skin. Causative agents include not only naked flames, but also such hot substances as metal—electric fire elements for example—glass, or even, under adverse circumstances, electric blankets and hot water bottles.

The risk of burning is proportional to the temperature of the agent concerned and to the duration of its application. Experimentally, it has been shown that it takes 5 or 6 hours for an agent at 44°C in contact with human skin to cause cutaneous burning, but

only 3–5 seconds when at 60°C.⁴

Several classifications of burns exist, but the simplest, and now the most commonly used, reflects depth of skin affected; it is well documented in several standard textbooks of forensic medicine and pathology^{1 5-10} and review articles.^{11 12}

PARTIAL THICKNESS (FORMERLY SUPERFICIAL OR FIRST DEGREE)

Burns are limited to the epidermis and do not extend into dermal tissues. As the basal epithelial layer remains intact, regeneration can occur, and with victim survival, no scarring will result. These burns often blister (vesication), and the adjacent skin is hyperaemic; although the blisters may become infected, healing will ultimately supervene.

FULL THICKNESS (FORMERLY DEEP OR SECOND DEGREE)

Burns extend through the epidermis, destroy basal layers, and affect dermal elements; regeneration of epidermis and dermal adnexal structures can not occur, and therefore healing inevitably involves some scar tissue formation, subsequent contraction of which may impair local function or disfigure. These burns show tissue coagulation and, when deep (formerly *third degree* burns), there may be charring of fat, muscle, and even bone. Healing, even of superficial full thickness burns, is slow and often complicated by local infection; in clinical practice it is usually facilitated by surgical excision and grafting.

The zone of hyperaemia around the periphery of a burn represents a vital reaction and therefore is a very useful indicator that the injury occurred during life; unfortunately, when death supervenes very shortly afterwards, it may not have time to develop. Histological examination is often disappointing, as several hours need to elapse before a typical acute inflammatory reaction can be identified. Many burns encountered on bodies removed from fires have occurred after death; when they are extensive, it is usually impossible to determine whether any ante mortem burning is present, although other observations may indicate whether the deceased was alive when the fire started.^{1 7 8}

Prognosis

This is related to the body surface area affected and to the age of the victim.

BODY SURFACE AREA

This is usually calculated by the so-called "rule of nines", where different parts of the body are allocated percentages of its total sur-

Home Office
Pathologist,
Manchester
W Lawler

Correspondence to:
Dr W Lawler, Pine House,
21 Alkington Green,
Middleton, Manchester
M24 1ED

Accepted for publication
17 March 1993

face area as nine or multiples of nine—whole of head 9%, front of trunk 18%, back of trunk 18%, each arm 9% and each leg 18%; the remaining 1% is usually assigned to perineum and external genitalia.^{1 6 7 10–13} Although this method of calculation was designed for clinical use, it is equally helpful when assessing the overall percentage of body surface area involvement at post mortem examination.

AGE

Presumably because it reflects general body health and overall resistance to trauma, age influences survival. Thus when the victim is up to 25 years old, appropriate treatment will provide a good chance of recovery from up to 60% body surface area burns; by 40 years of age, a similar chance of recovery is seen in up to 40% burns; by 60 years it is 20%, and by 80 years, it is down to 5%.^{5 12}

Causes of death for burned bodies

These are usually divided into early or late, but it must be remembered that death may, for whatever reason, have preceded the fire, and so it is perhaps better to divide causes of death for burned bodies into before, during, or after the fire.

DEATHS BEFORE THE FIRE

Although uncommon, numerous possible causes exist: most are natural, but a few are due to overdosage of alcohol or other drugs. Of course, the pathologist should always consider that every body removed from the scene of a fire may have been the victim of violence inflicted by some other person, and this possibility should actively be excluded. When death has occurred before the fire started, findings indicating life during the fire, such as hyperaemia around the burns present, inhaled soot particles intimately admixed with the mucus of the upper and lower major airways (and perhaps also the oesophagus and stomach), and a blood carbon monoxide saturation greater than 10%, will inevitably be absent, although, as discussed below, their absence does not automatically indicate death before the fire started: for example, in rapid deaths from burns in flash fires where there is a sudden burst of flame following ignition of combustible gases^{1 5 7 10 14–16} and in deaths from the effects of heat.

DEATHS DURING THE FIRE

These occur after the fire has started but before the victim is rescued—that is, these bodies are removed, after death, by the emergency services. Causes here include:

Burns when it is the tissue destruction with consequent “toxaemia”, “shock”, hypovolaemia, hypotension, haemoconcentration, and hyperkalaemia which are responsible for death. Although difficult to determine, such early deaths from burns are probably not as common as stated or implied in several textbooks, and are most likely to be encountered in very rapid (“flash” or “flashover”)

fires^{1 5 7 10 14–16}; even under these circumstances, the associated heat (as discussed below) could well be an important factor in causing death. It must always be remembered, as stated previously, that most of the burns found on bodies removed from fires will have occurred after death.

Heat This is undoubtedly responsible for some fatalities in fires. Below about 200°C, rapid onset peripheral circulatory failure is probably relevant; at higher temperatures, central cerebral factors may well apply.^{17 18} Death from the effects of heat can occur without burns to the skin, and carbon monoxide/cyanide concentrations (see below) may not be significantly increased¹⁸; as these victims are, of course, alive during the fire, there will probably be obvious soot staining of the mucus lining the major airways.

Toxic gases Here, any one or more of a large range of substances or environmental states may contribute to death. Over 300 toxic gases can be produced, particularly while the fire is smouldering, and their concentrations depend largely on the nature and composition of the combustible materials involved and the temperature of the fire.^{18–20} Such gases include carbon monoxide, hydrogen cyanide, nitrogen dioxide, nitrogen tetroxide, phosphene, benzene, ammonia, formaldehyde, acrolein, hydrogen fluoride, chlorine, hydrogen sulphide and other oxides of sulphur. Facilities for routine biochemical analysis of the most relevant (carbon monoxide and hydrogen cyanide) exist, but the others are difficult to analyse, and are not looked for routinely. In about half of all deaths during fires, the carbon monoxide saturation exceeds 50%—the level often considered to be the minimum required for fatality^{16–21}; in the context of fires, however, lower levels may be not only relevant to incapacitation, but also actually responsible for death,¹⁷ particularly in the very young or in older people with clinically relevant pre-existing natural diseases which reduce cardiac or pulmonary reserve.²¹ In addition to toxic gases, irrespirable ones, particularly carbon dioxide, may be produced and contribute to death by causing hypoxia. Finally, a point not noted by all textbooks, but one which some investigators consider important, is that a reduction in the oxygen content of the victim's environment within the fire may also contribute significantly to an overall hypoxic, “asphyxial” type of death.¹⁶

Physical trauma This is sustained, for example, while trying to get out of the fire or when burning buildings collapse. Of course, such injuries may be relevant to death after rescue, or, conversely, they may occur after death has already taken place from causes as discussed above.

DEATHS AFTER THE FIRE

These are often complicated and multifactorial, involving several organs and tissues.^{6 12 22 23}

Burns If these are extensive, they may contribute directly to death, particularly within 24 hours of removal from the fire. Fluid or electrolyte disturbances and the inevitable

hypermetabolic response, together with ill defined “shock” and “toxaemia” from necrotic tissues are probably important factors.

Infections These, with subsequent multi-organ system failure, remain the major cause of death after burns, with the burns themselves or the lungs (which may or may not have been damaged directly by smoke during the fire) being the main sources for sepsis.²³ Highly virulent, often opportunistic organisms are frequently involved, particularly as burns may induce various alterations in cellular or humoral immunity.²³

Lungs Pulmonary insufficiency with respiratory failure may be an important factor. It is thought that dry heat, although it may burn the upper airways, is unlikely to damage the lungs^{13 17 24}; nevertheless, smoke and toxic fumes undoubtedly can, with some inhaled water soluble substances dissolving to produce strong acids or alkalis. These cause local damage as indicated by congestion, oedema, haemorrhagic exudates, hyaline membranes, patchy collapse and, later, bronchopneumonia.^{13 17 18 23 25} Lipid soluble substances in the inhaled smoke are probably also important.²³ Occasionally, pulmonary problems may follow or be complicated by inhalation of gastric contents.¹³

Other factors Several other problems may develop after burns, and any one or more may contribute to death; these include the adult respiratory distress syndrome,²⁶ disseminated intravascular coagulation, acute renal tubular necrosis, fat embolism, acute upper gastrointestinal tract ulceration (“Curling’s ulcers”), hepatocellular necrosis, and deep venous thrombosis with subsequent pulmonary embolism. Of course, pre-existing natural (such as ischaemic heart and chronic obstructive airways) diseases may have an important role in the ultimate death of fire victims.²²

Post mortem injuries and artefacts of burned bodies

These are well recognised,^{1 7-9 18 27 28} but need to be considered in each case to ensure that wrong conclusions are not drawn. Many organs and tissues may be affected, but the major ones are:

SKIN

As noted previously, most skin burns seen on bodies removed from fires occur after death and show no vital reaction. When the body is exposed to considerable heat, skin and underlying soft tissues will contract and rupture or “split”; such *heat ruptures*, which are usually fairly short and superficial, but which may exceed 10 cm in length and be quite deep, can resemble lacerations or even incised wounds, and thus raise the suspicion of ante mortem injuries. Closer examination should reveal no vital reaction and no associated tissue bruising. Sometimes, “heat splits” occur during removal or transportation of the body; these are usually produced when the muscular rigidity (see below) is overcome, and tend,

therefore, to be around joints, particularly elbows and knees. Hairs on burned skin will be singed, scorched, or destroyed completely. Clothing may provide some or even total protection to the underlying skin from burning, but not necessarily from the effects of heat.

MUSCLES

It has been determined that all body muscle protein coagulates at temperatures above 65°C (149°F), and so heat generated by fires will cause muscle proteins to coagulate, to dehydrate, and to contract—*heat contractures*. As flexor muscles are stronger than extensors, arms, wrists, hands and, to a lesser extent, legs tend to be fixed rigidly in partial or almost total flexion. This produces the very commonly encountered “*pugilistic attitude*”, so-called because it is the posture adopted during fist-fights; it does not, of course, reflect attempts by the deceased at self protection.¹⁸ With more prolonged exposure to heat, muscles undergo drying, become pale, and are obviously “cooked”; under similar circumstances, the musculature of the anterior abdominal wall may rupture, exposing the intestines. Interestingly, deformities produced by heat contractures will cause an apparent shortening of a burned body when measured in the mortuary, and this, together with an apparent reduction in weight following water loss, can lead to early problems with identification if not appreciated.¹⁸

BONES

With very intense and continuous heat (of cremation), skin and soft tissues will be destroyed and underlying bones affected; such bones will “flake” and become brittle, and may fracture either spontaneously or with minimum trauma—for example, from falling masonry and during removal or transportation. Thus skull fractures may result in exposure of meninges and brain, simulating ante mortem injuries.

BRAIN AND MENINGES

Intense local heat will cook the brain and cause it to dehydrate and shrink, and, as explained immediately above, there may be associated skull fracturing. In addition, a “*heat haematoma*” may form; here, blood accumulates extradurally and resembles a traumatic extradural haemorrhage. The blood is soft and friable, light brown in colour and has a honeycomb appearance produced as its fluid component boils. The exact mechanism for its production is uncertain; it is thought to be derived from blood in venous sinuses or diploic and emissary venous channels, although its volume may exceed 100 ml; it is thought to be bilateral. As it is a post mortem phenomenon, and as considerable local heat is required for its formation, it is unassociated with ante mortem injuries to scalp, skull, or brain, and other manifestations of heat to these structures, as discussed above, will be seen; furthermore, its carbon monoxide saturation will be similar to that in the blood elsewhere in the body.

Circumstances: accident, suicide, or homicide?

Although the investigation, by the appropriate authorities, of the fire and its cause is often the most important aspect when dealing with fire deaths, the pathologist nevertheless has a valuable contribution to make, with observations and conclusions which may support or refute the views of the fire investigators.^{7 28}

ACCIDENT

Most deaths associated with fires are accidental. A range of circumstances is possible, but they tend to reflect either faulty domestic equipment, such as electrical wiring, cookers, heaters and fires, or some human error, such as dropping or inadequately discarding lighted cigarettes and matches, misuse of inflammable materials, and ignition of clothing from fires. In the latter context two precipitating factors should always be considered: first, the adverse influence of alcohol or other drugs, and appropriate analyses should always be requested. Second, precipitation by natural diseases, such as ischaemic heart, cerebrovascular and epilepsy—indeed, such diseases may result in death before the fire started.

SUICIDE

By burning, this is rare. Most victims have severe psychiatric problems, although self immolation is occasionally used to show political dissent. Usually, the person concerned pours petrol over him or herself and then ignites it. The ensuing burns are usually extensive, but relatively superficial, and the carbon monoxide saturation may not even be raised to any clinically relevant degree; heat may be an important factor in these deaths.

HOMICIDE

When considering deaths associated with fires, homicide is rare, but may be encountered under two circumstances:

Homicide before the fire, when the fire has been started deliberately in an attempt to destroy the body. In practice, such attempts are rarely successful, and post mortem examination should be able to indicate death before the fire started using criteria discussed earlier, and to identify appropriate injuries.

Homicide by fire, where the victim dies during or following a fire deliberately started by some other person—after igniting petrol thrown over the individual or after setting fire to a house. Under these circumstances, of

course, the pathologist can only determine the cause of death; the prosecution case rests entirely on the evidence of the other members of the investigating team.

- 1 Knight B. *The coroner's autopsy. A guide to non-criminal autopsies for the general pathologist*. Edinburgh: Churchill Livingstone, 1983:236–49.
- 2 Lawler W. Bodies recovered from water: a personal approach and consideration of difficulties. *J Clin Pathol* 1992;45:654–9.
- 3 Lawler W. The negative coroner's necropsy: a personal approach and consideration of difficulties. *J Clin Pathol* 1990;43:977–80.
- 4 Moritz AR, Henriques FC. Studies of thermal injury. II The relative importance of time and surface temperature in the causation of cutaneous burns. *Am J Pathol* 1947;23:695–720.
- 5 Cameron JM. In: Camps FE, ed. *Gradwohl's legal medicine*. 3rd edn. Bristol: John Wright, 1976:356–60.
- 6 Tedeschi CG. In: Tedeschi CG, Eckert WG, Tedeschi LG, eds. *Forensic medicine*. Philadelphia: WB Saunders, 1977:715–29.
- 7 Gee DJ. In: Mant AK, ed. *Taylor's principles and practice of medical jurisprudence*. 13th edn. Edinburgh: Churchill Livingstone, 1984:250–63.
- 8 Polson CJ, Gee DJ, Knight B. *The essentials of forensic medicine*. 4th edn. Oxford: Pergamon Press, 1985:320–7.
- 9 Gordon I, Shapiro HA, Berson SD. *Forensic medicine. A guide to principles*. 3rd edn. Edinburgh: Churchill Livingstone, 1988:134–40.
- 10 Knight B. *Forensic pathology*. London: Edward Arnold, 1991:281–93.
- 11 Bull JP. Burns. *Postgrad Med J* 1963;39:717–23.
- 12 Brown RF. In: Mason JK, ed. *The pathology of violent injury*. London: Edward Arnold, 1978:113–36.
- 13 Anderson RA, Watson AA, Harland WA. Fire deaths in the Glasgow area: I General considerations and pathology. *Med Sci Law* 1981;21:175–83.
- 14 Hirsch CS, Bost RO, Gerber SR, Cowan ME, Adelson L, Sunshine I. Carboxyhaemoglobin concentrations in flash fire victims. *Am J Clin Pathol* 1977;68:317–20.
- 15 Schwerd W, Schultz E. Carboxyhaemoglobin and methaemoglobin findings in burnt bodies. *Forens Sci Int* 1978;12:233–5.
- 16 Gormsen H, Jeppesen N, Lund A. The causes of death in fire victims. *Forens Sci Int* 1984;24:107–11.
- 17 Hill IR. Fires. *Med Sci Law* 1989;29:276–8.
- 18 Hill IR. Immediate causes of death in fires. *Med Sci Law* 1989;29:287–92.
- 19 Anderson RA, Harland WA. Fire deaths in the Glasgow area: III The role of hydrogen cyanide. *Med Sci Law* 1982;22:35–40.
- 20 Noguchi TT, Eng JJ, Klatt EC. Significance of cyanide in medicolegal investigations involving fires. *Am J Forensic Med Pathol* 1988;9:304–9.
- 21 Anderson RA, Watson AA, Harland WA. Fire deaths in the Glasgow area: II The role of carbon monoxide. *Med Sci Law* 1981;21:288–94.
- 22 Seviitt S. Death after burning. *Med Sci Law* 1966;6:36–44.
- 23 Demling RH. Burns. *N Engl J Med* 1985;313:1389–98.
- 24 Moritz AR, Henriques FC, McLean R. The effects of inhaled heat on the air passages and lungs. An experimental investigation. *Am J Pathol* 1945;21:311–22.
- 25 Purser DA, Buckley P. Lung irritation and inflammation during and after exposures to thermal decomposition products from polymeric materials. *Med Sci Law* 1983;23:142–50.
- 26 Getzen LC, Pollak EW. Fatal respiratory distress in burned patients. *Surg Gynaecol Obstetrics* 1981;152:741–4.
- 27 Dutra FR. Medicolegal examination of bodies recovered from burned buildings. *Am J Clin Pathol* 1949;19:599–607.
- 28 Simpson K. In: Simpson K, ed. *Taylor's principles and practice of medical jurisprudence*. 12th edn. London: Churchill Livingstone, 1965:329–39.