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Management of small fragment wounds in war: current research

G W Bowyer FRCS RAMC
Lecturer in Military Surgery

Department of Military Surgery, Royal Army Medical College, London

G J Cooper PhD
Senior Adviser/Trauma

P Rice BM MRCPath
Head of Pathology and Clinical Toxicology

Medical and Biological Divisions, The Chemical and Biological Defence Establishment, Porton, Sailsbury

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The majority of war wounds are caused by antipersonnel fragments from munitions such as mortars and bomblets. Modern munitions aim to incapacitate soldiers with multiple wounds from very small fragments of low available kinetic energy. Many of these fragments may be stopped by helmets and body armour and this has led to a predominance of multiple wounds to limbs in those casualties requiring surgery. The development of an appropriate management strategy for these multiple wounds requires knowledge of the contamination and extent of soft tissue injury; conservative management may be appropriate.

The extent of skin and muscle damage associated with a small fragment wound, the way in which these wounds may progress without intervention and their colonisation by bacteria has been determined in an experimental animal model. Results from 12 animals are presented.

There was a very small (~1 mm) margin of nonviable skin around the entrance wound. The amount of devitalised muscle in the wound track was a few hundred milligrams. Some muscles peripheral to the wound track also showed signs of damage 1 h after wounding, but this improved over 24 h; the proportion of fragmented muscle fibres in the tissue around the track decreased as time went on. There was no clinical sign or bacteriological evidence of the track becoming infected up to 24 h after wounding.

This preliminary work suggests that, in the absence of infection, the amount of muscle damage caused by small fragment wounds begins to resolve in the first 24 h after injury, even without surgical intervention.

Most wounds in warfare are caused by fragmenting antipersonnel weapons such as mortars and high-explosive shells. Over the past few years there have been changes in the design of these weapons to improve the probability of hit and increase the effective area of the munitions. The principal changes are the use of scattered submunitions (bomblets), and controlled fragmentation of the submunitions to create large numbers of similar fragments of predefined mass. The Gulf War saw these types of munitions used in large numbers.

These developments, and the widespread use of personal body armour has altered the pattern of injury in the wounded. The incidence of casualties with multiple wounds has increased, soft tissue wounds to limbs predominate *(1,2)* and the pathophysiology of the individual wounds may be different.

The treatment of penetrating wounds involving only the soft tissues remains controversial and there are differences in civilian and military priorities and practices. There are many supporters for a conservative approach, especially among those with experience of ballistic trauma in a civilian setting *(3)*. However, some

Correspondence to: G J Cooper, Medical Division, CBDE, Porton, Salisbury SP4 0JQ

military surgeons point to the different conditions that prevail on the battlefield, principally the delay in evacuation and the degree of bacterial contamination of wounds. Furthermore, those with more severe wounds that are more immediately life-threatening will take priority for operation, so a further delay is possible between reception of those with uncomplicated soft tissue wounds and their surgery. Because of these twin factors of delay and contamination, many military surgeons advocate a more interventional surgical approach *(1,4,5)*.

These considerations have notable implications for wound management in the military environment. A conservative approach that failed to control wound infection would have disastrous consequences for large numbers of casualties—up to 80% of casualties reaching forward surgical facilities may have multiple fragment wounds to limbs. A common, structured approach to wound management is necessary in war.

An approach employing wide débridement and excision is a well-established, safe approach for the repair of soft tissues and the control of infection. However, there are implications for operative time and the throughput of casualties; in addition, the excision of skeletal muscle that may actually be viable may not be warranted.

It is appropriate, therefore, to review periodically the approaches that form part of military surgical treatment protocols, in the light of changes in civilian practices, and developments in the nature and severity of wounds in war. The developments in fragmentation weapons now warrant such a review. The greater incidence of multiple wounds to limbs, and the low mass, very low energy transfer fragments warrant a re-evaluation of the role of a conservative approach to these wounds in war.

The Ministry of Defence is undertaking a research programme to elucidate the nature of the clinical threat from this new generation of antipersonnel fragments—the sole aim is to improve the treatment of military personnel injured in war.

The research will determine the biophysics and pathophysiology of uncomplicated limb wounds from these fragments. By ensuring that there is an understanding of the nature of the wounds, management protocols can be proposed and tested to ensure timely and effective treatment for these injuries to large numbers of servicemen in war.

This paper presents experimental work that describes the extent of soft tissue damage associated with a small fragment wound, and the way in which these wounds may progress without intervention. The tissues assessed were skeletal muscle and skin. The principal aims were threefold: (1) To judge the degree of soft tissue injury both within the track and peripheral to the track; (2) To assess resolution of the injury with time, and (3) To define the nature of foreign body and bacterial contamination of the wound.

Method

Twelve pigs were used, within the weight range 50–60 kg. The animals were sedated with intramuscular droperidol, then anaesthetised using halothane and intravenous pentobarbitone. Anaesthesia was maintained using the barbiturate, and 300 μg buprenorphine was administered intramuscularly. The pig skin was not cleansed and a bacteriology swab was taken to determine the normal skin flora.

A single, very low-energy transfer fragment wound was produced using a 200 mg steel cylinder (3 mm length, 3 mm diameter) at a measured velocity of about 500 m/s. The injury was to the hamstrings of the right hind-limb, avoiding bone and the major neurovascular structures. A sterile dressing was applied.

The animals were divided into three groups, each consisting of four pigs:

Group 1 had anaesthesia continued for 1 h, then they underwent wound exploration and sampling as detailed below.

Group 2 had anaesthesia continued, and wound bacteriology swabs were taken at 1 h. They were then allowed to recover from the anaesthetic. Analgesia and sedation (acylpromazine, ACP) was administered as required. At 6 h, the animals were re-anaesthetised, the wound was explored and samples taken as detailed below.

Group 3 had anaesthesia continued, and wound swabs taken at 1 h. They were then allowed to recover. At 6 h, wound swabs were taken under short-term anaesthesia. At 24 h the animals were re-anaesthetised for wound exploration and sampling as detailed below.

Wound samples

Skin. An ellipse of skin was cut around the entrance wound, to include the wound and a margin of normal skin. This was taken for histological examination.

Muscle. The fascia was divided and the wound track laid open using sharp and blunt dissection. Tissue was removed from the track, including clot and any tissue that did not meet the '4Cs' criteria for muscle viability (contractility, consistency, capillary bleeding and colour) *(6)*. The tissue was weighed and taken for histological examination. Bacteriological swabs were taken from the wound entrance and depths.

The animal was then killed while still under the influence of anaesthetic by a large dose of barbiturate. Samples of muscle were then taken from the track at 2 cm and 8 cm depths along the track, and at 2.5 cm and 4 cm peripheral to the track at these same depths.

Histopathology

Samples of excised wound tissue were placed in neutral buffered formalin and allowed to fix at room temperature for at least 48 h. Each sample was then trimmed, blocked into paraffin wax and cut to histological sections 5±2 μm thick on a Leitz microtome. All sections were routinely stained with Harris' haematoxylin and eosin and examined on a Zeiss Axioplan research photomicroscope at magnifications up to ×630.

Bacteriology

Transport swabs were plated onto the following media: columbia blood agar (5% sheep blood), cysteine lactose electrolyte deficient (CLED) agar, fastidious anaerobe agar (FAA) (5% horse blood). Columbia blood agar and CLED plates were incubated at 37°C for 24 h, extended to 48 h if negative at 24 h. The FAA plates were incubated in oxoid anaerobic jars for 48 h.

Bacteria were identified by colonial and microscopic morphology, Gram staining, and biochemical reactions (principally Analytical Profile Index, API).

Results

The mean velocity of the fragments was 549 m/s (SD 11 m/s).

The time to wound exploration and excision, the margin of non-viable skin and weight of excised tissues associated with the track are shown in Table I. The mass excised showed no statistically significant variation with time to excision (paired t test $P > 0.1$). The margin of skin viability was slightly less in the group excised at 1 h, but in all groups the margin was close to 1 mm, so there was thought to be no clinically significant difference.

Skin and muscle damage

The material excised from the wound track according to the '4Cs' criteria was all frankly necrotic on histological examination.

The principal observation in the muscle samples taken from the track and peripheral to the track after tissue failing the 4Cs criteria had been excised, was the absence of frank necrosis but the presence of fragmented fibres. This varied both with the depth down the track, the distance peripheral to the track, and the time at which the muscle was sampled. The extent of histological fragmentation of muscle samples is recorded in Table II.

In those samples showing marked muscle fibre framentation (65–75%) there were also features of significant haemorrhage both within and between fibres (designated intra- and intermuscular, respectively). Intermuscular acute inflammation and oedema were also evident in those samples. These features are shown in Fig. 1.

In those samples with less fragmentation there was no evidence of intramuscular haemorrhage, but there were

Table I. Weight of excised tissue failing the 4Cs criteria, and limit of skin viability. Four wounds were excised at each time point

Time of wound excision (h)	*Average weight of tissue excised (mg)*	*Average margin of skin non-viability (mm)*
1	160	0.7
6	260	1.4
24	220	1.4

Table II. Percentage muscle fragmentation. Samples were tissue remaining in the wound after excision of muscle failing the 4Cs criteria

Time of wound excision (h)	*Distance peripheral to track (cm)*	*Distance along track*	
		2 cm	*8 cm*
1	Adjacent	65–75%	65–75%
	2.5	20–25%	20–25%
	4	Nil	Nil
6	Adjacent	65–75%	50%
	2.5	10–15%	5–10%
	4	Nil	Nil
24	Adjacent	65%	<5%
	2.5	Nil	Nil
	4	Nil	Nil

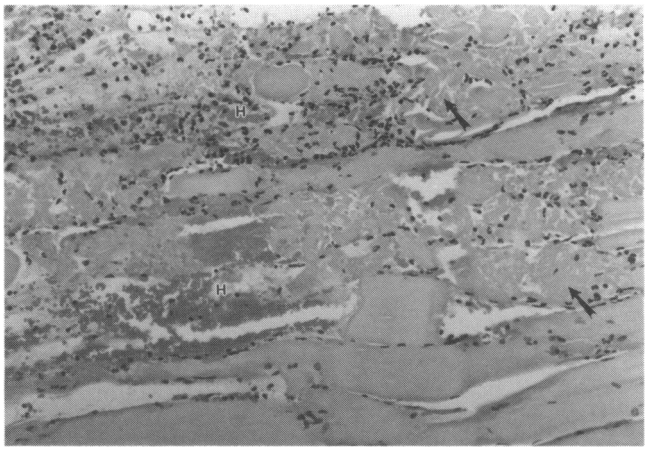

Figure 1. Muscle tissue, 1 h after injury. (Haematoxylin and eosin ×200.) Section of skeletal muscle from adjacent to the wound track, 2 cm from the entry wound. The section shows severe fragmentation of individual muscle fibres (arrows) and widespread inter- and intramuscular haemorrhage (H).

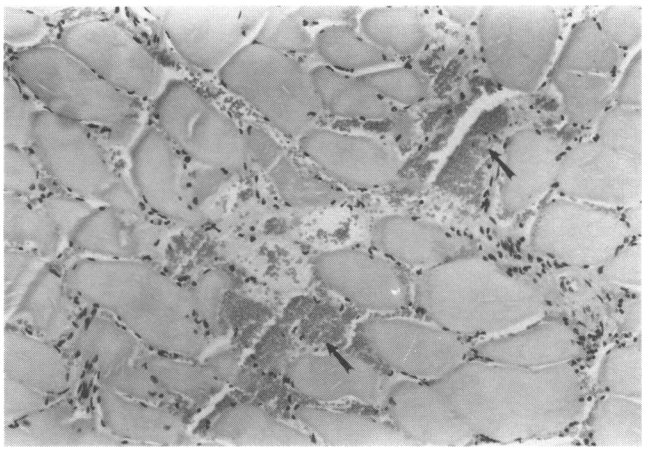

Figure 2. Muscle tissue, 24 h after injury. (Haematoxylin and eosin ×200.) Section of skeletal muscle from adjacent to the wound track, 2 cm from the entry wound. The section shows slightly oedematous, but intact, muscle fibres and minimal intermuscular haemorrhage (arrows).

inflammatory cells, red cells and oedema between fibres, shown in Fig. 2.

Bacteriology

Initial skin samples consisted predominantly of mixed staphylococci, with moderate to heavy growth. The staphylococcal flora included *S. capitis, warneri, sciuri, haemolyticus* and *lugendis*, all of which are regarded as non-pathogenic in the pig. Coliforms were also present in variable quantities: these were almost exclusively Acinetobacter species. Faecal streptococci were found in small numbers. There were no anaerobes detected.

The surface flora after injury showed little change, in type or numbers of bacteria, from the initial skin flora.

Only two wounds produced positive cultures from the wound track. One animal sampled at 1 h had a light mixed growth of non-pathogenic *Staphylococclus* spp and faecal streptococci in the wound track both close to the surface and in the depths of the wound. One wound sampled at 6 h had a light growth of mixed non-pathogenic staphylococci for the more superficial part of the wound track, but nothing in the wound depth. All anaerobic cultures were negative.

Discussion

The majority of fragments from modern antipersonnel munitions weigh only a few hundred milligrams. These are initiated at high velocity (> 1000 m/s), but their low mass and irregular shape leads to a rapid reduction in velocity. Most casualties surviving to reach surgical facilities will be struck by fragments with a velocity below about 600 m/s. The injuries produced in these experiments may be regarded as typical of the small fragment wounds to the soft tissues that are caused by the new generation of antipersonnel weapons. The fragments perforated (that is they produced an exit wound), but had little residual velocity on exit.

The study demonstrates the limited extent of skin damage with these fragments, extending only about 1 mm from the entry wound margin. The amount of devitalised muscle amounted to only a few hundred milligrams within the wound track; this did not change significantly when comparing excision at 1, 6 and 24 h.

The 4Cs criteria have been shown to be reliable discriminators between viability and non-viability in these experiments. All the material excised from the core failing to meet these criteria was shown to be frankly necrotic, with 100% fragmentation on histological examination. Some muscle, which would have been left *in situ* by these criteria, showed signs of injury with a proportion of fibre fragmentation, but this improved over 24 h. The inflammatory cell infiltrate is thought to be responsible for removing the fragmented fibres.

Conventional teaching, at least in the British Army, holds that ballistic wounds should be explored. Excision of skin should, however, be limited so that "only that which is clearly irreparably damaged should be excised" *(4)*. The wound "should be lined with healthy, bleeding, contractile muscle" at the completion of excision. The results here show that the extent of skin non-viability was very limited. Furthermore, there was little necrotic tissue within the track. The muscle damage peripheral to the track clearly improved over 24 h. These small fragment wounds are close to fulfilling the criteria for viable skin and a healthy muscle bed, even without surgical intervention.

These wounds did not become infected; however they were only followed for 24 h. It remains to be seen whether such results will hold over longer periods, particularly if the wounds were to be contaminated by clothing fibres as well as bacteria. Further work is planned to investigate this, and the options for the management of multiple fragment wounds.

The preliminary work presented here has shown that, in the absence of infection, most of the muscle damage in uncomplicated small fragment wounds may improve without surgical intervention, and conservative management in war may be appropriate.

References

1 Ryan JM, Cooper GJ, Haywood IR, Milner SM. Field surgery on a conventional battlefield: strategy and wound management. *Ann R Coll Surg Engl* 1991; 73: 13–20.

2 Spalding TJW, Stewart MPM, Tulloch DN, Stephens KM. Penetrating missile injuries in the Gulf war 1991. *Br J Surg* 1991; 78: 1102–4.

3 Marcus NA, Blair WF, Shuck JM, Omer GE. Low-velocity gunshot wounds to the extremities. *J Trauma* 1980; 20: 1061–4.

4 Kirby NG, Blackburn G. *Field Surgery Pocket Book*. London: HMSO, 1981.

5 Ryan JM, Cooper GJ, Maynard RL. Wound ballistics: contemporary and future research. *J R Army Med Corps* 1988; 134: 119–25.

6 Scully RE, Artz CP, Sako Y. An evaluation of the surgeons criteria for determining the viability of muscle during débridement. *Arch Surg* 1956; 73: 1031–5.

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