SPECIAL CONTRIBUTION
ballistic, injury; trauma, ballistic
injury; wound, ballistic injury

Ballistic Injury

Martin L Fackler, COL MC USA
Presidio of San Francisco, California

From the Wound Ballistics Laboratory,
Military Trauma Research Division,
Letterman Army Institute of Research,
Presidio of San Francisco, California.

Received for publication March 19, 1986.
Revision received June 25, 1986.
Accepted for publication August 11, 1986.

Presented at the Winter Symposium
of the American College of Emergency
Physicians in Orlando, Florida,
March 1986.

Address for reprints: Martin L Fackler,
COL MC USA, Wound Ballistics
Laboratory, Military Trauma Research
Division, Letterman Army Institute of
Research, Presidio of San Francisco,
California 94129.

Wound profiles made under controlled conditions in the wound ballistics laboratory at the Letterman Army Institute of Research showed the location along their tissue path at which projectiles cause tissue disruption and the type of disruption (crush from direct contact with the projectile or stretch from temporary cavitation). Comparison of wound profiles showed the fallacy in attempting to judge wound severity using velocity alone, and laid to rest the common belief that in treating a wound caused by a high-velocity missile, one needs to excise tissue far in excess of that which appears damaged. All penetrating projectile wounds, whether civilian or military, therefore should be treated the same regardless of projectile velocity. Diagnosis of the approximate amount and location of tissue disruption is made by physical examination and appropriate radiographic studies. These wounds are contaminated, and coverage with a penicillin-type antibiotic should be provided. [Fackler ML: Ballistic injury. Ann Emerg Med December 1986;15:1451-1455.]

INTRODUCTION

The belief that "high-velocity" projectiles cause wounds in which tissue damage is "well beyond that which is visually apparent,"[1] and that in treating these wounds "the surgeon must be much more aggressive in tissue excision — often excising tissue empirically and more widely than clinical judgment would normally dictate,"[1] is a widespread and dangerous fallacy.[2-8] Dugas and D'Ambrosia[9] reported that in only one-third of patients with gunshot wounds seen on the Louisiana State University Surgical Service in New Orleans was the wounding weapon known. Therefore, even if it were not fallacious, the usefulness of striking velocity as a determinant of treatment would be very limited in the clinical setting.

Data are presented showing the amount, type, and location of tissue disruption caused by six common bullets of varying velocities, masses, and constructions. Demonstrating the effect of other variables on tissue disruption exposes the error in the above generalizations based on the variable of velocity alone.[5]

The two mechanisms of tissue disruption caused by a penetrating projectile — *crush* (permanent cavity) of tissue hit by the projectile, and *stretch* (temporary cavity) of tissue surrounding the missile path — are shown (Figure 1). The sonic shock wave generated by the supersonic missile has been shown to cause no tissue displacement or detectable damage.[10]

MATERIALS AND METHODS

A method for capturing the entire projectile-tissue interaction in ordnance gelatin (25- x 25- x 50-cm blocks of a 10% by weight concentration shot at 4 C) that had been calibrated to reproduce the crush and stretch seen in living animal muscle was developed at the Letterman Army Institute.[1]

This method was used to measure the tissue disruption of the following bullets (Figures 2A-2E): a) .22 Long Rifle bullet; b) .38 Special lead round nose bullet; c) .45 Automatic full-metal-cased bullet; d) M-16 full-metal-cased bullet; and e) 7.62 NATO full-metal-cased bullet. A soft-point bullet fired from the 7.62 NATO cartridge case is shown (Figure 3).

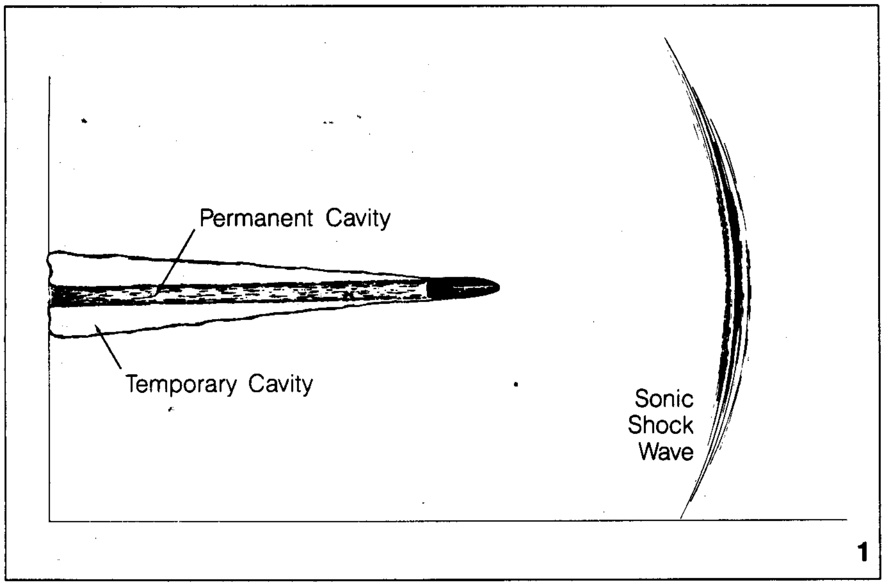


FIGURE 1. *Diagram of bullet path through tissue showing the sonic wave preceding the bullet. The stretch (temporary cavity) is formed after bullet passage by outward stretch of the walls of the bullet hole (permanent cavity).*

RESULTS

The amount, type, and location of tissue disruption caused by each bullet is portrayed as a scale drawing called the "wound profile." To facilitate comparison, the five wound profiles that show minimal disruption in the first 12 cm of projectile penetration are combined (Figures 2A-2E). The wound profile shown on a different scale (Figure 3) demonstrates marked disruption in the first 12 cm of projectile penetration.

An understanding of where maximum tissue disruption occurs is critical to predicting the effect of a given projectile on the body. The line drawn through the wound profiles (Figure 2) at 12-cm penetration depth facilitates comparison of tissue disruption *in the first 12 cm of the bullet path through tissue.* In most extremity soft-tissue wounds, total bullet travel does not exceed 12 cm. The disruption in this penetration distance varies little in the bullets compared, despite a fourfold difference in striking velocity and a 24-fold difference in striking kinetic energy.

Another line, drawn through the wound profiles at the 24-cm penetration depth, approximates the thickness of the average human abdomen from front to back.

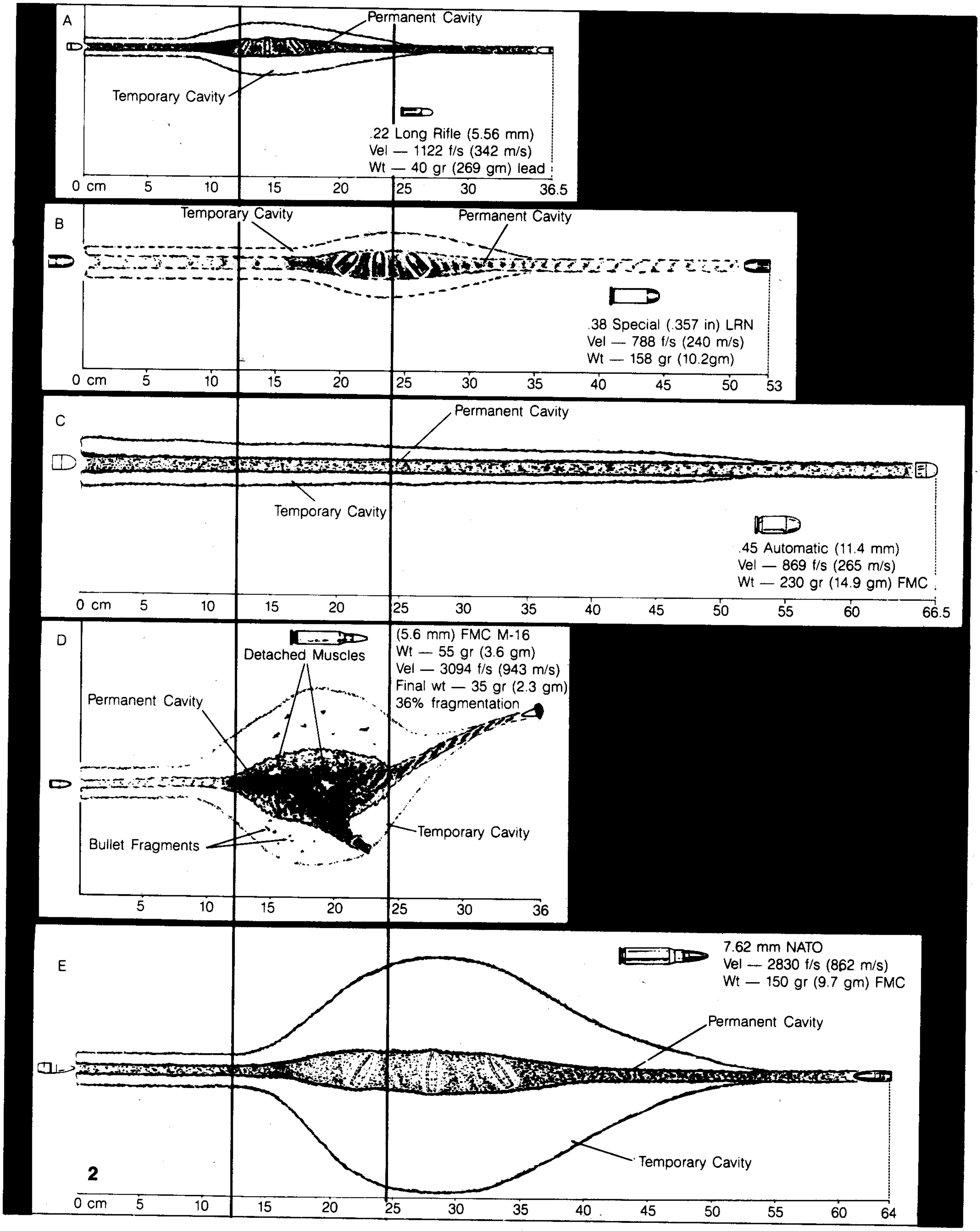
DISCUSSION

Based on the data presented, it is evident that for most soft-tissue extremity wounds (with less than 12 cm penetration), the high-velocity M-16 bullet would not be expected to cause more disruption than the low-velocity .22 Long Rifle bullet. Most certainly the M-16 has more potential for damage than does the .22 Long Rifle, but this potential generally is realized only with greater tissue penetration, as seen in the increased disruption between penetration depths of 12 and 24 cm (Figures 2A-2E). This explains why marked tissue disruption is typical on abdominal perforations with the M-16 bullet, while extremity wounds may be minimal. It is also understandable how viewing the marked disruption in an abdominal wound might mislead one to expect all wounds made by the M-16 to give a similar result. The wound profile format should clarify such misconceptions.

Generalizations based on velocity alone do not recognize the basic fact that in the projectile-tissue interaction a minimum of five variables, projectile mass, projectile shape, projectile construction, projectile striking velocity, and target tissue type, are involved. The *potential* for tissue disruption depends on projectile mass and projectile striking velocity; the *actual* amount, type, and location of the disruption depends on projectile shape, projectile construction, and target tissue type. This discussion will focus on the causes of actual tissue disruption and resultant wounds and management.

Of the bullets studied in the wound profiles, the M-16 (Figure 2D) and the 7.62 NATO (Figure 2E) bullets have the most sharply pointed shapes. This aerodynamic design allows less turbulence in flight, with better retention of velocity. The streamlined shape also causes minimal disruption in tissue, as long as the bullet remains undeformed and travels point forward, as illustrated in the first 12 cm of tissue penetration.

Bullets are rotated around their long axes by the rifling of the barrel in order to give them sufficient gyroscopic stability to maintain the point-


A
Permanent Cavity
Temporary Cavity
.22 Long Rifle (5.56 mm)
Vel — 1122 f/s (342 m/s)
Wt — 40 gr (269 gm) lead
0 cm 5 10 15 20 25 30 36.5
B
Temporary Cavity
Permanent Cavity
.38 Special (.357 in) LRN
Vel — 788 f/s (240 m/s)
Wt — 158 gr (10.2gm)
0 cm 5 10 15 20 25 30 35 40 45 50 53
C
Permanent Cavity
Temporary Cavity
.45 Automatic (11.4 mm)
Vel — 869 f/s (265 m/s)
Wt — 230 gr (14.9 gm) FMC
0 cm 5 10 15 20 25 30 35 40 45 50 55 60 66.5
D
Detached Muscles
(5.6 mm) FMC M-16
Wt — 55 gr (3.6 gm)
Vel — 3094 f/s (943 m/s)
Final wt — 35 gr (2.3 gm)
36% fragmentation
Permanent Cavity
Temporary Cavity
Bullet Fragments
5 10 15 20 25 30 36
E
7.62 mm NATO
Vel — 2830 f/s (862 m/s)
Wt — 150 gr (9.7 gm) FMC
Permanent Cavity
Temporary Cavity
0 cm 5 10 15 20 25 30 35 40 45 50 55 60 64

2

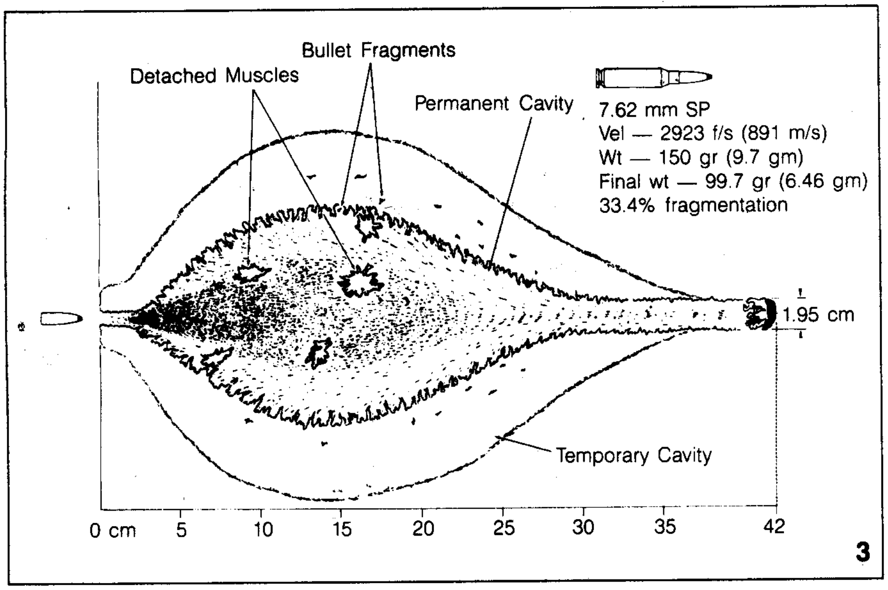


FIGURE 3. *Wound profile produced by the 7.62 NATO cartridge case loaded with a soft-point bullet of the same weight as the full-metal-cased bullet that produced the disruption shown in Figure 2E (scale differs from that in Figure 2E).*

forward position while traveling through air; however, this rotation is insufficient to stabilize bullets for point-forward travel in tissue. Pointed bullets that do not deform on striking tissue make a 180° rotation around their short axes (yaw) and terminate by traveling in the base-forward position, as shown on the wound profile of the 7.62 NATO full-metal-cased bullet (Figure 2E). The distance traveled in tissue by a particular bullet before it begins the rotation around its short axis is a relatively constant characteristic (unpublished data, 1985). This distance for a particular bullet is of critical importance in showing where along the bullet path maximum tissue disruption will occur.[5]

The increase in crush or permanent tissue disruption by the yawing bullet is determined by how much tissue the bullet strikes traveling sideways (long bullets would hit more tissue than would short bullets). This maximum amount of tissue crush occurs only at 90° of yaw, as shown on the wound profile of the 7.62 NATO full-metal-cased bullet (Figure 2E).

As bullet yaw increases, there is also a marked increase in stretch, or temporary cavitation. Temporary cavitation, however, is nothing more than a "splash" in tissue. It is the same mechanism that causes the increased turbulence when a diver hits the water sideways. The effect of temporary cavity stretch on tissue is similar to that seen in blunt trauma. Elastic tissues tolerate stretch better than do nonelastic tissues. This is exemplified by the marked permanent disruption of the relatively nonelastic liver, in comparison to the significantly lesser damage seen in more elastic tissue, such as muscle, after the same temporary cavitation.[11]

FIGURE 2. *Five wound profiles are compared. They show the disruption pattern caused by the following bullets: (A) .22 Long Rifle bullet; (B) .38 Special with lead round nose bullet; (C) .45 Automatic full-metal-cased bullet; (D) M-16 full-metal-cased bullet; and (E) 7.62 NATO full-metal-cased bullet.*

Comparison of the wound profiles of the 7.62 NATO full-metal-cased bullet (Figure 2E) with that of the same cartridge loaded with a soft-point bullet (Figure 3) reveals the large increase in tissue disruption obtained by changing the variable of bullet construction. The soft-point bullet causes two striking differences in tissue disruption — the permanent cavity is greatly increased in size, and it occurs at a shallower penetration depth. The massive tissue disruption at a penetration depth of 12 cm caused by the soft-point bullet is shown (Figure 3). This bullet is designed so that the tip flattens on impact. The bullet diameter thus is increased and its length decreased. For most soft-point bullets striking at a velocity of more than 600 m/s, the diameter increases by 2 to 2.5 times. This flattening destroys the bullet's aerodynamic shape, causing an increased temporary cavity as well as allowing the expanded bullet to make a larger hole by crushing more tissue.

Most of the massive permanent cavity caused by this soft-point bullet (Figure 3), however, is due to bullet fragmentation. On the upper right corner of the wound profile the final weight of the recovered bullet is given, and the percentage of bullet fragmentation (33.4%) is calculated. As the bullet deforms on impact, small pieces of it separate. In this case, 33.4% of the bullet's total weight leaves the main mass in the form of fragments. Each fragment crushes its own path through tissue as the multiple fragments spread out laterally away from the main projectile. These fragments penetrate up to 9 cm radially from the bullet path. The temporary cavity stretch comes after bullet passage (Figure 1), and in this case after the tissue has been penetrated by the many small fragments. Muscle that tolerates a given temporary cavity stretch from a nonfragmenting bullet with little gross disruption is severely disrupted and pieces of muscle are detached when the stretch that comes after it has been penetrated by a fragmenting bullet.[4]

The exit wound caused by this bullet in a shot through the thigh would pose little diagnostic problem because there would be a large hole (up to 13 cm in diameter) with much tissue loss. Despite the tissue loss, this large hole would ensure excellent drainage — a most favorable circumstance for healing to take place. Any attempt to close this wound primarily is contraindicated, and would be as great an error as removing viable-appearing tissue simply because the wound reportedly was made by a high-velocity weapon.

The M-16 wound profile (Figure 2D) shows a tissue disruption pattern that combines bullet yaw and bullet fragmentation. Its full-metal-jacketed bullet penetrates point forward for 12 cm, causing little disruption. As it yaws to 90°, the bullet flattens and breaks in half at the cannelure (knurled circumferential groove at the midportion of the bullet). The laterally flattened tip remains in one piece, but the part of the bullet behind the cannelure breaks into many small fragments, which have the same effect of multiply perforating the tissue as described for the soft-point bullet.

Thus the large permanent cavity associated with fragmenting bullets is seen, but at a greater depth of penetration because the bullet fragmentation depends on yaw to impose sufficient stress to break the bullet jacket. (The terms bullet jacket and bullet case are interchangeable, and refer to the harder metal covering that surrounds the core of the bullet.) The M-16 bullet does not break up at distances of more than 200 yards; at distances between 90 and 200 yards the bullet breaks into two large fragments rather than many fragments because of progressively less strain on the bullet at lower striking velocities.

CONCLUSION

Gunshot wounds are managed in the same manner as any other trauma in that the amount and location of tissue disruption are determined by physical examination and appropriate radiographic studies. Our results indicate that tissue disruption caused by bullets is no more mysterious or occult than that caused by the much-lower-velocity automobile. The fact that a particular high-velocity bullet causes massive tissue disruption (Figure 3) does not justify the conclusion that all high-velocity bullets cause massive tissue disruption along their entire tissue path, and that if the damage is not apparent then it must be occult. In wounds of the abdomen, in which laparotomy makes the tissue disruption more obvious, this erroneous conclusion is less likely to lead to harmful excess tissue removal than in extremity wounds. In the treatment of gunshot wounds, there are no objective data supporting removal of tissue that does not, on inspection, show signs of severe damage.

Our data strongly support the principle stated by Lindsey: "I will keep on treating the *wound*, not the weapon."[8]

REFERENCES

1. Gill W, Long WB III: *Shock Trauma Manual.* Baltimore, Williams & Wilkins, 1978, p 35.

2. Fackler ML: Wound ballistics, in Trunkey DD, Lewis FR (eds): *Current Therapy of Trauma*, vol 2. Philadelphia, BC Decker Inc, 1986, p 94-101.

3. Fackler ML: Letter to the editor. *Am Surg* 1984;50:515.

4. Fackler ML, Surinchak JS, Malinowski JA, et al: Bullet fragmentation: A major cause of tissue disruption. *J Trauma* 1984;24:35-39.

5. Fackler ML, Malinowski JA: The wound profile: A visual method for quantifying gunshot wound components. *J Trauma* 1985;25:522-529.

6. Fackler ML: Wound ballistics: Round 3. *Ann Emerg Med* 1985;14:936-937.

7. Fackler ML, Bellamy RF, Malinowski JA: Wounding mechanism of projectiles striking at over 1.5 km/sec. *J Trauma* 1986;26:350-354.

8. Lindsey D: The idolatry of velocity, or lies, damn lies, and ballistics. *J Trauma* 1980;20:1068-1069.

9. Dugas R, D'Ambrosia R: Civilian gunshot wounds. *Orthopedics* 1985;8:1121-1125.

10. Harvey EN, Korr IM, Oster G, et al: Secondary damage in wounding due to pressure changes accompanying the passage of high velocity missiles. *Surgery* 1946;21:218-239.

11. Fackler ML, Surinchak JS, Malinowski JA, et al: Wounding potential of the Russian AK-74 assault rifle. *J Trauma* 1984;24:263-266.

Reprinted from December 1986 Annals of Emergency Medicine©