

Chapter 9

Forensic Ballistics

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Abstract Forensic ballistics is the application of ballistics for forensic purposes. The basis is formed by wound ballistics. Two main mechanisms of injury are differentiated: the crush-mechanism resulting in the permanent cavity and the stretch-mechanism resulting in the temporary cavity. The missile-tissue interactions such as yawing, deformation, fragmentation, and bone contact are explained here and it is shown why the energy deposit or the missile velocity are not the sole or primary factors in determining the severity of a wound. The special wound ballistics of the head including indirect (“remote”) injuries in the

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brain and skull are discussed. Incapacitation is a necessarily occurring inability to perform complex movements and is therefore based on physiological effects independent of psychological mechanisms such as pain or surprise. Immediate incapacitation can only be caused by direct disruption of brain tissue and thus by penetrating gunshots to the head. Ballistic parameters and intracranial trajectories where sustained capability to act is possible are discussed. Rapid incapacitation is produced by massive blood loss via acute cerebral hypoxemia and subsequent unconsciousness. Targets of rapid incapacitation are the heart, aorta, and the truncus of the pulmonary artery. In cases of considerable ballistic injury to the lungs, liver, kidneys, spleen, large arteries or central veins, the latent period until incapacitation will be in the range of one or several minutes (*delayed incapacitation*). This potential for physical activity is not always exhausted due to psychological factors. Backspatter is biological material propelled retrogradely out of the entrance wound towards the firearm/the hand of the person shooting. Blood and tissue particles are accelerated by the subcutaneous gas effect, temporary cavitation, and tail splashing. Backspatter therefore is common in close-range gunshots to the head where blood and tissue can travel for several meters. The number of bloodstains can vary greatly and the stains are located in a semicircle of almost 180° in front of the entrance wound. Characteristic for backspatter are small or tiny droplet or splashing stains with the elongated shapes roughly aiming at the entrance wound. Magnification and appropriate lightning are necessary for investigating backspatter. DNA-analysis of stains can establish a clear link between a person or object and a clearly defined gunshot. Contact of a bullet with an intermediate target can alter the trajectory and stability of the bullet. Contact with fragile materials such as concrete, glass, asphalt, or gypsum-board regularly results in abundant deposits on the bullet, which can be visualized by SEM (scanning electron microscopy) and determined by X-ray microanalysis. Ductile materials such as wood and car body parts only transfer scarce deposits to the bullet which can be indicative of the intermediate target. This important trace evidence is not eliminated by subsequent perforation of tissue. Individualisation of deposits on FMJ bullets after perforation of a human body can be successfully carried out by PCR-typing of STRs and mitochondrial DNA. In cases of gunfights, this makes it possible to determine who was killed or injured by which bullet. The person shooting can be identified by additional comparison of rifling marks. Cellular material is recovered by swabbing the bullet, which should be protected against contamination and loss of material.

Keywords Backspatter · Capability to act · Cross sectional area · Deformation · Deposits on bullets · DNA-analysis · Energy deposit · Fragmentation · Gunshots to the head · Incapacitation · Intermediate target · Transfer of trace evidence · Wound ballistics

9.1 Introduction

Forensic ballistics can be briefly defined as the application of ballistics for forensic purposes. The major task of this heterogeneous discipline is the reconstruction of the events that produced a gunshot injury, fatal or not. So the very basis is formed by wound ballistics, i.e. the science of the penetration of a biological target. Every forensic pathologist should know how a gunshot injury is produced and wound ballistics therefore is the first field considered here. Although the principles of wound ballistics are not so complicated, bullets take a special position among the objects relevant in traumatology due to their physical characteristics: compared to other wounding agents, the mass is very small and the velocity is high. Unlike other blunt accelerated objects, this allows per se a deep penetration of tissue. But unlike sharp force, a dynamic penetration mechanism is effective which has not ended by the time the bullet exits. The resulting phenomena such as temporary cavitation can be misinterpreted and have given rise to myths and half-truths, which are discussed briefly later.

The second part on incapacitation is closely related to wound ballistics. It deals with the possible reaction of those being injured by a bullet, which can also be an integral part of reconstruction efforts.

A large variety of additional findings and evidence can be utilized for reconstructing the events leading to a gunshot injury such as gunshot residue or the geometry of the bullet tract. Since a complete presentation of all these aspects would clearly go beyond the scope of this chapter, a selection of topics was made. The third part is dedicated to blood and tissue particles exiting via the entrance wound: backspatter. The direction against the line of fire is the reason for the high evidential value of this phenomenon. The firearm and the body, especially the hand of the person shooting, are within the reach of the biological particles in cases of close-range gunshots and individualisation of backspatter stains can connect the person or gun to the gunshot injury at hand.

Trace evidence on spent bullets is the topic of the last part of this chapter. Contact of bullets with intermediate targets is an important field in exterior ballistics but also has wound ballistic and legal implications. A method for identifying deposits from intermediate targets on spent bullets is presented together with our experience in DNA-analysis of tissue deposits on bullets after perforation of a body.

9.2 Wound Ballistics

9.2.1 *Mechanisms of Injury*

Wound ballistics can be defined as missile-tissue-interaction [1]. The biological effect is therefore determined by:

- Parameters of the missile such as mass, caliber, velocity, shape, material or construction
- Parameters of the tissue such as density, elasticity, viscosity, and anatomical structure

Two different mechanisms of injury can be distinguished when a projectile penetrates a dense medium such as tissue [2, 3, 4]. Both represent distinct aspects of the same rapid fluiddynamic process and can therefore be regarded as theoretical abstractions essential for the understanding of wound ballistics.

9.2.1.1 Crush-mechanism and Permanent Tract

Tissue located in line with the trajectory is crushed by excessive pressures build-up directly in front of the tip of the moving projectile. The tissue is completely disintegrated, resulting morphologically in the permanent tract [2, 3, 5].

9.2.1.2 Stretch-mechanism and Temporary Cavity

Tissue located at the side of the trajectory is temporarily accelerated radially. This radial displacement of tissue creates a fusiform or conical cavity reaching a maximum 2–4 ms after the projectile has passed [3, 6, 7]. Tissue elasticity causes the cavity to collapse immediately, hence the name temporary cavity. The kinetic energy transferred to the tissue is expended after several cycles of expansion and contraction comparable to a pendulum: the temporary cavity is said to pulsate or “breathe” [3, 8, 9, 10].

During the formation of the temporary cavity, tissue is injured in three distinct modes (Fig. 9.1A–C), summarized as stretch-mechanism [2, 5] according to the predominating factor. Radial tissue displacement stretches the circumference of tubular areas of tissue around the permanent tract from $2\pi r$ to $2\pi(r+x)$ [3, 11]. Simultaneously, the thickness of these tubes is reduced resulting in compression of tissue. Moreover, the formation of the temporary cavity is a dynamic phenomenon. The whole process of temporary cavitation takes about 10 ms [7] during which the shape of the cavity changes continuously. Because of inhomogeneities and interfaces within the tissue, the cavity will push out along lines of least resistance such as fascial planes. This “principle of nonconfinement of the cavity” [3] produces an asymmetrical temporary cavity and consequently shear forces inside adjacent layers of tissue. The radial displacement of tissue and associated overpressures in the tissue around the expanding cavity gradually decrease with increasing distance from the tract like waves in calm water [3, 9].

So the stretch-mechanism of ballistic trauma is essentially nothing more than a localized blunt trauma analogous to a fist displacing tissue [1, 12, 13]. The maximum speed of moving tissue forming the boundary of the temporary cavity has been reported to be in the range of 40–100 m/s for so-called high-velocity missiles [14, 15, 16]. The structural integrity of tissue displaced by cavitation will generally

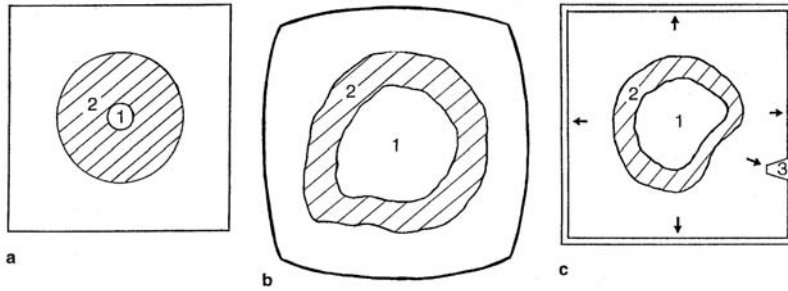


Fig. 9.1 Schematic illustration of the sectional area of soft tissue before and after a projectile has passed: temporary cavitation and stretch-mechanism. **A** Tubular tissue portions *before* the projectile has passed (1 = future trajectory; 2 = tubular tissue portion around the trajectory). **B** Tubular tissue portions *after* the projectile has passed at the moment of maximum temporary cavitation (1 = maximum) temporary cavity including future permanent tract; 2 = same tubular tissue portion as in **A**). The diameter of the tube has increased, resulting in stretching of tissue. Tissue inhomogenities are causing an asymmetrical shape of the tube (principle of nonconfinement), resulting in shearing of tissue. The thickness of the tube’s wall has decreased, resulting in compression of tissue. **C** Tubular tissue portions *after* the projectile has passed at the moment of maximum temporary cavitation, tissue confined in a rigid casing such as the head 1: the diameter of the temporary cavity is smaller compared to one in tissue not confined in a casing; 2: compared to **B**, the diameter of the tubular tissue circle is smaller, resulting in less stretching of tissue. The thickness of the wall of the tube is smaller, resulting in increased compression of tissue. Additionally, the surface of the tissue is pushed against the rigid casing (arrows), resulting in contusion of tissue remote from the tract analogous to blunt trauma; 3: prominent part of the casing (rim, edge, etc.). The prominence results in additional regional shearing and especially compression and contusion of tissue

not be completely destroyed [1, 5, 17, 18]. Therefore, the stretch-mechanism is not a reliable factor in wounding. The severity of injury decreases with increasing distance from the permanent tract, a zone of extravasation being located next to the permanent tract [3, 9]. Thus, the term “Seitenstoßkraft” (= sideways force) used in the past by German speaking experts in ballistics (e.g., [19]) probably illustrates the effect of the temporary cavity more vividly than the term cavitation, although the latter is perhaps more correct in terms of physics.

The sonic pressure wave (“shock wave”) originating from the impact of the projectile plays no part in wounding. Despite extremely high pressures, the duration of the amplitude is too brief (2 ms) to move or injure tissue [4, 20].

9.2.2 Missile-tissue-interactions

It becomes apparent by distinguishing two mechanisms of injury that the striking energy or the energy transferred to tissue only determines the potential of a given projectile for tissue disruption [2, 5, 12]. There are three crucial points for the

realization of this wounding potential. The first is the ratio of distribution of the energy between the crush- and the stretch-mechanism. The portion of the transferred energy used up in cavitation and stretching depends on ballistic parameters such as striking velocity, mass, and construction and generally increases with increasing velocity and decreasing mass [5, 21]. Therefore, bullets having the same striking energy made up of different velocities and masses (Fig. 9.2A,B) or even bullets with identical velocity and mass but different construction will produce very different injuries in identical tissue [2, 5, 22]. The second crucial point is the location of energy-transfer along the shot channel, considering the spatial aspect of a gunshot injury, and the third is the degree of elasticity of the tissues involved because tissue characteristics also determine the severity of a wound. In terms of the analogy used before: it does make a difference what kind of tissue is hit by a fist. The more flexible and elastic the tissue is, the less damage will be caused by the same amount of energy transferred to the stretch-mechanism [3, 5, 22]. Most soft tissues such as muscle, lung, skin, and bowel wall have the physical characteristics of a good energy or shock absorber, keeping the zone of

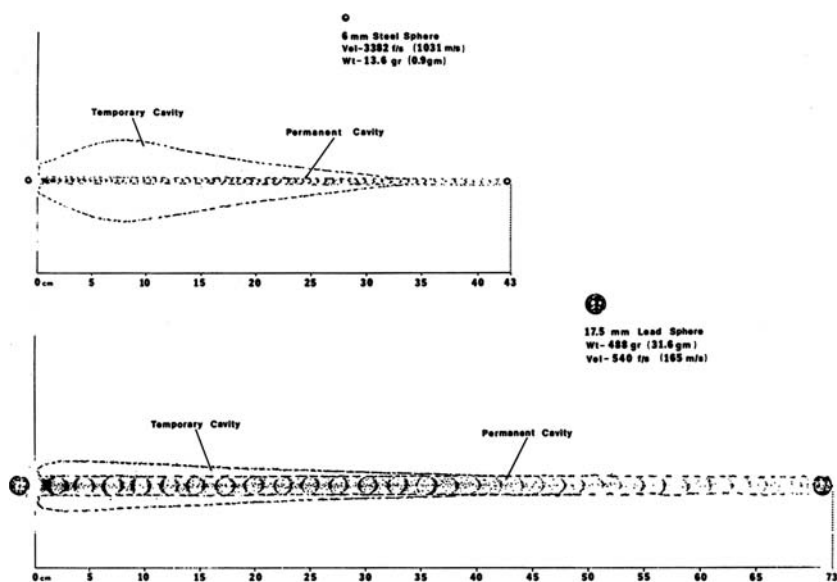


Fig. 9.2 Wound profiles according to Fackler [1, 2, 21] of two stable spheres depositing roughly the same amount of energy in gelatine blocks but producing strikingly different wound cavities. In the case of the fast, small and light-weight steel sphere (478 J), most of the kinetic energy is used up in the stretch mechanism, producing a large temporary cavity and a small permanent tract. In contrast to this, most of the large and heavy but slow sphere's energy (430 J) is used up in crushing of tissue, thus producing a large permanent tract but a small temporary cavity. This sharp contrast already demonstrates that gunshot injuries cannot be adequately described by terms like energy or energy deposit. The energy transfer of the fast sphere is also higher than that of the slow one

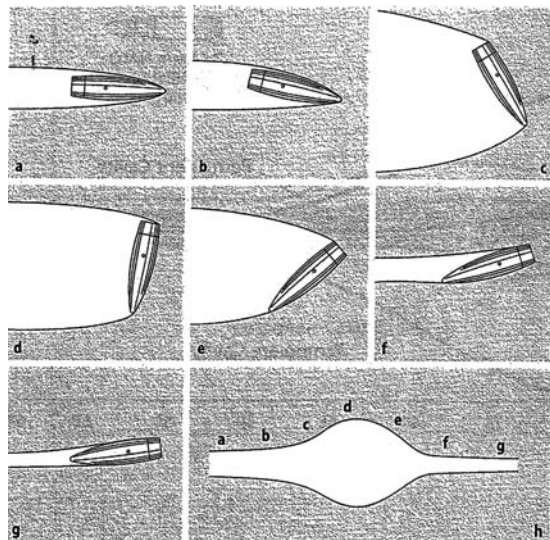
extravasation small [1, 5]. In contrast, in inelastic tissue such as the liver, spleen, or brain, the cavitation and resulting stretch-mechanism can produce devastating wounds up to a complete laceration or dispersion of the organ [8, 17, 22, 23, 24].

The general principles of wound ballistics outlined above are modified by yawing, deformation, and fragmentation of a projectile and by bone contact, all of which increase the missile's cross sectional area, i.e., the frontal surface of the bullet coming into contact with tissue.

In contrast to spheres, bullets commonly *yaw* or rotate around a lateral axis when moving in a dense medium such as tissue (Fig. 9.3). The angle between the trajectory and the long axis of the bullet increases due to destabilising forces until it reaches 90°, i.e., the bullet's long axis is perpendicular to the trajectory. The yawing motion then continues until 180° and the bullet will remain in this stable position where the base is the leading part. Increasing the cross sectional area of a bullet produces enhanced deceleration, reduced penetration depth and larger diameters of the wound cavities [2, 5, 9, 21]. The path of a projectile in a dense medium therefore shows three distinct sections, depending on the respective cross sectional area: narrow channel, increased cavities, and tail end (Figs. 9.3 and 9.4A).

Deformation will increase the missile's cross sectional area due to expansion (Fig. 9.4A,B) and a *fragmenting bullet* will also increase its cross sectional area (Fig. 9.5A,B), although distributed among multiple missiles of smaller mass and reduced penetration depth [16]. These so-called secondary missiles will produce multiple secondary shot channels, which, apart from their direct wounding effect, represent points of least resistance and will thus increase the

Fig. 9.3 Schematic illustration of the typical motion of a stable bullet during penetration of tissue ("yawing"). **a,b** The "narrow channel", no yawing. **c-e** Rotation around a lateral axis. This yawing motion considerably increases the cross sectional area of the bullet, thus increasing the diameters of the resulting wound cavities but decreasing penetration depth. **f,g** The "tail end". **h**: Schematic illustration of the resulting wound cavities in soap. The orientation of the bullet is indicated by the letters



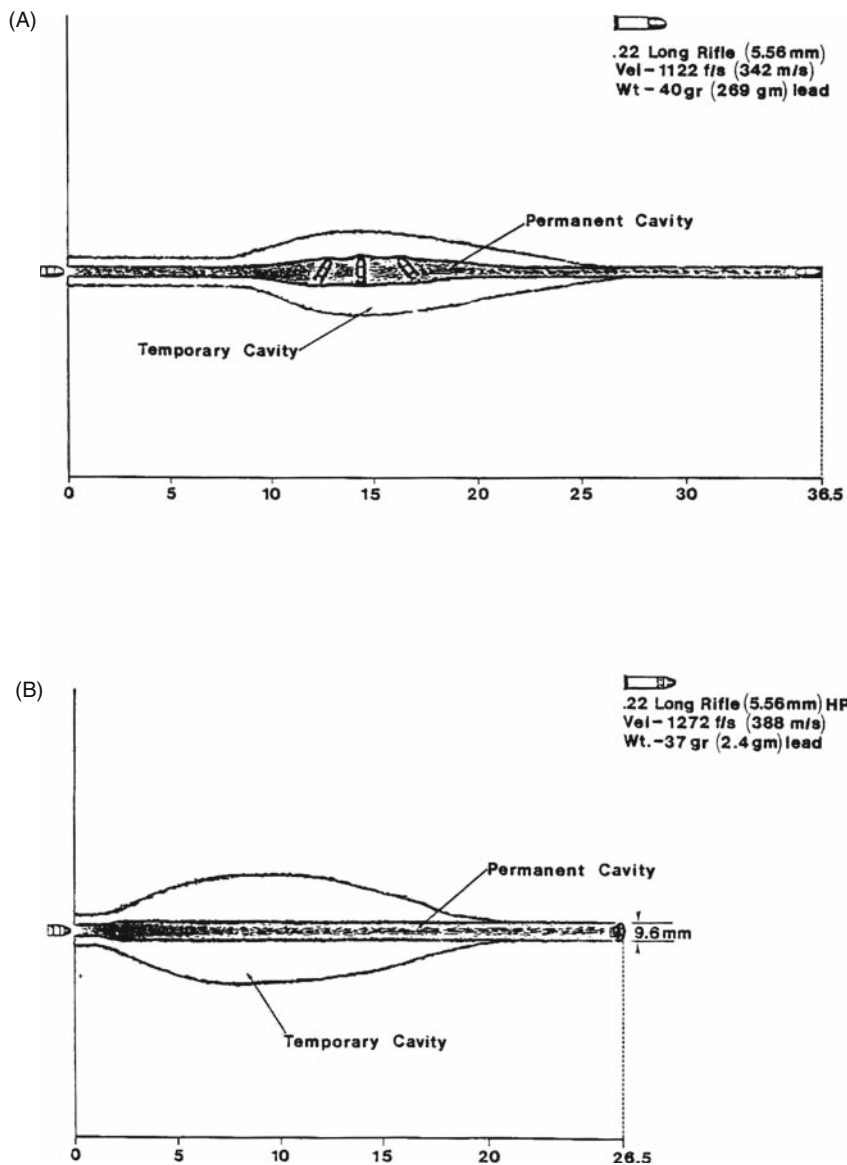


Fig. 9.4 Wound profiles according to Fackler [1, 2, 21] of two .22 lr bullets. **A** The stable lead bullet produces a long narrow channel and rather small wound cavities. **B** The deforming hollow point bullet, however, produces a short narrow channel and rather large wound cavities while the penetration depth is reduced. This difference is solely caused by the differing constructions since not only energy but also mass and velocity of the two bullets are almost identical

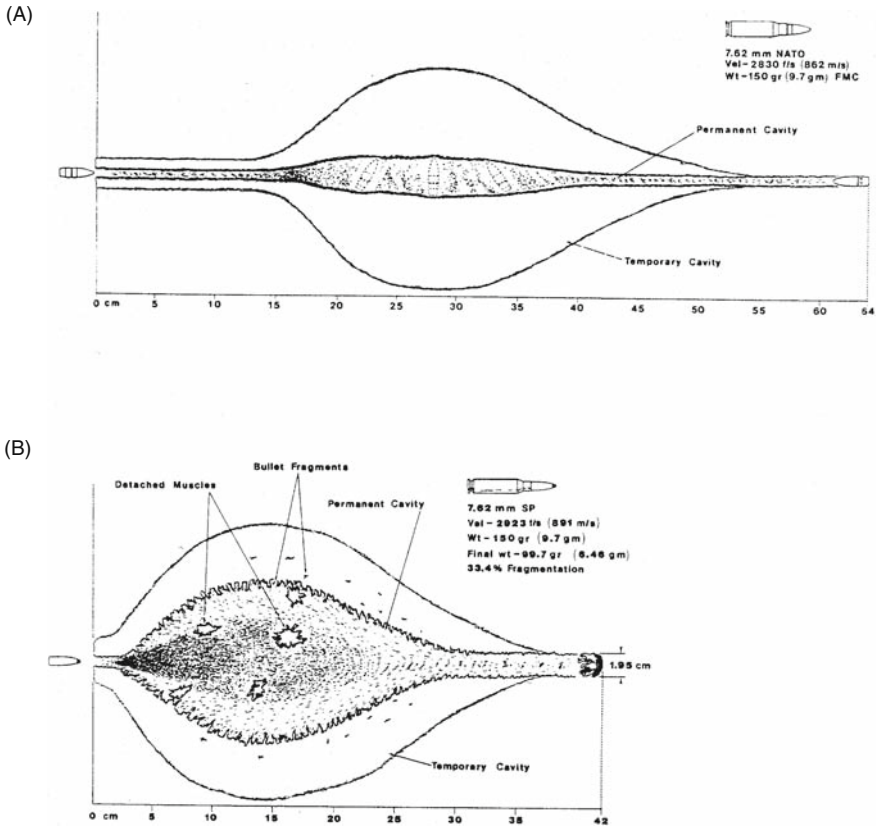


Fig. 9.5 Wound profiles according to Fackler [1, 2, 21] of two 7.62×51 mm NATO (.308 Winchester) bullets. **A** The stable FMJ bullet causes the typical wound morphology including a long narrow channel. **B** The soft point bullet possessing the same mass and velocity does deform and fragment due to the differing construction. This results in a non-existent narrow channel and in huge wound cavities

susceptibility for the subsequent stretching of tissue. There is a synergistic effect of secondary missiles and cavitation [2, 16, 23, 25].

Compared to soft tissue, bone is a hard and dense material, reducing the penetration depth of bullets that strike it. Depending on the construction, material, and velocity of the projectile, bone contact favors deformation, fragmentation, and increased yaw [26, 27] with the above-mentioned effects. Frequently, bone contact causes additional secondary missiles in the form of bone fragments [7, 23, 26, 28, 29]. The accelerated bone fragments travel in different directions in the vicinity of the bone, to a small extent even against the line of fire [28, 30]. The effect of secondary bone fragment missiles is analogous to bullet fragments including secondary shot channels (Fig. 9.6), enhancing the severity of the wound [5, 7, 23, 28].

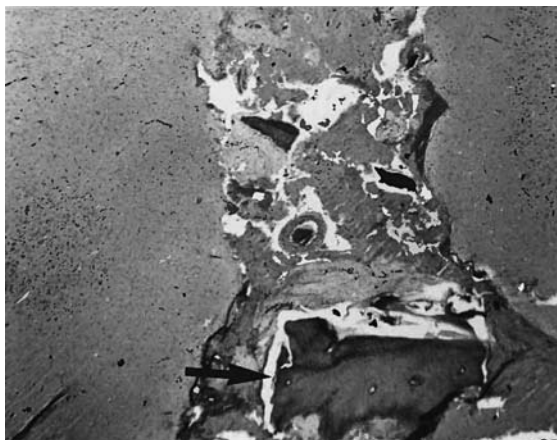


Fig. 9.6 Microscopical appearance of intracerebral bone fragment (arrow) at the end of a retracted secondary shot channel 3 cm in length (Hematoxylin & Eosin)

9.2.3 Energy Deposit, Energy Transfer, and High-velocity Missiles

The energy deposit concept states that the kinetic energy ($E = mv^2/2$) used up in the production of a gunshot wound determines the severity of the wound [35]. The energy transfer concept additionally considers the distribution along the shot channel [35]. In both concepts, the missile-tissue interaction is considered a black box and can therefore be reduced to an abstract physical value. However, the kinetic energy is only secondarily derived from two basic ballistic parameters, mass and velocity. Since the velocity is squared in this equation, the popular thesis about the primacy of velocity over mass for the wounding potential appears consistent but nevertheless is based on an oversimplifying approach. Therefore, the popular differentiation of high-velocity and low-velocity missiles should be dropped – the lack of a reasonable border velocity has even led to the introduction of medium-velocity [32]. So-called high-velocity missiles do not necessarily carry a high wounding potential and extremity amputations produced by tangential high-velocity gunshot wounds are nothing more than abstruse legends.

The differentiation of two ideal penetration mechanisms and the dynamics and variability of the missile-tissue interactions clearly demonstrate that a secondary ballistic parameter such as kinetic energy cannot adequately describe a gunshot wound. Instead, basic parameters such as mass, velocity, caliber, degree of deformation and fragmentation, length of the narrow channel, and penetration depth are decisive in determining the severity of a gunshot wound [33]. As a consequence, studies investigating the mass of non-vital tissue as a function of energy transfer [34, 35, 36, 37, 38, 39, 40] were not successful in establishing a close relationship but only found a tendency, which is not surprising considering the abstract relationship between energy transfer and the possible destruction work.

9.2.4 Special Wound Ballistics of the Head

In intracranial gunshot wounds, several of the above-mentioned factors enhance the degree of tissue disruption. The inelastic quality and the high water content of brain tissue make it per se very vulnerable to cavitation and stretch-mechanism. The penetration of the skull can imply the generation of secondary missiles in the form of bone (Fig. 9.7) or bullet fragments [28, 41, 42, 43, 44] and a tendency towards early tumbling or deformation of the bullet. Kirkpatrick and DiMaio [44], for example, were able to demonstrate intracerebral bone chips solely by digital palpation of the brain in 16 out of 42 cases of civilian gunshot wounds to the brain.

Even more important, intracranial trajectories gain a new quality by the rigid skull functioning as a non-yielding wall. Because brain tissue is almost incompressible, intracranial temporary cavitation and surrounding overpressure meet counter-pressure from the skull. The skull will, so to speak, try to overcome the principle of nonconfinement of the cavity by denying the free space necessary for a gradual decrease of radial tissue displacement and associated overpressure. The volume of the intracranial temporary cavity will consequently stay smaller than a cavity formed under identical conditions in tissue not confined in a casing. Intracranial overpressures around the expanding temporary cavity, however, clearly exceed the pressures found in nonconfined tissue [4, 10, 45, 46]. These high dynamic pressures, the asymmetric shape of the temporary cavity, and unilaterally fixed tissue structures lead to shear forces within brain tissue. The unyielding skull does not allow the brain to expand, so the brain will transfer the overpressures to the skull. In other words, the brain's surface gets pushed with great force against the inner table of the neurocranium and the brain stem gets forced down into the foramen magnum. Consequently, the layer of cerebral tissue

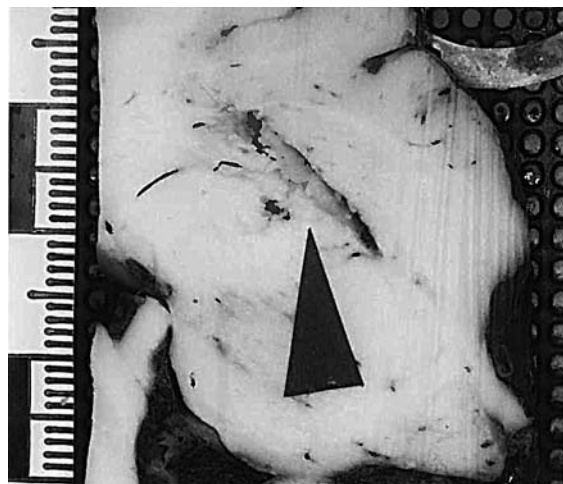


Fig. 9.7 Intracerebral bone fragment (*arrow*) at a distance of several cm from the tract and surrounded by petechial haemorrhages. The 3rd and 4th ventricles left of the arrow are filled with blood

Fig. 9.8 Subarachnoid haemorrhage and cortical contusion zone in the form of ball haemorrhages (Hematoxylin & Eosin)



between temporary cavity and skull is compressed much more strongly than tissue not confined in a rigid casing and shearing of brain tissue is increased by bone structures projecting into the skull cavity.

Analogous to blunt trauma, enhanced compression can result in contusion of brain tissue discernible as (cortical) contusion zones in superficial layers of the brain remote from the trajectory [28, 44, 47, 48, 49] (Fig. 9.8). The stretching and especially shearing of tissue is responsible for intracerebral petechial hemorrhages remote from the tract in the form of classical perivascular ring hemorrhages or spherical hemorrhages [28, 41, 43] (Fig. 9.9). They are simply the result of an enlarged zone of extravasation due to the enhanced effect of temporary cavitation. Preferential neuroanatomical sites are more central parts of the brain such as the basal ganglia, midbrain, pons, and cerebellum (Fig. 9.9).

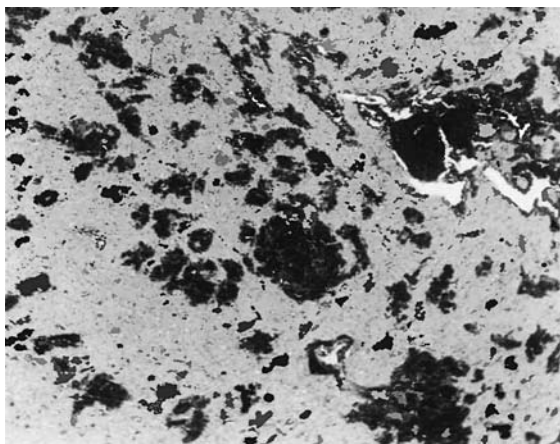
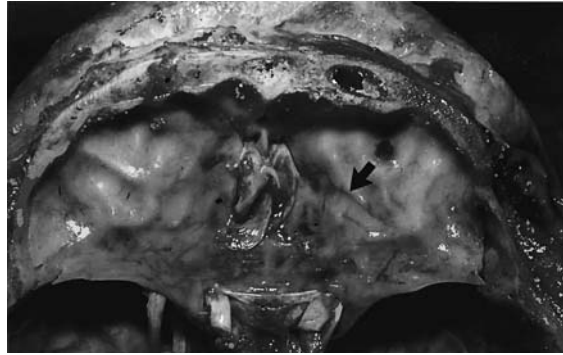


Fig. 9.9 Numerous intracerebral haemorrhages in the left thalamus remote from the tract. The macroscopical appearance is similar to the haemorrhages depicted in Fig. 9.7 (Hematoxylin & Eosin)

Fig. 9.10 Indirect fracture (*arrow*) in the right anterior cranial fossa after a suicidal gunshot with a .45 ACP FMJ bullet with the wound tract above the middle and posterior cranial fossa. There was immediate incapacitation in this case



The skull will at first be slightly stretched by intracranial overpressures. If the skull's capacity to elastically stretch is surpassed, there will be indirect skull fractures, i.e., fracture lines without contact to the primary bony entrance and exit defects. Because the base of the skull is inhomogenous and less resistant to stretching than the vault, preferential locations are the roofs of the orbitae (Fig. 9.10) and the ethmoidal plates in the anterior cranial fossa [50]. While secondary radial fractures originating from the gunshot defects are induced by the bullet's impact, tertiary concentric fractures connecting the radial fracture lines (Fig. 9.11) are indirect heaving fractures [51, 52, 53] functioning as additional stress relief for internal overpressures. If the internal pressures are high enough, indirect skull fractures will combine to an "explosive" type of head injury [54] with comminuted fractures of the skull and laceration of the brain (Fig. 9.12).

So the rigid skull which protects the brain from most blunt trauma also makes the brain by far the most susceptible organ in the body to penetrating ballistic injury. Intracranial pressure peaks and its effects vary greatly, depending on

Fig. 9.11 Indirect (concentric) heaving fractures around an exit wound in the left temple after gunshot with a .44 lead projectile. The concentric fracture lines are produced indirectly by intracranial overpressure while the radial fracture lines are caused directly by impact of the projectile

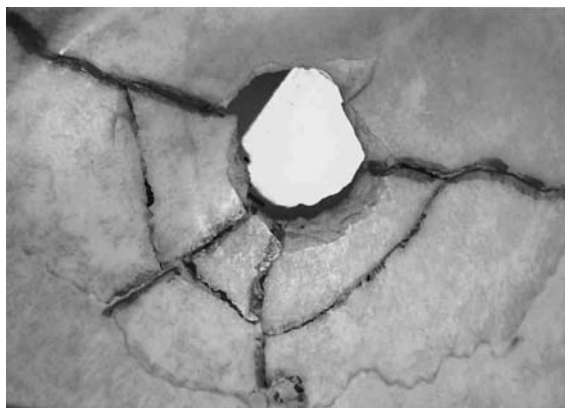


Fig. 9.12 Partial exenteration of the brain after a close-range gunshot with a 12/70 shotgun using a Brennecke slug (lead projectile, mass approx. 30 g, velocity approx. 450 m/s). This clearly demonstrates that no “high-velocity” missile is required for such an “explosive” type of head injury



ballistic and anatomical parameters. Mathematically, the peak pressures recorded vary in direct proportion to the projected cross-sectional area of the missile and the square of its velocity but in inverse proportion to the distance from the point of origin [4, 10, 45, 46]. In more practical terms, bullet wounds from handguns and rifles differ considerably with regard to their effect in penetrating gunshots to the head. Bullets from conventional handguns can produce indirect skull fractures and pronounced cerebral tissue disruption [28]. Centerfire rifles, whether military or hunting, almost invariably cause a strong “explosive” effect with comminution of bone and laceration of at least part of the brain [42, 55, 56, 57]. Hits from shotguns differ substantially depending on the range of fire. Close range shots have a tremendous effect similar to centerfire rifles by literally riddling brain tissue and blasting the skull [58, 59] (Fig. 9.12).

9.3 Incapacitation

Determining a person’s capability to act following a gunshot wound can be of major importance in crime scene reconstruction and in differentiation between homicide and suicide. If a person who has been shot is not able to shoot back, attack or escape, certain events can be ruled out. This can assist the identification of the person who fired the gun and the reconstruction of the sequence of shots and activities. Therefore, questions concerning the possibility of physical activity following a given gunshot wound are repeatedly raised in court.

Stopping power is a term very similar to incapacitation, although the point of view has changed to one from behind the trigger. The use of firearms by police officers is frequently intended to stop the momentary activity of a suspect by rapid incapacitation. Because stopping power and incapacitation both address the same phenomenon, this issue is also relevant to law enforcement agencies.

9.3.1 Definition and Mechanisms of Incapacitation

There are a variety of definitions mostly concentrating on the inability to act in a conscious and purposeful manner such as escaping or attacking [60, 61]. Others have used complex definitions primarily based on the degree of consciousness and on underlying neurophysiological processes [62]. In this context, a functional definition of capability to act independent of the state of consciousness or intention will be used: capability to act is the ability to participate in the interaction between victim and perpetrator or victim and environment, resulting in discernible events or stains. Thus, incapacitation is an early and necessarily occurring inability to perform complex and longer lasting movements. The activity does not have to be appropriate in the situation at hand. For example, pocketing or storing the firearm following a suicidal gunshot is not always achieved intentionally or purposefully. Incapacitation is based on physiological effects independent of psychological mechanisms such as pain or fright because it has to be independent of the victim's "cooperation".

Reliable incapacitation based on physiological effects according to the above definition is closely connected to death and can only be produced by decreasing the functioning capability of the central nervous system (CNS) [12, 63, 64]. The two sole mechanisms to accomplish this are direct disruption of brain tissue or indirect elimination of the CNS by cerebral hypoxemia from bleeding, both causing unconsciousness [12, 63, 64, 65].

There is no other way to prevent a determined person from further action. Many victims will collapse immediately when hit by a bullet as will some who were missed but think they were hit. They do so on a psychological basis but this is inconsistent and erratic [65]. The only thing to count on is that it will not work in the case of determined and highly motivated persons or in the case of those under the influence of drugs or adrenalin. Excessive pain, for example, must first be perceived and then this perception of pain must cause an emotional reaction. So reliable incapacitation is solely based on physiological effects independent of any unpredictable psychological factors.

Another alleged mechanism of incapacitation is high energy-transfer [31] or high energy deposit [66]. Energy deposit is an abstract value, considering neither the way (crush/stretch) or location energy is transferred nor the type of tissue involved (compare Sect. 2.2). Therefore, the amount of energy is of limited value in predicting effects in an actual shooting. The momentum transferred to the target does not knock the human body down or drive it significantly backwards [65, 67], even if shown so a thousand times on television. The impulse transferred to an adult from a .45 ACP round results in a negligible backwards motion of approximately 5 cm/s [67]. Injury or even incapacitation from shock waves [68] or a mysterious "nerve shock" postulated especially in hunting are not supported by a single experiment or by theoretical considerations [20, 33, 69].

Hampered physical activity but not incapacitation may be produced by injuries to sensory or optic brain areas, the spinal cord and large peripheral nerves, static structures such as long bones or joints and by pneumothorax [33].

9.3.2 Immediate Incapacitation

Instantaneous incapacitation can only be produced by direct disruption of brain tissue [64, 65]. In the case of gunshot injuries causing acute bleeding, the speed of blood loss is too slow and compensation mechanisms are too effective for immediate loss of consciousness and incapacitation [64]. So the only way to stop immediately the activity of another person are penetrating gunshots to the head.

Because of the enhanced intracranial tissue disruption and the functional significance of the CNS, craniocerebral gunshot wounds result in a high early mortality rate of 90% and more [70, 71] and commonly in immediate incapacitation. However, in the last century, numerous publications reported sustained capability to act following penetrating gunshot wounds of the head. Since these were case reports or small case series, no systematic correlations between wounding and capability to act could be detected. Therefore, all accessible cases have been reviewed [72]. A large number of case reports had to be excluded from this reexamination because of doubtful capability to act or lacking morphological or ballistic documentation; there remained 53 case reports from 42 sources for systematical analysis.

Favourable conditions for sustained capability to act are present in cases where the additional wounding resulting from the special wound ballistic qualities of the head are minimized. Thus, more than 70% of the guns used fired slow and light-weight bullets: 6.35-mm Browning, .22 rimfire or extremely ineffective projectiles such as ancient, improper, or self-made missiles (Table 9.1). Only two large handguns resulting in intracerebral wounding were used: A .38 spec. bullet which solely wounded the base of the right temporal lobe [73] and a .45 lead bullet which seriously injured the left frontal lobe but whose trajectory was limited to the anterior fossa of the skull [74]. A centerfire rifle or a shotgun from close-range were never employed in cases of intracerebral tracts. A coincidence of several lucky circumstances made sustained capability to act possible in two cases of military centerfire rifle bullets passing longitudinally between the frontal lobes without direct contact with brain tissue [75, 76].

Of the trajectories, 28% were outside the neurocranium. At least 70% of the craniocerebral tracts passed above the anterior fossa of the skull (Table 9.2), wounding the frontal parts of the brain. Apart from a neurophysiological approach, this preference can additionally be explained by the base of the anterior cranial fossa and the sella turcica area which serve as a bony barrier protecting those parts of the brain located in its "shadow" relative to the trajectory against cavitation tissue displacement and associated overpressures. This is particularly true for the brain stem. Intracerebral trajectories not located above the anterior

Table 9.1 Summary of the firearms used

Firearms used	<i>N</i> = 53
Pocket-revolvers and old, low-energy handguns	<i>n</i> = 10 (19%)
Modified blank handguns or selfmade/improper ammunition	<i>n</i> = 5 (9%)
.22 pistol	<i>n</i> = 2 (4%)
5.6-mm rimfire rifle	<i>n</i> = 4 (7%)
6.35-mm pistol	<i>n</i> = 13 (25%)
7.65-mm pistol	<i>n</i> = 8 (15%)
.45 Colt revolver	<i>n</i> = 1 (2%)
.38 special revolver	<i>n</i> = 1 (2%)
Centerfire rifle	<i>n</i> = 3 (6%)
Shotgun (contact shot)	<i>n</i> = 1 (2%)
Not exactly known	<i>n</i> = 5 (9%)

fossa were caused by slow and light-weight bullets preferring one temporal lobe. Additionally, one parietal and one occipital lobe were each injured once by a very ineffective projectile [77] and by a 7.65-mm bullet reduced in velocity [78]. Morphological signs of high intracranial pressure peaks (cortical contusion zones, indirect skull fractures, perivascular hemorrhages) and secondary missiles were poorly documented but appear to be very rare.

Therefore, sustained capability to act following craniocerebral gunshots is very unlikely if one of the following two conditions are fulfilled:

1. Use of a firearm from about 9-mm Parabellum upwards in terms of penetration power and wounding potential (large handguns, centerfire rifles). To increase further the probability of incapacitation, intracerebral trajectories above the anterior cranial fossa or very short ones can be excluded.
2. Definite occurrence of signs of high intracranial overpressures: indirect skull fractures, intracerebral petechial hemorrhages remote from the tract and cortical contusion zones.

Incapacitation can be determined beyond any doubt if central nervous centers essential for physical activity are wounded directly. In the literature reviewed [72], not a single case of injury to the brain stem, the diencephalon, the cerebellum, or major paths of motor conduction has been described. The

Table 9.2 Summary of brain areas injured or wound tracts

Wounded brain areas or trajectories	<i>N</i> = 53
One or both frontal lobes	<i>n</i> = 17 (32%)
Transtemporal (no autopsy or no precise cerebral morphological findings)	<i>n</i> = 11 (21%)
One temporal lobe	<i>n</i> = 6 (11%)
Right parietal lobe	<i>n</i> = 1 (2%)
Extraneurocranial	<i>n</i> = 15 (28%)
Intraneurocranial but extracerebral	<i>n</i> = 3 (6%)

Table 9.3 Targets of immediate incapacitation

Upper cervical spinal cord, brain stem including the mid-brain, diencephalon incl. central grey matter, cerebellum, major paths of motor conduction, motor cortex

central grey matter can also be included, for there was only one “grazing” shot of the most ventral parts of the caput of the caudate nucleus [79]. The motor cortex was injured once by a slow projectile probably restricting wounding to the permanent tract and resulting in acute hemiplegia [77]. Immediate incapacitation, therefore, can only be produced reliably by injury to the brain regions listed in Table 9.3.

9.3.3 *Commotio Cerebri and Cerebral Pressure*

Theoretically, incapacitation from gunshots to the CNS can result from primary or from secondary effects of the bullet. The major primary effect is disruption of brain tissue resulting in focal disturbances or loss of consciousness and has been discussed above. *Commotio cerebri* where the major symptoms are immediate unconsciousness and loss of muscle tone may be another primary effect. The generation of *commotio cerebri* has been discussed in cases of penetrating ballistic head injury for a long time [77, 80, 81] and was thought to originate from the momentum transferred from the impacting projectile to the skull. However, the mechanogenesis of *commotio cerebri* is a matter of sudden acceleration of the skull, which by means of inertia results in wounding of the brain. The crucial physical parameter in this is the change of impulse per unit time or in other words the product of mass and acceleration of the head. A maximum acceleration of the skull will be achieved when the mass of the impacting object is equivalent to that of the head and when the velocity of the object is relatively high.

A projectile has a very small mass but a very high velocity resulting in an ultra-short time span during which the projectile is acting upon the skull. Because of inertia, the skull as a whole will not really move during transfer of the impulse. Instead, during impact there will be a high transfer of momentum and energy locally but no direct load on the entire skull. The result is the perforation of the skull without marked acceleration of the head. The penetrating character of gunshots to the head thus does not allow a substantial transfer of impulse to the head as a whole. In accordance with these theoretical considerations are observations from battlefields reporting lack of *commotio cerebri* in penetrating gunshots to the head [49, 82].

Cerebral pressure is the major secondary effect of ballistic brain injury. However, the latent period in the range of minutes until the intracranial pressure rises substantially in animal experiments [83, 84, 85, 86] is too long to produce immediate or very rapid incapacitation following a head shot, although during

the further course elevated intracranial pressure can of course become symptomatic. So immediate incapacitation can only be the result of disruption of brain tissue by the bullet.

9.3.4 *Rapid Incapacitation*

Acute cerebral hypoxemia can be caused by massive blood loss (or a double-sided pneumothorax). Injuries associated with acute and massive bleeding will cause circulatory depression and reduced perfusion of the CNS with subsequent unconsciousness. However, immediate circulatory arrest is very rare in cardiac or vascular gunshot wounds and even if this occurs, the oxygen stored in the CNS ensures a potential for physical activity for about 10s [64, 65]. This is illustrated by numerous case-reports. Marsh et al. [87] described two six-shot suicides, one of which involved three .22 bullets striking the heart. A young man had been conscious for several minutes after receiving two perforating and one grazing gunshot wound of the heart from .22 bullets [88]. The highest number of suicidal gunshots recorded is nine including a complete disruption of the apex of the heart from seven .25 FMJ bullets [89]. Other cases of physical activity following penetrating gunshot wounds to the heart have been published by Spitz et al. [60] and Levy and Rao [90]. In a “worst-case” scenario described by DiMaio [91], a man was able to walk 20 m after sustaining a hit from a 12-gauge shotgun from a range of 3–4 m which destroyed his entire heart. Missliwetz [92] reported a similar close range shotgun case with laceration of the posterior wall of the heart and complete transection of the thoracic aorta where the young man still walked a distance of 6 m. These very rare examples of immediate circulatory arrest from gunshot wounds demonstrate without any doubt that a potential for physical activity is present in such cases.

Therefore, trajectories involving the heart, the aorta (especially the thoracic part) or the truncus of the pulmonary artery can cause rapid incapacitation (Table 9.4) but they cannot be relied upon to terminate the physical activity of the victim immediately [64, 65].

9.3.5 *Delayed Incapacitation*

The latent period until incapacitation in cases of considerable ballistic injury to the lungs, liver, kidneys, spleen, and large vessels originating from the aorta or central veins will be substantially longer [64, 65]. The slower rate of

Table 9.4 Targets of rapid incapacitation

Heart, aorta, truncus pulmonalis

Table 9.5 Targets of delayed incapacitation
Large arteries and veins, lungs, liver, spleen, kidneys

bleeding and the circulatory compensation mechanisms [64] usually offer the potential for sustained physical activity in the range of one or several minutes depending on the injury present (Table 9.5) and a considerable number of such case reports have been published (e.g., [60, 63, 90, 91]). So just about every person sustaining one or more gunshot wounds to the thorax including the heart possesses the potential for physical activity for at least a short period of time. But this potential for physical activity is not always exhausted: 80% of 62 gunshot fatalities collapsed following the assault [60], although it is not clearly stated how promptly they collapsed. Obviously, the mental or emotional condition of the victim, especially the expectancy of or being prepared for a hit, play an important role. Some may be stunned by surprise, fright or pain and some may instinctively choose not to act. But this psychological aspect can be neither predicted nor reconstructed. The only thing to rely on is that it will have no effect in the cases of trained, motivated, excited or stimulated individuals. Determination, adrenaline, or chemicals even enable persons to discharge aimed gunshots after sustaining a penetrating heart injury: A victim of a .32 caliber gunshot wound penetrating his heart, lung, and liver managed to fire back and wounded the assailant in the chest [60].

9.4 Backspatter

In most perforating gunshot wounds, blood and tissue is ejected from the exit wound. In many gunshot wounds, biological material is also propelled retrogradely out of the entrance wound towards the firearm. This phenomenon has been recognized for a long time as “Rückschleuderspuren” [94, 95, 96] and was later named backspatter [97, 98].

The stains resulting from backspatter can be very important in crime scene reconstruction because of the direction against the line of fire. There can be a transfer of stains from the victim to the interior of the barrel, the outside of the weapon, the person shooting, and persons or objects in the vicinity. Unlike gunshot residues, these stains can be individualized so that the transfer of stains can be demonstrated to be specific for the production of a clearly defined gunshot injury. Determination of the weapon used and the person firing, the shooting distance or the posture of the victim can be accomplished by analysis of backspatter stains, which should therefore form an integral part of all reconstructions of shooting incidents.

9.4.1 Mechanisms of Production

In terminal ballistics, backward hurling of gelatine [11, 99] and isolated muscle [100] and backward fragmentation of glass [101, 102] and bone [30] are well documented. The occurrence and the quantity of backspatter depend on a variety of ballistic and anatomical parameters. The kinetic energy necessary for the acceleration of blood and tissue particles can come from three different sources:

1. *Subcutaneous gas effect*: The rapid expansion of hot muzzle gases trapped below the elastic skin and especially between the skin and a bony abutment causes the temporary development of a pocket-like subcutaneous space. When this space collapses immediately, the biological particles are expelled together with the resulting backwards stream of escaping gases [94, 96, 98, 103, 104, 105].
2. *Temporary cavity*: Temporary cavitation produces overpressures inside the wound which also escape retrogradely to the line of fire [102, 103, 105]. Anatomical structures similar to liquid-filled cavities such as the head, the heart, or the eye provide the best conditions for violent temporary cavitation and backward hurling of particles.
3. *Tail splashing*: If a projectile penetrates tissue, there is always backward streaming of fluid and tissue particles along the lateral surface of the bullet in the direction of the entrance wound [11, 99, 103].

In most cases, these three driving forces act combined. The hot muzzle gases can only be effective in contact and close-to-contact gunshots, especially in the presence of a bony abutment. The presence of backspattered brain particles [106, 107] can only be explained by intracranial temporary cavitation, which is clearly enhanced by the confined space of the neurocranium (see Sect. 2.4). Therefore, backspatter mainly occurs in close-range gunshots to the head but is not limited to such cases, as is demonstrated by a gunshot to the heart (liquid-filled cavity) from a distance of 4 m resulting in massive backspatter travelling up to 2.5 m [105]. The spin of the bullet [108] or a momentary suction effect of the barrel aspirating material into the muzzle [106, 109] clearly do not contribute to backspatter [102, 103].

9.4.2 Experiments

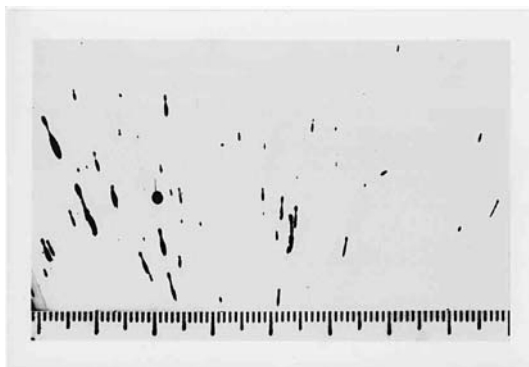
While case reports (e.g., [94, 105, 110]) only shed some light on the topic, experimental investigations are difficult for obvious reasons. Mock experiments using primitive “head-models” constructed from blood-soaked sponges wrapped in plastic, rubber or tape [98, 104] resulted in small droplets from contact or almost contact shots only travelling a maximum distance of 30–60 cm. However, these findings are not reliable because there is neither a confined subcutaneous

space for the hot gases to expand, nor is there a rigid skull to give rise to the high intracranial pressures during the formation of the temporary cavity. This applies even more to gunshots into soft pine covered with polyurethane foam saturated with blood [97].

Only controlled animal experiments interpreted by comparison with man can produce reliable results. Wagner [108] fired gunshots to rabbits using a 7.65-mm pistol but lacerated the small animals rather than creating backspatter. MacDonnell [97] performed shooting experiments on dogs but did not give detailed results or the experimental set-up. Burnett [111] has used pigs for the investigation of microscopic bone and bone-plus-bullet particles in backspatter and found tiny bone chips by SEM/EDX-analysis (scanning electron microscopy and energy dispersive X-ray analysis) up to a distance of 37 cm from the entrance wound.

In experimental gunshots to the heads of calves [103, 112, 113] using 9-mm Parabellum ammunition from shooting distances of 0–10 cm, backspatter was documented after every gunshot. The number of macrostains (stains with a diameter >0.5 mm) varied from 31 to 324 per gunshot and appeared to be independent of the short shooting distances. The maximum distance macrodroplets travelled varied from 72 to 119 cm but the majority of droplets accumulated between 0 and 50 cm. The number of microbackspatter stains (diameter <0.5 mm) per gunshot varied between 39 and 262 and the maximum travelling distance was 69 cm while the vast majority of microdroplets accumulated between 0 and 40 cm. Microstains exclusively were circular to slightly oval due to the high surface tension of the tiny droplets. The morphology of macrobackspatter stains varied from round to elongated with circular, drop-like stains in the form of exclamation marks predominating (Fig. 9.13). Small macrostains (0.5–4 mm) made up more than 90% of the macrostains and no systematic relationship between distance travelled and size of the stains could be established. The direction a single droplet can take is every possible angle between the most tangential ones to the skin surface. This resulted in a semicircle of 180° (or a semisphere) covered with stains

Fig. 9.13 Morphology of macrobackspatter stains on a horizontal surface in front of and 60 cm below the entrance wound after a near contact gunshot to the temple using a 9-mm Parabellum FMJ bullet. The entrance wound was roughly located where the long axis of the elongated stains converge



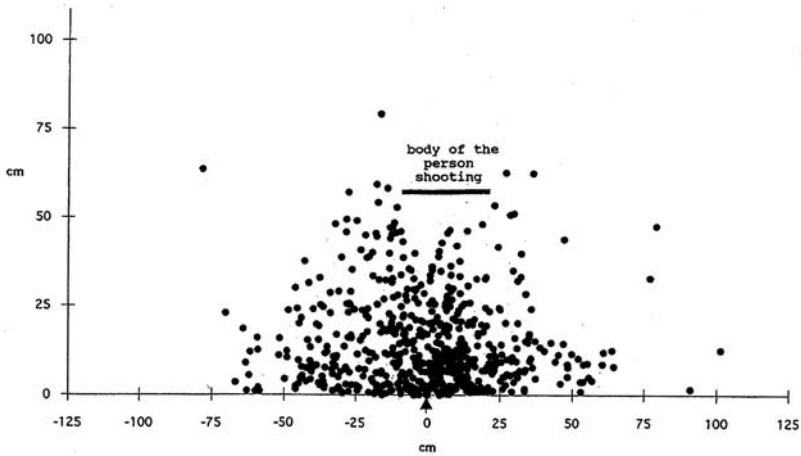


Fig. 9.14 Superimposed stain distribution of six close-range gunshots to the temple in front of and 60 cm below the entrance wound. The *small arrow* (0 cm) marks the location of the entrance wound

and not a triangle as assumed by MacDonnell [97]. The superimposed distribution of the stains from six gunshots is depicted in Fig. 9.14. The occurrence of skin ruptures of the entrance wound is not necessary for backspatter. Backspatter of tiny fragments of bone, fat, muscle and skin was recovered in most cases.

The succession of events was documented on high speed film and started with the recoil of the firearm, immediately followed by a blow-out effect of the skin. Large droplets exited approximately 0.7–4 ms after the bullet impacted the skin. The calculated minimum initial velocity of these droplets was in the range of 10 m/s.

In the same investigation [113], the firearms, the surgical gloves and the right sleeve worn by the person shooting were examined with a stereomicroscope. Backspatter of blood was found on the firearms and sleeves in half and on the gloves in two thirds of the cases. Most droplets were 1–3 mm in size and circular or elongated in shape but there was also a fine spray of tiny blood deposits. The distribution of the droplets on the firearms varied including regions shielded by prominent parts while the droplets on the person shooting were predominantly located on the extensor side of the fingers and the radial aspect of the hands and sleeves (Fig. 9.15).

9.4.3 Empirical Observations

In addition to blood, backspatter of brain tissue, fat, muscle, bone fragments, skin, hair, and even ocular tissue has been recovered from the shooting hand and/or the firearm in case-work following gunshots to the head [94, 96, 105, 106, 107]. Maximum travelling distances of 2 and 4 m have been documented

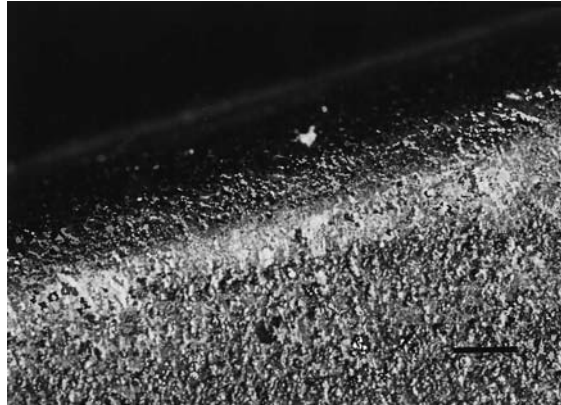
Fig. 9.15 Bloodstains on the right glove predominantly located at the extensor side of the fingers after firing a contact gunshot to the head



[103, 110]. After suicidal gunshots to the head, bloodspatter on the hands was found with the naked eye at the time of autopsy in 14–35% [107, 114]. In a large series of 1200 suicides, the exterior surface of the barrel reacted positive for blood in 75% and the interior surface in 55% [115]. However, the morphology of the blood deposits was not evaluated and blood can also be transferred by contact or other mechanisms.

So in close-range gunshots to the head, backspatter is produced in all cases and is deposited frequently on the firearm and person shooting. The presence of backspatter stains establishes a clear link between a person or object and a clearly defined gunshot, especially if the stains are individualized by DNA-analysis. Characteristic for backspatter are small or tiny droplet or splashing stains; elongated shapes are roughly aimed at the entrance wound while the distribution can vary greatly. The number and size of the stains can be very small. Thus, the detection or exclusion of backspatter stains in practical case-work necessitates magnification and appropriate lighting or chemical analysis, especially if the tiny stains do not contrast with the background, e.g., a dark-coloured firearm (Fig. 9.16) or clothing. However, no rash conclusions should be drawn from the absence of backspatter stains on the firearm and hands because the number of droplets in a single gunshot can be so small that these structures may not be hit by a single droplet. In these instances, special attention should be directed to the shoes and pants of a suspect because these objects are likely to be in the downwards parabolic flight path of the droplets. In our case-work experience, the number of positive findings also depends on the delay until the examination is carried out

Fig. 9.16 A tiny bloodstain (*below*) and a tiny bone chip (*above*) on the barrel of a firearm after a near-contact gunshot to the head. The *black bar to the right* marks a distance of 1 mm



because the small droplets can detach from the metal, skin or textiles during manipulations or transportation. Early inspection is therefore recommended.

9.5 Trajectory Reconstruction from Trace Evidence on Spent Bullets

Contact between two objects commonly results in bidirectional transfer of material. This principle forms the basis of modern trace evidence analysis. Trace evidence may be the decisive factor in reconstruction of the scene of crime and in identifying the persons and objects involved. Due to the substantial impact forces involved, bullets striking an object appear to represent favourable conditions for such a transfer of material but air friction and subsequent impacts may also cause loss of deposits. In principle, biologic and non-biologic deposits can be present on a spent bullet and both forms of trace evidence can be relevant depending on the case constellation.

9.5.1 Intermediate Targets

Contact of a fired bullet with an intermediate target of sufficient resistance is a very important factor: the bullet will ricochet, fragment or perforate, the trajectory will show a deviation and the stability of the bullet will be affected. The latter will cause the bullet to tumble and a tumbling bullet will produce an atypical gunshot entrance wound. The verification of a ricochet by analysis of trace evidence can have considerable legal implications because the intention to kill will commonly not be assumed. In addition, valuable information concerning the trajectory of the bullet can be gained.

In a few case reports dedicated to this field, the bullet deposits were only investigated on deformed lead bullets [116, 117], which represent very favourable conditions for the deposition of trace evidence.

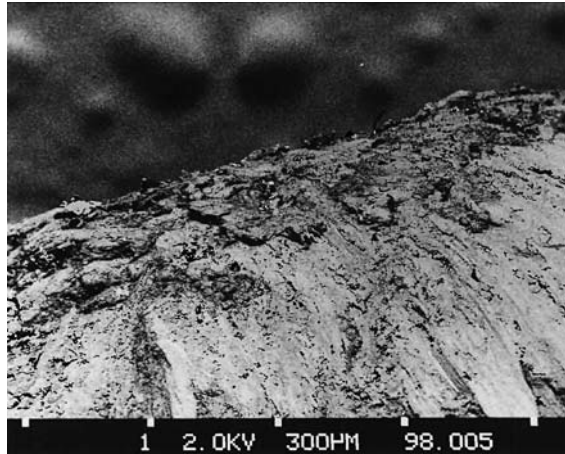
In an experimental investigation [118], 9-mm Parabellum FMJ bullets were fired at various intermediate targets and at combinations of intermediate targets and tissue located in line. After recovery from a bullet collector, the bullets were examined using a scanning electron microscope and an energy-dispersive X-ray spectrometer (SEM/EDS). Elements of preexisting environmental deposits, mostly in the form of small spheres (Fig. 9.17), were already present on unfired bullets and elements from the bullet collector, the jacket, the charge and primer could be consistently detected as a “background”.

In impact dynamics, ductile and fragile target media are distinguished [119]. Ductile material is elastic and can be stretched before perforation whereas fragile material is brittle and will break before a noticeable stretching occurs. Abundant deposits of the fragile materials concrete (Fig. 9.18), flat glass, asphalt and gypsum-board (Fig. 9.19) could be visualised on every bullet by SEM. The transfer dynamics involved a direct imprint of target material on the bullet surface and thus preferential locations at the tip but also indirect deposition over the entire surface (“powder effect”) when the bullet travels through a cloud of shattered target particles. X-ray microanalysis demonstrated matching spectra of the elemental composition of these fragile deposits and of the targets contacted (Fig. 9.20A,B). After perforation of the ductile materials wood and car body parts, the scarce deposits on the bullets did not show characteristic spectra. This was due to the complex perforation dynamics of ductile materials involving elastic radial displacement or punching out of a plug. If multi-layered car metal targets were hit, few and variable fragments were scattered on the bullet surface and titanium indicative of paintwork could be determined on only a minority of bullets. The elemental composition of wood itself was heterogeneous but the fibrous morphology of the deposits was typical.

Fig. 9.17 Scanning electron microscopy image: preexisting deposits on the surface of a brand-new and unfired 9-mm FMJ bullet. Most contaminations presented in the form of small spherical Al-Si-combinations



Fig. 9.18 Scanning electron microscopy image: concrete deposits on the tip of a 9-mm FMJ bullet after perforation of concrete



The SEM/EDS findings in gunshots including subsequent perforation of tissue were similar. In particular, the trace evidence primarily transferred to the bullets was not eliminated by secondary contact and the determination of the fragile target materials was not affected.

So when a person is killed or injured by a gunshot, the presence of a ricochet and the target material can be determined. It appears clear from these results and also from own case-work experience that a complete loss of fragile trace evidence in subsequent impacts or penetration of a human body does not occur. The determination of fragile target materials is therefore possible by the combined evidence of morphological and analytical findings. This possibility needs to be considered before an evidential bullet is mishandled: Blood, tissue, and nonbiological material may be washed from the bullet and loss or contamination may occur during transportation or storage. A directed search of the scene should then be conducted. The intermediate target will show a bullet impact mark/perforation defect, which additionally represents an ideal reference point

Fig. 9.19 Scanning electron microscopy image: gypsum-board deposits on a 9-mm FMJ bullet after perforation of gypsum-board



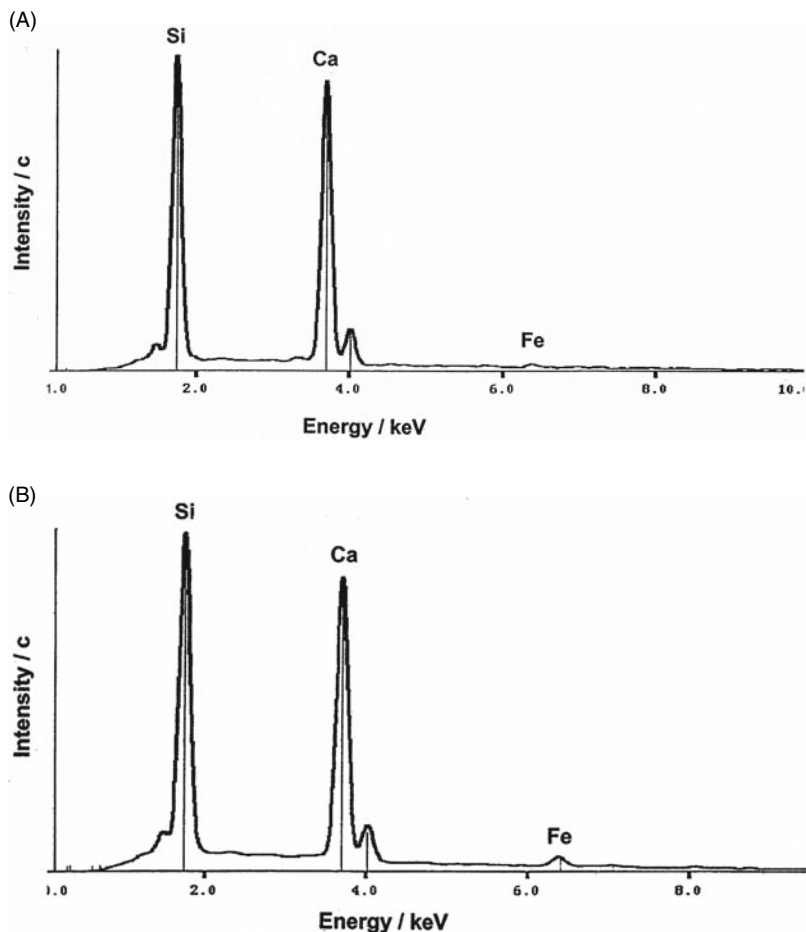


Fig. 9.20 Energy-dispersive X-ray spectrometer (EDS)-spectra: Comparison of the EDS-spectrum of: **A** the concrete perforated; **B** the bullet deposits shown in Fig. 9.18

for trajectory reconstruction. Investigation of the scene is also necessary because contact with an intermediate target may occur before or after a human body is perforated but the sequence of events can usually not be derived from trace evidence on FMJ bullets.

9.5.2 Biological Deposits

In cases of gunfights involving several persons, it can be crucial to know who killed or injured whom. A perforating bullet found at the scene can commonly be linked to one of the firearms involved via comparison of rifling marks but in

addition, the bullet must be linked to the person through whom it has passed. A technique capable of demonstrating and simultaneously individualizing biological material is therefore necessary to reliably determine who killed or injured whom using which bullet.

Human trace evidence on bullets has been investigated by routine cytological methods but individualisation is commonly not possible, and cells or even cell layers can be found in the cavities of hollow point bullets but are rarely found on the smooth surfaces of FMJ bullets [120, 121, 122].

Individualisation of tissue deposits on FMJ bullets was successfully carried out by polymerase chain reaction (PCR)-typing of short tandem repeats (STRs) in both experiments [123] and case-work [124]. It could also be shown that the amplification of mitochondrial DNA on spent bullets is a useful additional method and that subsequent impacts on intermediate targets do not eliminate enough biological deposits to render DNA analysis impossible [125]. Neither visualisation by SEM nor separate swabbing was successful in establishing preferential locations of cells on bullets [125].

So DNA analysis of tissue deposits on spent bullets represents a sensitive method capable of demonstrating and at the same time individualising minute tissue fragments transferred during perforation of a human body. This makes it possible to determine who was killed or injured by which bullet. Additional comparison of the bullet with the firearms involved can possibly identify the person shooting this particular bullet. The decision whether DNA typing is to be performed should be made as soon as possible because the bullets must be protected from contamination and loss of material. The recovery of cellular material by swabbing the bullet can be easily and rapidly performed at the scene so that further examinations will not be delayed.

References

1. Fackler ML (1988) Wound ballistics. A review of common misconceptions. *JAMA* 259:2730–2736
2. Fackler ML, Malinowski JA (1985) The wound profile: a visual method for quantifying gunshot wound components. *J Trauma* 25:522–529
3. Harvey EN, Butler EG, McMillan JH, Puckett WO (1945) Mechanism of wounding. *War Med* 8:91–104
4. Harvey EN, Korr IM, Oster G, McMillan JH (1947) Secondary damage in wounding due to pressure changes accompanying the passage of high velocity missiles. *Surgery* 21:218–239
5. Bowen TE, Bellamy RF (1988) Emergency war surgery, Second United States Revision of the Emergency War Surgery Handbook. United States Department Of Defence, Washington, DC, pp 13–33
6. Callender GR, French RW (1935) Wound ballistics. Studies in the mechanism of wound production by rifle bullets. *Mil Surg* 77:177–201
7. Scott R (1983) Pathology of injuries caused by high-velocity missiles. *Clin Lab Med* 3:273–294

8. Krauss M (1957) Studies in wound ballistics: temporary cavity effects in soft tissues. *Mil Med* 121:221–231
9. Sellier K, Kneubuehl BP (1994) Wound ballistics and the scientific background. Elsevier, Amsterdam London New York Tokyo, pp 144–149, 245
10. Zhang J, Yoganandan N, Pintar FA, Gennarelli TA (2005) Temporary cavity and pressure distribution in a brain simulant following ballistic penetration. *J Neurotrauma* 22:1335–1347
11. Black AN, Burns BD, Zuckerman S (1941) An experimental study of the wounding mechanism of high-velocity missiles. *Br Med J* 2:872–874
12. Karger B (1995) Penetrating gunshots to the head and lack of immediate incapacitation. I. Wound ballistics and mechanisms of incapacitation. *Int J Legal Med* 108:53–61
13. Lindsey D (1980) The idolatry of velocity, or lies, damn lies, and ballistics. *J Trauma* 20:1068–1069
14. Beyer JC (1962) Wound ballistics. Office of the Surgeon General, Department of the Army, Washington, DC, p 135
15. Boyer CN, Holland GE, Seely JF (2005) Flash X-ray observations of cavitation in cadaver thighs caused by high-velocity bullets. *J Trauma* 59:1463–1468
16. Fackler ML, Surinchak JS, Malinowski JA, Bowen RE (1984) Bullet fragmentation: a major cause of tissue disruption. *J Trauma* 24:35–39
17. Dzieman AJ, Mendelson JA, Lindsey D (1961) Comparison of the wounding characteristics of some commonly encountered bullets. *J Trauma* 1:341–353
18. Hopkinson DAW, Watts JC (1963) Studies in experimental missile injuries of skeletal muscle. *Proc R Soc Med* 56:461–468
19. Kocher T (1895) Zur Lehre von den Schußwunden durch die Kleinkalibergeschosse. G. Fischer & Co, Cassel
20. Fackler ML, Peters CE (1991) The “shock wave” myth (and comment). *Wound Ballistics Rev* 1:38–40
21. Fackler ML, Bellamy RF, Malinowski JA (1988) The wound profile: illustration of the missile-tissue interaction. *J Trauma* 28(Suppl):S21–S29
22. Fackler ML, Surinchak JS, Malinowski JA, Bowen RE (1984) Wounding potential of the russian AK-74 assault rifle. *J Trauma* 24:263–266
23. Cooper GJ, Ryan JM (1990) Interaction of penetrating missiles with tissues: some common misapprehensions and implications for wound management. *Br J Surg* 77:606–610
24. Metter D, Schulz E (1983) Morphologische Merkmale der Schußwunden in Leber und Milz. *Z Rechtsmed* 90:167–172
25. Fackler ML (1989) Wounding patterns of military rifle bullets. *Int Defense Review* 1:59–64
26. Ragsdale BD, Josselson A (1988) Experimental gunshot fractures. *J Trauma* 28(Suppl):S109–S115
27. Sellier K (1971) Über Geschossablenkung und Geschossdeformation. *Z Rechtsmed* 69:217–251
28. Karger B, Puskas Z, Ruwald B, Teige K, Schuirer G (1998) Morphological findings in the brain after experimental gunshots using radiology, pathology and histology. *Int J Legal Med* 111:314–319
29. Robens W, Küsswetter W (1982) Fracture typing to human bone by assault missile trauma. *Acta Chir Scand, Suppl* 508:223–227
30. Lorenz R (1948) Der Schußkanal im Röntgenbilde. *Dtsch Z Gerichtl Med* 39:435–448
31. Sturdivan L (1969) Terminal behavior of the 5.56mm ball in soft targets. Rep 1447. Ballistic Research Laboratory, Aberdeen Proving Grounds
32. Ragsdale BD (1984) Gunshot wounds: historical perspective. *Mil Med* 149:301–315

33. Karger B (2003) Schussverletzungen. In: Brinkmann B, Madea B (eds) *Handbuch der Rechtsmedizin* I. Springer, Berlin Heidelberg New York, pp 593–682
34. Albrecht M, Scepanovic D, Ceramilac A, Milivojevic V, Berger S, Tasic G, Tatic V, Todoric M, Popovic D, Nanusevic N (1979) Experimental soft tissue wounds caused by standard military rifles. *Acta Chir Scand Suppl* 489:185–198
35. Berlin R, Gelin LE, Janzon B, Lewis DH, Rybeck B, Sandegard J, Seeman T (1976) Local effects of assault rifle bullets in live tissues. *Acta Chir Scand Suppl* 459:1–84
36. Berlin R, Janzon B, Rybeck B, Sandegard J, Seeman T (1977) Local effects of assault rifle bullets in live tissues: Part II: Further studies in live tissues and relations to some simulant media. *Acta Chir Scand Suppl* 477:5–57
37. Berlin R, Janzon B, Nordström G, Schantz B (1978) The extent of tissue damage in missile wounds one and six hours after the infliction of trauma studied by the current method of debridement. *Acta Chir Scand* 144:213–217
38. Jussila J, Kjellstrom BT, Leppaniemi A (2005) Ballistic variables and tissue devitalisation in penetrating injury – establishing relationship through meta-analysis of a number of pig tests. *Injury* 36:282–292
39. Scepanovic D, Albrecht M, Erdeljan D (1982) A method for predicting effects of military rifles. *Acta Chir Scand Suppl* 508:29–37
40. Tikka S, Seeman T (1988) Local tissue destruction in high-energy missile trauma and its dependence on energy transfer. *Ann Med Milit Fenn* 62:17–20
41. Allen IV, Scott R, Tanner JA (1982) Experimental high-velocity missile head injury. *Injury* 14:183–193
42. Clemedson CJ, Falconer B, Frankenberg L, Jönsson A, Wennerstrand J (1973) Head injuries caused by small-calibre, high-velocity bullets. *Z Rechtsmed* 73:103–114
43. Finnie JW (1993) Pathology of experimental traumatic craniocerebral missile injury. *J Comp Pathol* 108:93–101
44. Kirkpatrick JB, DiMaio VJM (1978) Civilian gunshot wounds of the brain. *J Neurosurg* 48:185–198
45. Dittmann W (1986) Wundballistische Untersuchungen zur Klinik der Schädel-Hirn-Schußverletzungen. *Wehrmed Monatsschr* 33:3–14
46. Watkins FP, Pearce BP, Stainer MC (1988) Physical effects of the penetration of head simulants by steel spheres. *J Trauma* 28(1)(Suppl):S40–S54
47. Freytag E (1963) Autopsy findings in head injuries from firearms. Statistical evaluation of 254 cases. *Arch Pathol* 76:215–225
48. Henn R, Liebhardt E (1969) Zur Topik außerhalb des Schußkanals gelegener Hirnrindenblutungen. *Arch Kriminol* 143:188–191
49. Spatz H (1941) Gehirnpathologie im Kriege. Von den Gehirnwunden. *Zentralbl Neurochir* 6:162–212
50. Klaue R (1949) Die indirekten Frakturen der vorderen Schädelgrube beim Schädeldachschuß. *Dtsch Z Nervenheilkd* 161:167–193
51. Kolsky H (1980) The role of stress waves in penetration processes. In: Labile RC (ed) *Ballistic materials and penetration mechanics*. Elsevier, New York, pp 185–223
52. König HG, Schmidt V (1989) Beobachtungen zur Ausbreitungsgeschwindigkeit und Entstehungsursache von Berstungsfrakturen beim Schuß. *Beitr Gerichtl Med* 47:247–255
53. Smith OC, Berryman HE, Lahren CH (1987) Cranial fracture patterns and estimate of direction from low velocity gunshot wounds. *J Forensic Sci* 32:1416–1421
54. Butler FG, Puckett WO, Harvey EN, McMillan JH (1945) Experiments on head wounding by high velocity missiles. *J Neurosurg* 2:358–363
55. DiMaio VJM, Zumwalt RE (1977) Rifle wounds from high velocity, center-fire hunting ammunition. *J Forensic Sci* 22:132–140
56. Knudsen PJT, Theilade P (1993) Terminal ballistics of the 7.62mm NATO bullet. Autopsy findings. *Int J Legal Med* 106:61–67

57. Peng L, Cheng Z, Guangji Z, Yinqiou L, Reifeng G (1990) An experimental study of craniocerebral injury caused by 7.62 mm bullets in dogs. *J Trauma (China)* 6(Suppl): 187–191
58. Karger B, Banaschak S (1997) Two cases of exenteration of the brain from Brenneke shotgun slugs. *Int J Legal Med* 110:323–325
59. Sight WP (1969) Ballistic analysis of shotgun injuries to the central nervous system. *J Neurosurg* 31:25–33
60. Spitz WU, Petty CS, Fisher RS (1961) Physical activity until collapse following fatal injury by firearms and sharp pointed weapons. *J Forensic Sci* 6:290–300
61. Walcher K (1929) Über Bewußtlosigkeit und Handlungsunfähigkeit. *Dtsch Z Gerichtl Med* 13:313–322
62. Petersohn F (1967) Über die Aktions- und Handlungsfähigkeit bei schweren Schädeltraumen. *Dtsch Z Gerichtl Med* 59:259–270
63. Karger B, Brinkmann B (1997) Multiple gunshot suicides: potential for physical activity and medico-legal aspects. *Int J Legal Med* 110:188–192
64. Newgard K (1992) The physiological effects of handgun bullets. *Wound Ballistics Rev* 1:12–17
65. Fackler ML (1992) Police handgun ammunition selection. *Wound Ballistics* 1:32–37
66. DiMaio VJM, Jones JA, Caruth WW III, Anderson LL, Petty CS (1974) A comparison of the wounding effects of commercially available handgun ammunition suitable for police use. *FBI Law Enf Bull* 43:3–8
67. Karger B, Kneubuehl BP (1997) On the physics of momentum in ballistics: can the human body be displaced or knocked down by small arms projectiles? *Int J Legal Med* 109:147–149
68. Suneson A, Hansson H-A, Seeman T (1988) Central and peripheral nervous damage following high-energy missile wounds in the thigh. *J Trauma* 28(Suppl):S197–S203
69. Jason A (1991) The “twilight zone” of wound ballistics. *Wound Ballistics Rev* 1(1):8–9
70. Kaufman HH, Loyola WP, Makela ME, Frankowsky RF, Wagner KA, Bustein DP, Gildenberg PC (1986) Gunshot wounds to the head: a perspective. *Neurosurg* 18:689–695
71. Siccardi D, Cavaliere R, Pau A, Lubinu F, Turtas S, Viale GL (1991) Penetrating craniocerebral missile injuries in civilians: a retrospective analysis of 314 cases. *Surg Neurol* 35:455–460
72. Karger B (1995) Penetrating gunshots to the head and lack of immediate incapacitation. II. Review of case reports. *Int J Legal Med* 108:117–126
73. Bratzke H, Pöll W, Kaden B (1985) Ungewöhnliche Handlungsfähigkeit nach Kopfsteckschuß. *Arch Kriminol* 175:31–39
74. Smith S (1943) Voluntary acts after a gunshot wound of the brain. *Police J* 16:108–110
75. Fryc O, Krompecher T (1979) Überlebenszeit und Handlungsfähigkeit bei tödlichen Verletzungen. *Beitr Gerichtl Med* 37:389–392
76. Krauland W (1952) Zur Handlungsfähigkeit Kopfschußverletzter. *Acta Neurochir* 2:233–239
77. Klages U, Weithoener D, Frössler H, Terwort H (1975) Überlebenszeit, Handlungsfähigkeit und röntgenologische Diagnostik bei Schußverletzungen des Schädels. *Z Rechtsmed* 76:307–319
78. Maxeiner H, Schneider V, Betsch J, Piefke K (1986) Suizid mit drei Kopfsteckschüssen. In: Eisenmenger W, Liebhardt E, Schuck M (eds) *Medizin und Recht*. Springer, Berlin Heidelberg New York, pp 317–325
79. Herlich J (1955) Neun Jahre überlebte Gehirn- und Herzschußverletzung mit Einheilung beider Geschosse, nach Selbstmordversuch. *Beitr Gerichtl Med* 20:22–34
80. Goroncy C (1924) Handlungsfähigkeit Kopfschußverletzter. *Dtsch Z Gerichtl Med* 4:145–164
81. Naegeli O (1884) Zwei perforierende Hirnschüsse. Mord oder Selbstmord? *Vjschr Gerichtl Medicin* 40:231–264

82. Payr E (1922) Der frische Schädelchuß. In: Schjerning O von (ed) Handbuch der ärztlichen Erfahrungen im Weltkrieg 1914/1918, Bd 1. Barth, Leipzig, pp 285–410
83. Carey ME, Sarna GS, Farrel JB, Happel LT (1989) Experimental missile wound to the brain. *J Neurosurg* 71:754–764
84. Crockard HA, Brown FD, Johns LM, Mullan S (1977) An experimental cerebral missile injury model in primates. *J Neurosurg* 46:776–783
85. Crockard HA, Brown FD, Calica AB, Johns LM, Mullan S (1977) Physiological consequences of experimental cerebral missile injury and use of data analysis to predict survival. *J Neurosurg* 46:784–794
86. Gerber AM, Moody RA (1972) Craniocerebral missile injuries in the monkey: an experimental physiological model. *J Neurosurg* 36:43–49
87. Marsh TO, Brown ER, Burkhardt RP, Davis JH (1989) Two six-shot suicides in close geographic and temporal proximity. *J Forensic Sci* 34:491–494
88. Hudson P (1981) Multishot firearm suicide. Examination of 58 cases. *Am J Forensic Med Pathol* 2:239–242
89. Habbe D, Thomas GE, Gould J (1989) Nine-gunshot suicide. *Am J Forensic Med Pathol* 10:335–337
90. Levy V, Rao VJ (1988) Survival time in gunshot and stab wound victims. *Am J Forensic Med Pathol* 9:215–217
91. DiMaio VJM (1985) Gunshot wounds. Practical aspects of firearms, ballistics and forensic techniques. Elsevier, New York Amsterdam Oxford
92. Missliwetz J (1990) Ungewöhnliche Handlungsfähigkeit bei Herzdurchschuß durch Schrotgarbe. *Arch Kriminol* 185:129–135
93. Introna F, Smialek JE (1989) Suicide from multiple gunshot wounds. *Am J Forensic Med Pathol* 10:275–284
94. Fraenckel P, Straßmann G (1924) Zur Entfernungsbestimmung bei Nahschüssen. *Arch Kriminol* 76:314–316
95. Hofmann v ER (1898) Lehrbuch der Gerichtlichen Medizin, 8. ed. Wien, Leipzig, p 389
96. Werkgartner A (1924) Eigenartige Hautverletzungen durch Schüsse aus angesetzten Selbstladepistolen. *Beitr Gerichtl Med* 6:148–161
97. MacDonell HL (1982) Bloodstain pattern interpretation. Laboratory of Forensic Science Publishers, New York, pp 16–21
98. Stephens BG, Allen TB (1983) Back spatter of blood from gunshot wounds – observations and experimental simulation. *J Forensic Sci* 28:437–439
99. Amato JJ, Billy LJ, Lawson NS, Rich NM (1974) High velocity missile injury. An experimental study of the retentive forces of tissue. *Am J Surg* 127:454–458
100. Lamprecht K (1959) Schuß durch Fensterglas. *Arch Kriminol* 97:128–132
101. Sellier K (1982) Schusswaffen und Schusswirkungen I. Schmidt-Römhild, Lübeck
102. Karger B, Nüsse R, Schroeder G, Wüstenbecker S, Brinkmann B (1996) Backspatter from experimental close-range shots to the head. I. Macrobackspatter. *Int J Legal Med* 109:66–74
103. Pex JO, Vaughan CH (1987) Observations of high velocity bloodspatter on adjacent objects. *J Forensic Sci* 32:1587–1594
104. Weimann W (1931) Über das Verspritzen von Gewebeteilen aus Einschußöffnungen und seine kriminalistische Bedeutung. *Dtsch Z Gerichtl Med* 17:92–105
105. Brüning A, Wiethold F (1934) Die Untersuchung von Selbstmörderschusswaffen. *Deutsche Zeitschrift Gerichtliche Med* 23:71–82
106. Zwingli M (1941) Über Spuren an der Schießhand nach Schuß mit Faustfeuerwaffen. *Arch Kriminol* 108:1–26
107. Wagner HJ (1963) Experimentelle Untersuchungen über Art und Ausmaß der Rückschleuderung von Blut und Gewebeteilen beim absoluten und relativen Nahschuß. *Dtsch Z Gerichtl Med* 54:258–266

109. Knight B (1977) Firearm injuries. In: Tedeschi CG, Eckert WG, Tedeschi LG (eds) Forensic medicine. Saunders, Philadelphia London Toronto, pp 510–526
110. Verhoff MA, Karger B (2003) Atypical gunshot entrance wound and extensive backspatter. *Int J Legal Med* 117:229–231
111. Burnett BR (1991) Detection of bone and bone-plus-bullet particles in backspatter from close-range shots to heads. *J Forensic Sci* 36:1745–1752
112. Karger B, Nüsse R, Tröger HD, Brinkmann B (1997) Backspatter from experimental close-range shots to the head. II. Microbackspatter and the morphology of bloodstains. *Int J Legal Med* 110:27–30
113. Karger B, Nüsse R, Bajanowski T (2002) Backspatter on the firearm and hand in experimental close-range gunshots to the head. *Am J Forensic Med Pathol* 23:211–213
114. Betz P, Peschel O, Stiefel D, Eisenmenger W (1995) Frequency of blood spatters on the shooting hand and of conjunctival petechiae following suicidal gunshot wounds to the head. *Forensic Sci Int* 76:47–53
115. Stone IC (1992) Characteristics of firearms and gunshot wounds as markers of suicide. *Am J Forensic Med Pathol* 13:275–280
116. DiMaio VJM, Dana SE, Taylor WE, Ondrusek J (1987) Use of scanning electron microscopy and energy dispersive X-ray analysis (SEM/EDX) in identification of foreign material on bullets. *J Forensic Sci* 32:38–47
117. Petraco N, DeForest PR (1990) Trajectory reconstructions I: trace evidence in flight. *J Forensic Sci* 35:1284–1296
118. Karger B, Hoekstra A, Schmidt PF (2001) Trajectory reconstruction from trace evidence on spent bullets. I. Deposits from intermediate targets. *Int J Legal Med* 115:16–22
119. Goldsmith W (1999) Non-ideal projectile impact on targets. *Int J Impact Eng* 22:95–395
120. Knudsen PJT (1993) Cytology in ballistics. An experimental investigation of tissue fragments on full metal jacketed bullets using routine cytological techniques. *Int J Legal Med* 106:15–18
121. Nichols CA, Sens MA (1990) Recovery and evaluation by cytologic techniques of trace material retained on bullets. *Am J Forensic Med Pathol* 11:17–34
122. Nichols CA, Sens MA (1991) Cytologic manifestations of ballistic injury. *Am J Clin Pathol* 95:660–669
123. Karger B, Meyer E, Knudsen PJT, Brinkmann B (1996) DNA typing of cellular material on perforating bullets. *Int J Legal Med* 108:177–179
124. Karger B, Meyer E, DuChesne A (1997) STR analysis on perforating FMJ bullets and a new VWA variant allele. *Int J Legal Med* 110:101–103
125. Karger B, Stehmann B, Hohoff C, Brinkmann B (2001) Trajectory reconstruction from trace evidence on spent bullets. II. Are tissue deposits eliminated by subsequent impacts? *Int J Legal Med* 114:343–345