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REVIEW ARTICLE

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Penetrating gunshots to the head and lack of immediate incapacitation

I. Wound ballistics and mechanisms of incapacitation

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Abstract There are two distinct mechanisms of ballistic injury. Crushing of tissue resulting in a permanent tract is the primary factor in wounding of most tissues and most body regions. Temporary cavitation causes radial tissue displacement and subsequent shearing, compression and especially stretching of tissue analogous to blunt trauma. In contrast to the effect in elastic tissue, temporary cavitation can contribute substantially to wounding of inelastic tissue, such as the brain. This is the case in penetrating gunshot wounds to the head. Additionally, the penetration of the bony cranium can produce secondary missiles in the form of bone or bullet fragments and a tendency of the bullet to deformation and early yaw. Most important, wounding resulting from temporary cavitation is greatly augmented by the confined space provided by the unyielding walls of the skull. Bone contact and enhanced effects of temporary cavitation result in an enlarged zone of disintegrated tissue and in high intracranial peak pressures. Morphological signs of powerful intracranial pressure effects are cortical contusion zones, indirect skull fractures and perivascular haemorrhages remote from the tract. Depending on ballistic and anatomical parameters, the intracranial effect varies from slightly more severe injury than in isolated soft tissue to an "explosive" type of injury with comminuted fractures of the skull and laceration of the brain. Incapacitation is the physiologically based inability to perform complex and longer lasting movements independent of consciousness or intention. Immediate incapacitation is possible following craniocerebral gunshot wounds or wounds that disrupt the upper cervical spinal cord only. Rapid incapacitation can be produced by massive bleeding from major vessels or the heart. Immediate incapacitation is the result of primary intracranial effects of the bullet. A mechanism similar to commotio cerebri applied extracranially does not exist in cases of penetrating gunshot wounds to the head.

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Introduction

Not every severe or even fatal gunshot wound causes immediate incapacitation (Spitz et al. 1961; Levy and Rao 1988; Newgard 1992). This can be illustrated by a case of suicide involving five perforating wounds to the heart; the man concerned fired four of the bullets from the same 7.65 mm pistol (Schrader 1942). In another case, a man was able to run more than 20 m after sustaining a chest wound from a 12-gauge shotgun at a range of 3–4 m. The pellets literally shredded the heart (DiMaio 1985).

A person's capability to act following a gunshot wound can be of major importance in reconstruction of a crime 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 and it may be possible to identify the person who fired the gun and to reconstruct 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.

The potential for physical activity following penetrating gunshot wounds to the head is especially difficult to assess. Penetrating craniocerebral gunshot wounds in civilian practice are reported to have a high early mortality rate of 90% or over (Kaufman et al. 1986; Siccardi et al. 1991). Immediate unconsciousness and incapacitation are therefore expected to be a necessary consequence and a constant finding. On the other hand, there are numerous publications reporting sustained capability to act follow-

ing penetrating ballistic head injury. These case reports will be systematically reviewed in the companion paper. The basis of this re-examination is formed by theoretical considerations regarding general principles of ballistic head injury and mechanisms of incapacitation from projectiles.

General wound ballistics

Wound ballistics can be defined as missile-tissue interaction (Fackler 1988). The biological effect is determined by parameters of the missile (mass, calibre, velocity, shape, material, construction, etc.) as well as of the tissue (density, elasticity, viscosity, anatomical structure, etc.). Two different mechanisms of injury can be distinguished when a projectile penetrates a dense medium, such as tissue (Harvey et al. 1945, 1947; Fackler and Malinowski 1985). Both are the result of one rapid dynamic process and can therefore be regarded as theoretical abstractions essential for the understanding of wound ballistics.

1. Crush mechanism. Tissue located in line with the trajectory is crushed by excessive pressure build-up directly in front of the tip of the moving projectile. The tissue completely disintegrates, resulting morphologically in the permanent tract (Harvey et al. 1945; Fackler and Malinowski 1985; Bowen and Bellamy 1988; Hollerman et al. 1990).

2. Stretch mechanism. 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 (Callender and French 1935; Harvey et al. 1945; Scott 1983). 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" (Harvey et al. 1945; Krauss 1957; Sellier and Kneubuehl 1994).

During the formation of the temporary cavity tissue is injured in three distinct modes (Fig. 1 a–c), summed up as the stretch mechanism (Fackler and Malinowski 1985; Bowen and Bellamy 1988; Hollerman et al. 1990) 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 $2\pi (r+x)$ (Black et al. 1941; Harvey et al. 1945). 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 (Scott 1983), 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" (Harvey et al. 1945) produces an asymmetrical temporary cavity and, consequently, shear forces inside adjacent layers of tissue (Hollerman et al. 1990). 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 (Harvey et al. 1945; Sellier and Kneubuehl 1994).

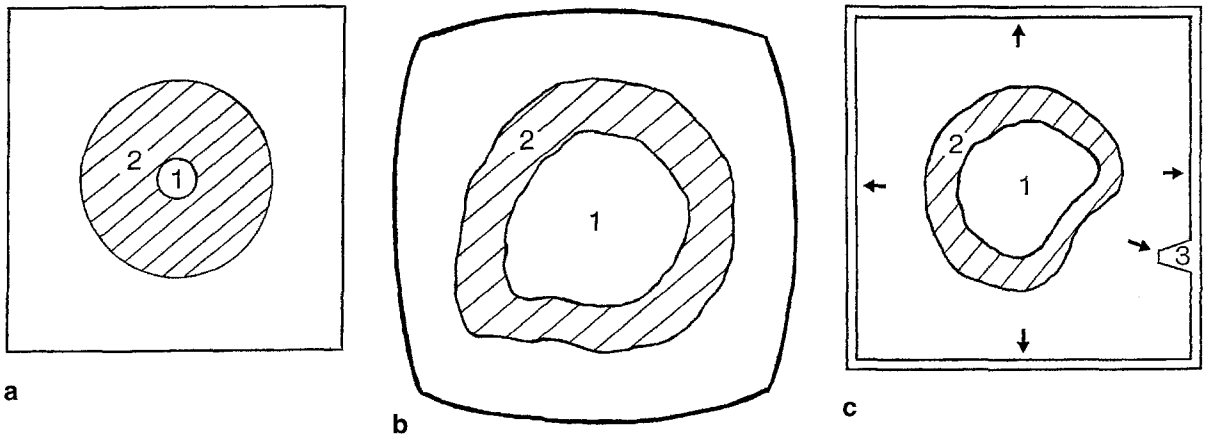

Fig. 1 a–c Temporary cavitation and stretch mechanism. Schematic illustrations of the sectional area of soft tissue before and after a projectile has passed. **a** Tubular tissue portions *before* the projectile has passed. *1* future trajectory, *2* tubular tissue portion around the trajectory. **b** 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 inhomogeneities cause 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 is confined in a rigid *casing*. *1* Maximum temporary cavity including future permanent tract. The diameter of the temporary cavity is smaller than in tissue not confined in a casing. *2* Same tubular tissue portion as in **a** and **b**. Compared with **b** the diameter of the 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. In addition, 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

The stretch mechanism of ballistic trauma then, is essentially nothing more than a localized blunt trauma analogous to displacement of tissue by a blow from a fist (Lindsey 1980; Fackler 1988). The maximum speed of moving tissue forming the boundary of the temporary cavity has been calculated to be in the range of 40 m/s for a so-called high-velocity missile (Beyer 1962; Fackler et al. 1984a). Because of the all-round tissue displacement in ballistic trauma, stretching usually outweighs shearing and compression, whereas in the case of a fist hitting a body compression predominates. In any case, the structural integrity of tissue displaced by cavitation will generally not be completely destroyed (Dzieman et al. 1961; Hopkinson and Watts 1963; Bowen and Bellamy 1988; Fackler 1988). 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 (Harvey et al. 1945; Sellier and Kneubuehl 1994). Thus, the term "Seitenstoßkraft" (= sideways force) used in the past by German-speaking ballistics experts (e.g. Kocher 1895) 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 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 μs) to move or injure tissue (Harvey et al. 1947; Fackler and Peters 1991).

It becomes apparent on differentiation of 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 (Fackler and Malinowski 1985; Bowen and Bellamy 1988). The crucial point for the realization of this wounding potential is the location of energy transfer and the ratio of distribution of the energy between the crush and the stretch mechanisms and the degree of elasticity of the tissues involved. The portion of the transferred energy used up in cavitation and stretching depends on several parameters of ballistics, such as striking velocity, mass and construction and generally increases with increasing velocity and decreasing mass (Bowen and Bellamy 1988; Fackler et al. 1988; Fackler 1991). Therefore, bullets having the same striking energy made up of different velocities and masses or even bullets with identical velocity and mass but different construction will produce very different injuries in identical tissue (Fackler et al. 1984b; Fackler and Malinowski 1985; Bowen and Bellamy 1988).

On the other hand, 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 tissue is, the less damage will be caused by the same amount of energy transferred to the stretch mechanism (Harvey et al. 1945; Fackler et al. 1984b; Bowen and Bellamy 1988). 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 extravasation small (Bowen and Bellamy 1988; Fackler 1988). 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 (Krauss 1957; Dzieman et al. 1961; Metter and Schulz 1983; Fackler et al. 1984b; Cooper and Ryan 1990).

These general principles of wound ballistics are modified by the deformation and fragmentation of a projectile. Deformation will increase the missile's cross-sectional area due to expansion, resulting in inhanced deceleration. The permanent as well as the temporary cavity will be larger and the penetration depth reduced compared to a non-deforming projectile (Fackler and Malinowski 1985; Fackler et al. 1988; Bowen and Bellamy 1988; Sellier and Kneubuehl 1994). A fragmenting bullet will also increase its cross-sectional area, although distributed among multiple missiles of smaller mass and reduced penetration depth (Fackler et al. 1984a). 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 susceptibility for the subsequent stretching of tissue. There is a synergistic effect of secondary missiles and cavitation (Fackler et al. 1984a; Fackler and Malinowski 1985; Fackler 1989; Cooper and Ryan 1990).

Compared with 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 favours deformation and fragmentation (Sellier 1971; Ragsdale and Josselson 1988) with the above-mentioned effects. Furthermore, a projectile tends to yaw after perforating bone (Ragsdale and Josselson 1988), resulting in a larger cross-sectional area and an enlarged permanent and temporary cavity. Frequently, bone contact causes additional secondary missiles in the form of bone fragments (Robens and Küsswetter 1982; Scott 1983; Ragsdale and Josselson 1988; Cooper and Ryan 1990). The accelerated bone fragments travel in diffeent directions in the vicinity of the bone, to a small extent even against the line of fire (Lorenz 1948). The effect of secondary bone fragment missiles is analogous to bullet fragments, enhancing the severity of the wound (Scott 1983; Bowen and Bellamy 1988; Cooper and Ryan 1990).

Special wound ballistics of the head

In intracranial gunshot wounds, several of the above-mentioned factors enhance the degree of tissue disruption. Because of the inelastic quality and the high water content of brain tissue, it is per se very vulnerable to cavitation and stretch mechanism (Fackler 1991). The penetration of the skull can imply the generation of secondary missiles in the form of bone or bullet fragments (Clemedson et al. 1973; Kirkpatrick and DiMaio 1978; Allen et al. 1983; Finnie 1993) and a tendency towards early tumling or deformation of the bullet. Kirkpatrick and DiMaio (1978), 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.

Inelastic tissue and preceding bone contact are also encountered in other regions of the body, but intracranial trajectories have a particular quality because the rigid skull functions as a non-yielding wall. Because brain tissue is almost incompressible, intracranial temporary cavitation and surrounding overpressures meet counterpressure 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 overpressures. 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 (Fig. 1).

Intracranial overpressures around the expanding temporary cavity, however, exceed the pressures found in nonconfined tissue. In a head model constructed from Plexiglas filled with 20% gelatin, fronto-occipital trajectories produced pressure peaks underneath the skull of 75 kPa (.22 lr, 2.57 g, 346 m/s) and 96 kPa (7.65 mm, 4.70 g, approx. 270 m/s) (Dittmann 1989). In dried human skulls filled with 20% gelatin the following quasistatic pressure peaks (as opposed to shock wave pressures) were measured for transverse shots with 6-mm steel spheres: 75 kPa (205 m/s), 430 kPa (450 m/s) and 2200 kPa (806 m/s) (Watkins et al. 1988). A 3-mm steel sphere (459 m/s) resulted in an overpressure of 65 kPa. In tangential shots, the peak pressures only reached about 10% of those from transverse shots. In nonconfined tissue, maximum peak pressures very close (!) to the temporary cavity of approximately 400 kPa have been reported for high-velocity spheres (Harvey et al. 1947).

Static overpressure does not necessarily injure tissue. That high intracranial pressure has an important role in ballistic wounding, however, is demonstrated by shooting a cat's head from which the brain has been removed by way of the foramen magnum, resulting in only minor damage to the skull compared with shots to intact heads (Butler et al. 1945). The 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, which means 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 (Fackler 1991). Consequently, the layer of cerebral tissue between temporary cavity and skull will be compressed much more strongly than tissue not confined in a rigid casing (Fig. 1). Analogous to blunt trauma, this compression can result in contusion of brain tissue, which is descernible as contusion zones in superficial layers of the brain remote from the trajectory (Freytag 1963; Henn and Liebhardt 1969). Inside the skull, then, the stretch mechanism of cavitation is greatly augmented by enhanced compression of tissue possibly causing contusion.

The skull will at first be slightly stretched by intracranial overpressures. In case the skull's capacity to stretch elastically is surpassed, there will be indirect skull fractures. If the internal pressures are high enough, indirect skull fractures will combine to an "explosive" type of head injury (Butler et al. 1945) with comminuted fractures of the skull and laceration of the brain.

Fig. 2 Autopsy finding: typical indirect fracture of the right anterior cranial fossa (roof of the orbita) after suicidal gunshot with a .45 ACP bullet. The entrance wound was located in the right temporal region, and the exit wound, in the left occipital region. Immediate incapacitation.

Therefore, three direct morphological effects of high intracranial overpressures can be identified:

1. Indirect fractures of the base or the vault. Fracture lines without contact to the primary bony entrance defect are certainly caused by internal overpressure. 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. 2), the ethmoidal plates in the anterior cranial fossa and the roofs of the cavities of the tympanum in the middle cranial fossa (Klaue 1949). Secondary radial fractures originating from the primary entrance defect most probably represent release of rapid circumferential stresses inside the bone induced by the bullet's impact (Kolsky 1980; Smith et al. 1987; König and Schmidt 1989). They are simply a sign of high local energy transfer during the missile's penetration of bone and are therefore caused before intracranial pressure rises. Tertiary concentric fractures connecting the radial fracture lines, on the other hand, are indirect heaving fractures (Kolsky 1980; Smith et al. 1987) functioning as additional stress relief for internal overpressures. This intracranial force directed outwards causes the wedges of bone between the bordering radial fractures to be levered upwards and outwards. Because concentric fractures are caused by internal overpressures several milliseconds after the bullet penetrates the bone, this heaving or levering mechanism results in independent concentric fractures of similar radii connecting the pre-existing radial fracture lines (Kolsky 1980; Smith et al. 1987). Isolated fracture lines, especially in the anterior cranial fossa, and concentric fractures around the primary penetration defect can therefore be regarded as direct signs of the intracranial pressure peaks in relation to the stretching capacity of the individual skull.

2. Cortical contusion zones. Superficial contusion remote from the tract is caused by impact of the brain tissue

against the skull, especially against pre-existing rims and edges. Impact against falx or tentorium can possibly have similar effects (Freytag 1963). Consequently, cortical contusions are reported to be most frequently located in the cerebellar tonsils and at the base of the frontal, temporal and occipital lobes (Freytag 1963; Henn and Liebhardt 1969; Kirkpatrick and DiMaio 1978). Not infrequently, skull fractures are found at corresponding sites (Henn and Liebhardt 1969). According to Freytag (1963), the morphology is identical with that of contusions occurring in head injury due to blunt and nonpenetrating impact: there are small petechial haemorrhages at the top of the gyri. The haemorrhages can be confluent and even occupy the entire gyrus or several of them (Henn and Liebhardt 1969).

Spatz (1941) described contusion foci in the form of small haemorrhages at the surface of the brain, which formed circles located at a certain distance around the brain wounds of entrance and exit. This location obviously differs from those described by the aforementioned authors. Although the contusion foci (Spatz 1941) might represent perivascular ring haemorrhages at the surface of the brain, the inconsistency underlines the fact that cortical contusion zones are not well understood (Unterharnscheidt 1993). A systematic and histological investigation of the frequency, location, morphology and mechanical genesis of cortical contusion zones in bullet injuries would help to clarify the situation (F. Unterharnscheidt, personal communication).

3. Intracerebral petechial haemorrhages remote from the tract in the form of classical perivascular ring haemorrhages or spherical haemorrhages (Allen et al. 1982, 1983; Finnie 1993). Allen et al. (1982) speculated on pressures disseminated through the vascular system but the author is of the opinion that it is the stretching and, especially, shearing of tissue that is responsible for these haemorrhages. They are simply the result of an enlarged zone of extravasation caused by 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 (Allen et al. 1982; Finnie 1993). But intracerebral petechial haemorrhages can also develop secondary to wounding (Illchmann-Christ 1951; Petersohn 1967; Tamaska 1968), so that in this respect autopsy findings do not necessarily reflect the situation immediately after injury.

The extent of "explosive" cerebral and cranial injury depends on a variety of ballistic parameters. In a simple experimental model for tangential wounds, the peak pressure was approximately dependent on the square of the projectile's velocity but not on the mass (Clemedson et al. 1973). The results of pressure measurements by Dittmann (1989) and Watkins et al. (1988), however, showed that projectile mass is also important in nontangential trajectories at least. 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 (Harvey et al. 1947; Watkins et al. 1988).

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 are reported to produce neural alterations 3–6 cm in diameter, as determined by light microscopical methods (Oehmichen et al. 1985). Lightweight bullets, such as 6.35 mm Browning or .22 rimfire, perforate ("through-and-through") in a minority of cases (Berg 1964; Sellier 1982). Projectiles possessing a higher penetration power, e.g. 9 mm Luger (= Parabellum) FMJ, commonly perforate the head (Berg 1964; Sellier 1982). Intracranial pressure peaks and their effects vary greatly depending on ballistic and anatomical parameters.

Centerfire rifles, whether designed for military or hunting purposes almost invariably cause a strong "explosive" effect with comminution of bone and laceration of at least part of the brain (Clemedson et al. 1973; DiMaio and Zumwalt 1977; Peng et al. 1990; Knudsen and Theilade 1993). A very rare and extraordinary form is the "Krönlein shot" (exenteratio cranii) where a more or less intact brain is ejected from the exploded vault (Krönlein 1899), especially in cases where the trajectory is close to the base of the skull (Mertens 1917; Pankratz and Fischer 1985). 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 (Sight 1969).

Incapacitation: definition

To Walcher (1929) and most other authors, incapacitation means the inability to act in a conscious and purposeful manner, such as escaping or attacking. In the title of their paper, Spitz et al. (1961) paraphrased delayed incapacitation as physical activity until collapse. Petersohn (1967) also used the term physical activity to define lack of incapacitation. He differentiated 4 degrees of physical activity:

Degree 1: Reflexes.

Degree 2: Automatisms such as continuing an activity started before unconsciousness began.

Degree 3: Instinctive reactions that are meaningful in the given situation like repulsing someone in a dreamy state of mind.

Degree 4: Conscius, purposeful activity according to the definition by Walcher (1929).

This complex classification is based primarily on the degree of consciousness and on underlying neurophysiological processes. But reflexes (degree 1) are part of agony and can be found in most forms of acute death. Automatisms (degree 2) are difficult to distinguish from other forms of movement. Goroncy (1924) postulated remnants of consciousness, and Meixner (1931) completely rejected automatisms in connection with delayed incapacitation. Although there certainly are different degrees of capability to act, then, degrees 1 and 2 at best represent primitive and short-

lived movements that are rapidly terminated. Their effect on the events and resulting stains will usually be negligible and easy to reconstruct from a forensic point of view.

On the other hand, completely undisturbed consciousness is not a prerequisite for purposeful activity (Strassmann 1935), as is demonstrated by degree 3 according to Petersohn (1967). Consequently, unconsciousness necessarily results in incapacitation, but determined physical activity does not necessarily depend on a completely undisturbed state of consciousness.

Therefore, a functional definition of capability to act regardless of the state of consciousness or intention will be used, in accordance with Strassmann (1935), Goroncy (1924) and degrees 3 and 4 of Petersohn (1967): 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 unavoidable inability to perform complex and longer lasting movements. The activity does not have to be appropriate in the current situation. For example pocketing or storing the firearm following a suicidal gunshot is not always achieved intentionally or purposefully. In contrast to reflexes or automatisms, however, this activity has a goal and does make sense in itself. The term "unavoidable" means based on physiological effects and not depending on psychological mechanisms such as pain or fright. Incapacitation has to be independent of the victim's cooperation. The latent period from hit to incapacitation should be short, although actual figures would be arbitrary.

Mechanisms of incapacitation

Decreasing the functioning capability of the central nervous system (CNS) is the only method of reliably producing incapacitation with bullets according to the above definition (Newgard 1992). There are two mechanisms by which this can be accomplished (Smith 1987; Fackler 1987, 1992; Newgard 1992): direct hits to the CNS will produce disruption of brain tissue. Hits causing acute and massive bleeding will result in reduced perfusion of the CNS with subsequent lack of oxygen and unconsciousness. This indirect elimination of the CNS is usually produced by perforating wounds of the heart or major vessels.

There is no other mechanism for reliable production of incapacitation. Many victims will collapse immediately when hit by a bullet, as will some who were missed but think they were hit. They collapse for psychological reasons (Fackler 1992). Nonetheless, collapse on a psychological basis is inconsistent and erratic. The only reliable fact 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 adrenaline. Excessive pain, for example, must first be perceived, and then this perception of pain must cause an emotional reaction (Smith 1987). Reliable incapacitation is based solely on physiological effects independent of any unpredictable psychological factors.

Another alleged mechanism of incapacitation is high energy transfer (Sturdivan 1969) or high energy deposit (DiMaio et al. 1974). Energy deposit is an abstract value considering neither the way (crush/stretch) nor the location of energy transferrence nor the type of tissue involved. Therefore, the energy deposited is of limited value in predicting effects in an actual shooting.

The momentum transferred to the target (Hatcher 1935) does not knock the human body down or drive it significantly backwards (Fackler 1992), even if such effects are shown a thousand times on television. The impulse transferred to an adult from a .45 ACP round results in a negligible backward motion of approximately 5 cm/s (Sellier and Kneubuehl 1994). Injury or even incapacitation from shock waves (Suneson et al. 1988) or a mysterious "nerve shock" postulated especially in hunting (Lampel and Langenbach 1961) are not supported by a single experiment or by theoretical considerations (Fackler and Peters 1991; Jason 1991). Instantaneous incapacitation can only be produced by direct hits to the CNS (Fackler 1987; Newgard 1992). In the case of hits causing acute blood loss, the speed of bleeding is too slow and compensation mechanisms are too effective for immediate incapacitation (Newgard 1992). Even if the heart is shredded (DiMaio 1985), the oxygen stored in the CNS will ensure a potential for physical activity for about 10 s more.

Theoretically, immediate incapacitation from gunshots to the CNS can result from primary or from secondary effects of the bullet. Cerebral pressure is the major secondary effect. Brain oedema usually shows a free interval before symptoms of cerebral pressure can be observed (Goroncy 1924; Meixner 1931). In the case of massive intracranial bleeding leading to cerebral pressure, the source and the speed of bleeding are important. But neurosurgical or combat surgical studies concerned with this subject (Schorstein 1947; Barnett and Meirowsky 1955) are of no help because the immediate posttraumatic time interval necessarily eludes evaluation.

Animal experiments demonstrated a continuous rise of intracranial pressure several seconds after impact up to maximum mean values of 40 mmHg after 30 s (Gerber and Moody 1972), 40–90 mmHg after 1–10 min (Crockard et al. 1977 a, b) and 20–60 mmHg after 1 min (Carey et al. 1989). Different trajectories and especially different firearms–animal combinations render the comparison with man difficult.

In a clinical study (Crockard 1975) of early intracranial pressure (ICP) after gunshot wounds to the brain (earliest measurement 20 min and most within 1 h after injury), there were both elevated (60–100 mmHg) and reduced (0–10 mmHg) and normal ICPs. All cases with reduced ICP bled profusely from large bony defects, whereas all cases with elevated ICP showed very little blood loss from small bony defects and small intracranial haematoma but severe, generalized oedema. The fact that most of intracranial bleeding in case of ballistic head injury can drain off through entrance or exit wounds has already been made responsible for similar observation by Schorstein (1947) and Barnett and Meirowsky (1955). The latter proposed a

"closed-box" mechanism in cases of substantial intracranial haematoma.

Therefore, ICP can show an early rise following ballistic craniocerebral injury. Massive bleeding, though, is a very unlikely reason for elevated ICP (Crockard 1975). Rather, rapidly developing cerebral oedema seems to be responsible, which is supported by the magnitude of ICP roughly depending on the amount of energy transferred (Carey et al. 1989). The latent period in the range of minutes until the ICP rises substantially in animal experiments is too long to produce immediate or very rapid incapacitation following a head shot, although during the further course elevated ICP can and will of course become symptomatic. Consequently, immediate incapacitation following gunshot wounds of the brain is the result of direct or primary bullet effects. Primary effects consist of destruction of essential areas of the CNS resulting in focal disturbances or elimination of consciousness (Goroncy 1924). Essential for voluntary physical activity are at least the vital areas (brain stem, diencephalon), motor paths of conduction (capsula interna, radiatio thalami) and motor areas (basal ganglia, cerebellum, motor cortex). Injury to sensory or optical areas may hamper the physical activity (Walcher 1929), but considering the complex relationships and unresolved questions there may also be other regions that are important for the capability to act.

Unconsciousness occurs when certain brain areas are injured by the bullet. The generation of a commotio cerebri, where the major symptoms are immediate unconsciousness and loss of muscle tone, has long been discussed in connection with cases of penetrating ballistic head injury (e.g. Naegeli 1884; Goroncy 1924; Klages et al. 1975). A commotio cerebri was thought to originate from the momentum transferred from the impacting projectile to the skull. Cerebral effects of an extracranial projectile have been described for nonpenetrating gunshots to the head in the form of cortical contusion zones underneath the impact site (Noetzel 1948). According to the classical and frequently corroborated studies by Denny-Brown and Russel (1941), however, the mechanogenesis of commotio cerebri is a matter of sudden acceleration of the skull, which by means of inertia of the brain results in wounding at coup and contrecoup. 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 (Unterharnscheidt 1993). A maxium acceleration of the skull will be achieved when the mass of the impacting object is equivalent to that of the head. The velocity of the object should be relatively high. In comparison, a projectile has a very small mass and a very high velocity, resulting in an ultrashort time span during which the projectile is acting upon the skull. Because of inertia, the skull as a whole will not essentially move during transfer of 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 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 the battlefields reporting lack of commotio cerebri in penetrating gunshots to the head (Payr 1922; Spatz 1941) as well as own unpublished results from gunshots to the heads of calves using two types of 9 mm Luger ammunition. During the first 30 ms after temporal impact, no systematic translocation of the head could be determined using high-speed films (B. Karger, unpublished results).

Conclusions

Crushing of tissue resulting in a permanent tract is the primary factor in wounding of most tissues and most body regions. The temporary cavity itself does not hurt or injure. It is a dynamic gas-filled cavity. The tissue displaced by temporary cavitation is injured by a mechanism analogous to blunt trauma, resulting in shearing, compression and especially stretching of tissue. This usually does not contribute significantly to wounding of elastic tissue. Temporary cavitation can play a substantial role in trauma to inelastic tissue such as liver or brain.

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. Bone contact can produce secondary missiles and a tendency to deformation and early yaw. Wounding from temporary cavitation is greatly augmented by the confined space provided by the unyielding walls of the skull. Bone contact and enhanced effects of temporary cavitation result in an enlarged zone of disintegrated tissue and in high intracranial peak pressures. Cortical contusion zones, indirect skull fractures and perivascular haemorrhages remote from the tract are morphological signs of powerful intracranial pressure effects. Depending on ballistic and anatomical parameters, the intracranial effect varies from slightly enhanced compared to isolated soft tissue to a devastating injury with explosion of the neurocranium and laceration of large areas of the brain.

Incapacitation is defined as a physiologically based inability to perform complex and longer lasting movements. Immediate incapacitation is possible from craniocerebral gunshots only. Rapid incapacitation can be achieved with hits producing massive bleeding (heart, major vessels). Immediate incapacitation is the result of primary intracranial (or upper cervical spinal cord) effects of the bullet. Cerebral pressure (secondary effect) is commonly associated with a latent period. A mechanism similar to commotio cerebri does not exist in cases of penetrating gunshot wounds to the head.

Despite special wound ballistic characteristics of the head producing enlarged intracranial tissue disruption, there have been numerous publications in the forensic literature reporting lack of incapacitation following penetrating gunshots to the head. Therefore, these case reports will be reviewed in the companion paper on the basis detailed in the present paper.

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