Electroencephalogram, Circulation, and Lung Function After High-Velocity Behind Armor Blunt Trauma

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Background: Behind armor blunt trauma (BABT) is defined as the nonpenetrating injury resulting from a ballistic impact on personal body armor. The protective vest may impede the projectile, but some of the kinetic energy is transferred to the body, causing internal injuries and occasionally death. The aim in this study was to investigate changes in electroencephalogram (EEG) and physiologic parameters after high-velocity BABT.

Methods: Eight anesthetized pigs, wearing body armor (including a ceramic plate) on the right side of their thorax, were shot with a 7.62-mm assault rifle (velocity approximately 800 m/s). The shots did not penetrate the armor and these animals were compared with control animals (n = 4), shot with blank ammunition. EEG and several physiologic parameters were thereafter monitored during a 2-hour period after the shot.

Results: All animals survived during the experimental period. Five of the exposed animals showed a temporary effect on EEG. Furthermore, exposed animals displayed decreased cardiac capacity and an impaired oxygenation of the blood. Postmortem examination revealed subcutaneous hematomas and crush injuries to the right lung.

Conclusion: The results in our animal model indicate that high-velocity BABT induce circulatory and respiratory dysfunction, and in some cases even transient cerebral functional disturbances.

Key Words: Behind armor blunt trauma, BABT, EEG, Blunt trauma, Pulmonary contusion.

J Trauma. 2007;63:405–413.
much larger in BABT and covers a smaller surface area, although the peak velocity of the thoracic wall should not be considered equal to bullet velocity.

The aim of the present study was to investigate the pathophysiologic changes in brain function, circulation, and respiration that may arise after BABT caused by a high-velocity projectile. Our results demonstrate that high-velocity BABT induce circulatory and respiratory disturbances and in some cases even EEG changes.

MATERIALS AND METHODS

Measurement of Back Face Deformation

These tests were performed before animal experiments to evaluate the deformation of the body armor, measured as impression in ballistic plasticine (National Institute of Justice [NIJ] Standard 0101.04). The body armor was a specially manufactured vest segment, corresponding to the Swedish Armed Forces standard issue, Mark m/94 (Åkers Krutbruk Protection AB, Åkers Styckebruk, Sweden), size 255 × 300 mm, consisting of a ceramic plate and 14 underlying layers of aramid fabric. A layer of cotton fabric was placed between the block of plasticine and the armor to simulate a field shirt. Four body armors were used for the back face deformation test and only one shot was fired at each.

A standard assault rifle (Swedish Armed Forces Mark AK4) equipped with a laser-aiming device (Diode laser type S 1889, Melles Griot, Täby, Sweden) was used throughout the experiments. The weapon was fixed to a small gun carriage and placed 10 m in front of the target. All ammunition was of Danish issue, NATO type, 7.62 × 51 mm; (M/94, Ammunition-arsenalet, Frederikshavn, Denmark). Projectile velocity was measured with an optical shutter device (Chronograph Beta model, Shooting Chrony, Inc. Mississauga, Ontario, Canada).

Mean bullet impact velocity was 802 (799–806) m/s and mean impression in plasticine was 28 (24–31) mm. In the subsequent animal experiments, the eight exposed pigs were protected by an equivalent body armor as the one during the impression depth measurements.

Animal Experiments

An ethics committee (Permit A1-2000, Umeå, Sweden) approved the investigation. All animal studies were performed according to the Guide for the Care and Use of Laboratory Animals.13 Animals were accommodated in an accredited animal facility at least 2 days before the experiment and fed a standard diet with free access to tap water. Ambient room temperature was maintained at 21°C to 22°C with a photoperiod of 12 hours of light and 12 hours of darkness. Twelve Swedish landrace pigs (females or castrated males) with a mean body mass of 63 (50–91) kg were used. Eight were shot with live ammunition and four were used as controls (shot with blank ammunition).

Animal Preparation, Blood Sampling, Recording of Circulatory and Respiratory Parameters

The experimental setting is displayed in Figure 1. The anesthesia was performed with infusion of ketamine hydrochloride 50 mg/mL (Ketalar, Parke-Davis, Pontypool, Gwent, Great Britain) and pethidin hydrochloride 50 mg/mL (Petidin, Pharmacia-Upjohn, Stockholm, Sweden). Tracheotomies were performed and the animals were mechanically ventilated in a volume-controlled mode with room air (Siemens Servo Ventilator 900C, Siemens-Elema, Solna, Sweden) at a rate of 20 breaths/min and the tidal volume was adjusted to achieve normoventilation.
Through a cervical cut-down, the left carotid artery was cannulated with a polyethylene catheter (Portex Ltd., Kent, England) to measure systolic, diastolic, and mean arterial blood pressure (MAP).

An optical pulmonary thermodilution catheter (Opticath, Abbot, 7 French Critical Care Systems, Al Zwolle, Netherlands) was inserted into the right external jugular vein, for measurements of central venous pressure (CVP), mean pulmonary artery pressure (MPAP), cardiac output (CO), mixed venous saturation (SvO₂), and body core temperature. After the preparation was completed, animals were allowed 30 minutes rest to achieve steady state.

Electrocardiogram (ECG), MAP, CVP, and MPAP were measured and monitored with a Sirecust 960 (Siemens Medical Electronics, Danvers, MA).

Blood samples were obtained from the arterial line for analysis of arterial blood saturation (SaO₂), Po₂, Pco₂, Na⁺, K⁺, Ca²⁺, pH, base excess (GEM Premier Plus analyzer, Instrumentation Laboratories, Milano, Italy), lactate (Miniphotometer, 8, DR Lange GmbH, Berlin, Germany), whole blood hemoglobin (Hb) (Hemoglobin Photometer Electrolux, Mecatronic AB, Helsingborg, Sweden), and blood glucose (B-Glucose analyzer, Hemoce AB, Angelholm, Sweden).

All parameters were recorded at baseline, 1, 5, 10, and 15 minutes after impact, and thereafter every 15 minutes until the end of the experiment at 120 minutes.

In addition to the directly measured physiologic variables, a number of variables were calculated according to the formulas in Table 1.

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**Recording of Electroencephalogram**

Electrical activity from the brain cortex was registered by bipolar electroencephalogram (EEG) in seven of the eight experimental animals and in the four controls. In the exposed group, EEG recording was not performed in one of the pigs because of technical problems. Registration of the EEG signal was performed with five electrodes screwed into the outer part of the skull bone in the midline over the frontal and parietal lobes, bilaterally over the temporal lobes and at the vertex. The registration was made with a mobile eight-channel EEG recorder (Model No. EEG-7209, Nihon Kohden Corporation, Tokyo, Japan). Two ground electrodes were placed subcutaneously in the neck. An equilibration time of 30 minutes was allowed before starting of the EEG recording.

The EEG recording started 5 minutes before the firing of the weapon, to get a baseline pattern, and was continued until 15 minutes after the impact followed by 2-minute recordings every 15 minutes. This recording protocol continued until the animal was killed at 120 minutes. An experienced specialist in clinical neurophysiology manually analyzed the EEG sheets.

The EEG pattern was graded into one of five different levels according to the estimated change in frequency and amplitude over time: (1) slight to moderate reduction in frequencies, i.e., a reduction of the fast frequency band (slowing in frequency range); (2) pronounced reduction in frequencies, i.e. a dominance of the slow frequency band (marked slowing in frequency range); (3) overall reduction in amplitudes of 50% or more (depression pattern); (4) short bursts of slow activity with an otherwise global suppression of all cortical activity (burst-suppression pattern); (5) a totally suppressed EEG pattern (isoelectric pattern). An illustration of the EEG levels is outlined in Figure 2A.

This grading was based on earlier observed changes in EEG pattern after experimental concussion in awake animals, which in its turn was graded according to a previous established staging system for concussion in humans. The pre-exposure baseline pattern was graded as zero.

**Armor and Shooting Procedure**

The armor was firmly attached to the right side of the thorax with two 3-cm broad girdles.

Twelve animals were randomized, immediately before the shooting with live ammunition (8 in the exposed group) or blank ammunition (4 in the control group). The firing of the gun was synchronized to the endpoint of the inspiratory phase.

The control animals were subjected to gun shot with the same weapon but using blank ammunition. The amount of gunpowder in the blanks was adjusted to produce a similar sound level as the live ammunition.

**Postmortem Examination**

After 2 hours observation time, the animals were killed with pentobarbital 60 mg/mL, 70 mL or more intravenously

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**Table 1 Abbreviations and Calculations for Variables Used in the Present Study**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Variable</th>
<th>Source</th>
<th>Unit</th>
</tr>
</thead>
<tbody>
<tr>
<td>SV</td>
<td>Stroke volume</td>
<td>CO/HR</td>
<td>mL/stroke</td>
</tr>
<tr>
<td>SVR</td>
<td>Systemic vascular resistance</td>
<td>80 × (MAP – CVP)/CO</td>
<td>mm Hg/L/min</td>
</tr>
<tr>
<td>SvO₂</td>
<td>O₂ saturation, mixed venous</td>
<td>Pulse oximetry (a. pulmonalis)</td>
<td>%</td>
</tr>
<tr>
<td>SaO₂</td>
<td>O₂ saturation</td>
<td>Pulse oximetry (a. carotis)</td>
<td>%</td>
</tr>
<tr>
<td>VO₂</td>
<td>Oxygen uptake</td>
<td>CO × 0.0139 × Hb × (SaO₂ – SvO₂)</td>
<td>mL/min</td>
</tr>
<tr>
<td>DO₂</td>
<td>Oxygen delivery</td>
<td>CO × (0.0139 × Hb × SaO₂)</td>
<td>mL/min</td>
</tr>
<tr>
<td>O₂ER</td>
<td>Oxygen extraction ratio</td>
<td>VO₂/DO₂ × 100</td>
<td>%</td>
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until the ECG became isoelectric. An autopsy was performed on all 12 animals directly after the euthanasia and they were examined for gross pathologic findings. The thorax wall, the lungs, the heart, the liver, and the bowels of the upper part of the abdomen were examined.

**Statistical Methods**

The experimental design was two groups followed over time. Measurements were performed at 12 predefined time points. Because some data were missing at the late phase of the experimental period, we used a linear mixed-effects model for the statistical analysis.16 PROC MIXED in the statistical package SAS (SAS Institute, Inc., Cary, NC) was used for the analysis. In the diagrams, the least square means are depicted inside 95% confidence intervals. \( p \) values of 0.05 or less were considered significant.

**RESULTS**

**Animal Experiments**

Body mass in the exposed group was 62 (50–91) kg, and in the control group 66 (58–72) kg. In four of the eight exposed animals, bullet velocity was recorded and found to be 802 (797–811) m/s, which is in agreement with the results from the back face deformation study.

All animals in the two groups survived the whole observation period of 120 minutes postinsult. No hemoptysis was observed.

**EEG**

There were no differences in the baseline EEG pattern between exposed and control animals or between the individual animals in the experimental and control groups, respectively. In five of the seven EEG-monitored exposed animals there was a change in the EEG pattern within 20 seconds after the trauma (Fig. 2B). The observed changes were light to moderate reductions in frequencies, which were seen bilaterally over the hemispheres without any obvious side differences. In all of these animals, there were gradual returns to the baseline pattern within 2 minutes after exposure. These animals remained at baseline during the rest of the observation period. In the two remaining exposed animals, there were no changes in EEG activity during the observation period. In all four control animals the EEG activity was unchanged compared with baseline throughout the experimental course.

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**Fig. 2.** (A) Grading of EEG-changes. Recorded EEG in an exposed animal is outlined. EEG levels 0 (baseline)–5 (isoelectric EEG) are depicted. (B) EEG-changes 1 minute after impact. Decrease in EEG activity is shown for 7 of 8 exposed animals.
Circulatory Effects

In the exposed group, blood pressure expressed as MAP dropped from 116 mm Hg (baseline) to 91 mm Hg during the first minute (Fig. 3A). This change was significant compared with baseline \((p < 0.05)\), but there was no significant difference compared with the control group at this time. Exposed animals demonstrated a fast recovery of MAP in this study. We observed baseline values of MAP as early as 5 minutes after the shot.

CO in exposed animals demonstrated a marked drop from 5.5 L/min to 4.2 L/min during the first minute after the shot (Fig. 3B). The difference between the groups was significant at 1 to 10 minutes postimpact \((p < 0.05)\), followed by a gradual recovery in exposed animals. Control animals (shot with blank ammunition) demonstrated a gradual decrease of CO during the experimental course. We did not observe any significant overall differences in CO between control and exposed animals.

The difference in stroke volume (SV) between the groups was significant at 1, 5, and 10 minutes after impact \((p < 0.05)\). We observed a marked drop of 25% in stroke volume (SV) during the first minute (Fig. 3C). At 15 minutes after impact, exposed animals had recovered to a certain degree, followed by a gradual decrease. Control animals demonstrated a gradual decrease of SV during the experimental course. We did not observe any significant overall differences of SV between control and exposed animals.

Respiratory Effects

We observed a decline of \(\text{SaO}_2\) immediately after impact and a continued decrease during the first 30 minutes (Fig. 4A). The lowest value for \(\text{SaO}_2\) was recorded at 30 minutes after impact followed by a gradual recovery. We did not observe any significant overall differences between control and exposed animals.

The mixed venous oxygen saturation (\(\text{SvO}_2\)) displayed a somewhat different pattern. We observed a gradual decrease until 15 minutes after the shot and low remaining values during the rest of the experiment (Fig. 4B). The overall difference between the groups was significant \((p < 0.05)\).

Oxygen Transport Effects

Oxygen delivery (\(\text{DO}_2\)) and oxygen extraction (\(\text{O}_2\text{ER}\)) are calculated values influenced by both reduction of CO and saturation of the arterial blood (Table 1). For \(\text{DO}_2\), we observed a significant difference between exposed and control animals 1, 5, 10, and 30 minutes after impact \((p < 0.05)\) (Fig. 5A). We did not observe any significant overall differences between control and exposed animals \((p = 0.076)\). The reduction in \(\text{DO}_2\) was compensated by an increased \(\text{O}_2\text{ER}\), which was enhanced as early as 1 minute after the impact (Fig. 5B). The difference in \(\text{O}_2\text{ER}\) between groups was significant at 5 and 15 minutes \((p < 0.05)\). We did not observe any significant overall differences of \(\text{O}_2\text{ER}\) between control and exposed animals \((p = 0.066)\).

Blood Analysis

Lactate showed a significant overall difference between exposed and control animals \((p < 0.05)\). The blood levels of lactate increased over time in exposed animals but seemed to stabilize at the later phase of the observation period (Fig. 6). Control animals were unchanged during the whole experiment. \(\text{Na}^+, \text{K}^+, \text{Ca}^{++}\), pH, base excess, hemoglobin, blood
glucose, and the temperature did not show any significant difference (data not shown).

**Postmortem Examination**

Beneath the body armor point of impact, an almost circular skin lesion consisting of cutaneous abrasion with a subcutaneous hematoma were evident in all exposed animals but there was no laceration of the skin. The mean diameter of the skin lesion was 6 (4.5–8) cm. In four exposed animals, fractures of one or two ribs were found beneath the skin abrasions. Lacerations and hemorrhage in the intercostal musculature were noted.

All exposed animals exhibited a hematoma in the lung lobe underlying the impact area (most often the middle lobe of the right lung). In two animals, a small laceration of lung tissue was also demonstrated. There was no hemothorax or pneumothorax. The examination of the left lung, heart, liver, and bowels did not show any macroscopic injuries. No gross pathologic finding was demonstrated in the control animals except for blue discoloring of the dorsal parts of the lung.

**DISCUSSION**

It is evident that the BABT exists as the injury that can be induced by the impact of a bullet into body armor. There is unpublished information, from military channels, about...
EEG and Physiologic Parameters After BABT

fatal incidents after nonpenetrating impacts. However, a problem in the study of BABT is the lack of epidemiologic data. It is also impracticable to perform experiments on living humans. Human corpses can provide some information about structural injuries, like fractures, but not on pathophysiologic events in vivo over time, which are possible to monitor during animal experiments. Simulation models have been used, but the models are no better than their input data, and it is obvious that the existing knowledge about the mechanisms is too weak to permit reliable descriptions of the relationship between applied violence and resulting injury. Information about the consequences of BABT is important for development of protective equipment, for military tactics, and for medical planning. The experimental setup in our study, with a shot from a military rifle fired at the right thorax side, is the standard proposed by the North Atlantic Treaty Organisation (NATO) Task Group on BABT (TG-BABT = TG HFM-001). It gives a maximal exposure of the lung and a lesser exposure of the heart. If the target had been over the heart, or in full expiration, the physiologic response could have been different. Some previous studies have been performed on impacts against other anatomic points of the chest using different experimental models. In one study, when the strikes were against the mid-sternum they observed sternal fractures, cardiac contusion, and dysrhythmias such as ventricular fibrillation. Disrupted aortic valves were observed in one animal model using goats and targeting the “cardiac window.” None of these cardiac injuries or arrhythmia were observed in our study. In those cases where the impacts were over the lung parenchyma, the injuries were similar to ours.

The AK-47 assault rifle (Kalashnikov), with a peak velocity of approximately 700 m/s, was the most commonly used weapon of the native Somalia troops, demonstrating the relevance of using high velocity weapons in BABT studies. In our study, the bullet weight of 9.5 g with a velocity of 802 m/s led to a kinetic energy of 3.06 kJ, which was reduced to 0 in about 25 microseconds after impact.

If the pig had been shot without the armor, it would have led to immediate death. During this study, none of the exposed animals died, but on the contrary, no animal was unaffected. Half of the shot animals (4 of 8) had rib fractures and all had a lung contusion. This contusion with the accompanying lung hemorrhage was the most serious gross injury in exposed animals. A similar BABT-induced contusion has been described previously by several authors.

The most prominent disturbances of physiologic parameters in the present study were the changes in brain activity, circulation, and oxygenation. The initial drop in CO2, which seems to be most related to a drop in SV resulting from the fact that heart rate was nearly unaffected, resulted in a temporary drop in systemic blood pressure (MAP). The arterial saturation was deteriorated and this, together with the reduced CO2, led to a marked drop in DO2. An increased O2ER compensated for the reduced DO2, but this compensation did not seem to be sufficient, since the blood lactate level increased as a sign of peripheral anaerobic metabolism. It is unlikely that this reduction in oxygenation should be caused only by the primary lung contusion. Other effects, such as intrapulmonary shunt, with a similar pathophysiology as pulmonary embolism, may lead to excessive capillary flow in nontraumatized lung areas. These lung areas might normally be less ventilated leading to decreased capacity of blood oxygenation. Furthermore, hemorrhage inside the lung resulting in occluded alveoli, or production of inflammatory mediators leading to decreased respiratory function in both lungs must be taken into account. The hypothesis that difference in saturation between the groups in our study is caused by lung edema declivis is not likely, because the unexposed control animals also were maintained in a supine position during the experimental course. However, it should be mentioned that the control animals demonstrated a slightly decreased saturation, which might be caused by edema declivis.

EEG changes were one of the most interesting findings in this study. This is, to our knowledge, the first study to demonstrate that high-velocity BABT induce EEG changes, which may be an indicator of brain dysfunction. The rapidly occurring generally transient changes in the EEG pattern, which was demonstrated in five of the seven exposed animals, were different from the baseline patterns recorded in the control animals, indicating that the trauma almost instantly elicited a global cerebral dysfunction.

The EEG showed within 20 seconds a pattern similar to the one observed in animals subjected to experimental head concussion. Similar factors responsible for producing experimental concussion in animals seem responsible for EEG changes after head injury in humans. An immediate slowing of the EEG activity has been previously described in pigs after high energy missile trauma in the hind limb. It has also been shown that pressure waves generated by high-energy missile impact in the thigh can be recorded in the abdomen as well as within the brain.

The EEG pattern is registered from the brain cortex, but because the cortical activity is dependent on input from subcortical brain structures, the observed changes could reflect a direct effect on vital centers in the brain stem such as the center for wakefulness. Also, the fact that the EEG changes were general could be an indication that the signals were elicited from deep brain structures, and consequently were not signs of local processes from more superficial structures of the hemispheres.

The lack of EEG changes in two of the recorded exposed pigs might be a result of a more profound anesthesia, because anesthesia is known to stabilize neuronal membrane function. However, ketamine (10.0 mg/kg) has previously been shown to increase the spectral amplitudes of low- and intermediate-frequency EEG components and decrease high-frequency voltage in pigs. Although our experimental protocol is standardized, small differences in body weight or where the impact was on the torso must be taken into account.
Biologic variation in sensitivity to trauma might also cause some deviation in our results.

The mechanisms behind the depressed brain activity remain unclear. The changes may occur as a result of a direct cerebral reaction to the transmitted shock wave, a sensory signal transmitted via the peripheral nervous system, hemodynamic effects of circulatory disturbances, and hypoxia, or a combined effect. The assumption that the EEG changes in our study would be secondary to hypoxia caused by transient apnea is not likely because all animals were mechanically ventilated.

Irrespective of the pathophysiologic mechanisms behind these EEG changes, the suppression most likely indicate a transient cerebral functional disturbance. These results clearly demonstrate the importance of studying a possible transient cerebral functional disturbance. These results indicate the suppression most likely indicate a transient cerebral functional disturbance. These results as have been shown in other trauma models. 37

In the future, it would be desirable to have a protection that could defeat realistic threats (like 7.62-mm rifle rounds) without any injuries or negative effects on the performance of the bearer. One obvious wish is to make the body armors lighter, because combatants already carry or wear as much as 45 kg (100 lb) of equipment into combat. 38 Another attribute to strive for is increased protection level, to meet projectiles that are heavier, with higher speed and that are more penetrative. Given these facts, it seems reasonable that the BABT problems will increase and it is therefore desirable that the pathophysiologic effects of BABT are further investigated. Spontaneously breathing exposed animals and the effects of apnea on EEG activity should be addressed in future studies.

ACKNOWLEDGMENTS

This work was supported by the Swedish Armed Forces. We thank Dr Peter Juel Thias Knudsen, Department of Forensic Medicine, University of Southern Denmark, Odense, for valuable advice; Magnus Backheden for excellent statistical analysis; and Elisabeth Malm for skilful technical assistance.

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