Research Article

Optoacoustic detection of intra- and extracranial hematomas in rats after blast injury

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A B S T R A C T

Surgical drainage of intracranial hematomas is often required within the first four hours after traumatic brain injury (TBI) to avoid death or severe disability. Although CT and MRI permit hematoma diagnosis, they can be used only at a major health-care facility. This delays hematoma diagnosis and therapy. We proposed to use an optoacoustic technique for rapid, noninvasive diagnosis of hematomas. In this study we developed a near-infrared OPO-based optoacoustic system for hematoma diagnosis and cerebral venous blood oxygenation monitoring in rats. A specially-designed blast device was used to inflict TBI in anesthetized rats. Optoacoustic signals were recorded from the superior sagittal sinus and hematomas that allowed for measurements of their oxygencations. These results indicate that the optoacoustic technique may be used for early diagnosis of hematomas and may provide important information for improving outcomes in patients with TBI.

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1. Introduction

About 150,000 patients per year suffer moderate or severe traumatic brain injury (TBI) in the USA. All of these are at risk for intracranial hematomas including extra-axial hematomas (both subdural and epidural), as are some of the 1.35 million patients per year who suffer mild traumatic brain injury. Subdural hematomas result from bleeding between the arachnoid mater and dura mater, while epidural hematomas results from bleeding between the dura mater and the skull. Extracranial hematomas are also common in TBI patients, but pose less risk compared to intracranial hematomas. All acute neurologic emergencies require rapid identification of pathophysiologic processes that are amenable to surgical or medical treatment [1–9]. For example, patients with acute intracranial hematomas that are sufficiently large to compromise cerebral blood flow require emergent surgical drainage in order to increase the likelihood of an acceptable neurologic outcome. Evacuation of acute intracranial hematomas within four hours is recommended [3].

In general, hematomas are formed by rupture of blood vessels and internal bleeding in tissues. Instead of circulating in blood vessels, blood accumulates in tissues and can induce damage, in particular, damage to brain tissue directly exposed to blood in intracranial hematomas [1–4]. Before the advent of computed tomography (CT) and magnetic resonance imaging (MRI), acute intracranial hematomas were diagnosed using physical examination and cerebral angiography. The development of CT and MRI permitted rapid, high-resolution, noninvasive diagnosis, but could not be used until a patient arrived at a major health-care facility. However, more rapid, accurate diagnosis before arrival at a major facility could reduce the time required before definitive evacuation.

We proposed to use an optoacoustic technique to detect and characterize intracranial and extracranial hematomas. Optoacoustics (which is based on absorption contrast) is a promising technology for both blood vessels and hematoma detection and characterization [10–15] because hemoglobin is a major chromophore in the near-infrared spectral range [16]. In cases of severely reduced brain perfusion, more rapid diagnosis and treatment could improve outcome. Unlike bulky CT and MRI equipment, an effective optoacoustic system can be small, portable and easily transported in...
an emergency vehicle. Unlike CT or MRI, heavy shielding and avoidance of magnetic objects is unnecessary. Although diagnosis of delayed intracranial hematomas in hospitalized patients can be definitively accomplished using CT or MRI, more rapid optoacoustic assessment that does not require transport to the radiology department could save time and reduce morbidity in hospitalized patients who suffer sudden neurologic deterioration.

Optoacoustic technology that can detect intracranial hematomas can also recognize abnormalities in overall brain perfusion because of associated reductions in oxygen saturation, including that in the superior sagittal sinus (SSS) [10–15,17–19]. The same pre-hospital device can both diagnose intracranial hematomas and document inadequate cerebral perfusion by detecting low SSS hemoglobin saturation.

Patients with mild, moderate or severe traumatic brain injury and nontraumatic coma represent a population of at least 2,000,000 patients who are likely to be emergently transported to the hospital and for whom pre-hospital diagnosis of or exclusion of intracranial hematomas may dramatically reduce the time necessary for definitive diagnosis and therapy after hospital arrival. Formulating a preliminary diagnosis during pre-hospital transport could provide invaluable information to guide emergency diagnosis and treatment after hospital admission. For instance, if a patient with a deteriorating level of consciousness before hospital admission had an optoacoustic diagnosis of epidural hematoma, surgeons and other hospital personnel could initiate preparations for emergent craniotomy. If that patient were located in a rural area remote from a major medical center, evidence of an intracranial hematoma could prompt transport by helicopter rather than ambulance to speed arrival at a center that could provide emergency neurosurgical treatment.

In the present study, we used a prototype optoacoustic system to detect blast-induced extra- and intracranial hematomas in a small animal model. The mechanisms of blast injuries in humans can be classified into at least 4 types [20]. A primary injury results directly from blast over/underpressure. Secondary blast injury results from shrapnel (energized fragments from the explosive) or debris accelerated by the blast wind. Tertiary injuries result from acceleration of the body by the blast wave and/or wind and may include tumbling, impact onto hard surfaces, or crushing. Quaternary injuries include all other effects, such as burns and poisoning. All of these classifications apply to animal models as well.

2. Methods

In optoacoustics, a short pulse of light (typically, near-infrared light for deeper penetration) induces a rapid increase in pressure in an absorbing sample due to stress confinement (i.e. the duration of the light pulse is significantly shorter than the time required for the pressure to dissipate). Given constant pulse energy and duration, the magnitude of the pressure depends on the Gruneisen parameter (\(\Gamma\)) of the sample and the absorbed energy density:

\[
P(z) = \Gamma \mu_a F(z)
\]

(1)

\[
\Gamma = \frac{\beta c^2}{C_p}
\]

(2)

Here \(P(z)\) is the axial pressure distribution (1-D case), \(\mu_a\) is the optical absorption coefficient of the sample, and \(F(z)\) is the depth dependence of fluence (light energy delivered per unit area). In many biological tissues (e.g. blood), the acoustic properties are essentially those of water, but \(\mu_a\) depends on the concentration of chromophores (e.g. hemoglobin) and on the excitation wavelength (\(\lambda\)) [16]. Irradiating a sample with multiple wavelengths will produce acoustic signals that can be used for quantitative analysis of the sample’s absorption spectrum. For blood, we can determine the oxygen saturation using the \(\mu_a\) values obtained from a multi-wavelength optoacoustic measurement and the spectra of oxy- and deoxyhemoglobin [10–15].

The optoacoustic system used in this study (Fig. 1) was designed for continuous monitoring of oxygenation in a variety of blood vessels [11,12,15]. An optical parametric oscillator (OPO) (Oplette 532 II, Optotek Inc., Carlsbad, CA) outputs short (<20 ns) pulses of near-infrared (NIR) light in the range from 680 to 2400 nm, with pulse energy <8 mJ and repetition rate 20 Hz. This spectral range includes the 700–1064 nm spectral range which is best for accurate oxygenation monitoring in deeper blood vessels as shown in our previous studies in large animals (sheep) when we validated our noninvasive method using invasive measurements of the SSS blood oxygenation (blood sampling followed by Co-Oximeter measurements) [11,12,15]. As in the previous works, the ratio of peak-to-peak optoacoustic amplitudes was used for accurate oxygenation measurements. Although direct validation of noninvasive oxygenation measurements in small animal studies is impossible with the invasive measurements due to the size limitations, the high accuracy of SSS blood oxygenation measurements obtained with the system in thicker tissues in our large animal studies justified its application in the rat studies.

The optoacoustic probe is a broadband ultrasonic receiver made of a 6 cm wide disk of piezoelectric film (110 \(\mu\)m PVDF), with a 1-mm optical fiber in the center for object irradiation and an integrated 20 dB preamplifier [19]. The energy emitted from the probe did not exceed 0.5 mJ, so given the circular laser spot with diameter 1 mm, the radiant exposure on skin was <64 mJ/cm². The lateral resolution of the probe is 1–2 mm depending on target depth, while the axial resolution is limited by the system’s ultrasound bandwidth and is <0.2 mm. The probe displacement was performed manually, however the peak amplitudes were
measured and displayed automatically by the system itself. After the blast, new peaks appeared indicating to formation of new areas with blood. The acoustic signal is amplified by a low-noise 40 dB second-stage amplifier and recorded by a high-speed digitizer (NI USB-5132, National Instruments, Austin, TX) at 8 bits resolution and 50 MS/s. The operator interface is a laptop (Dell Precision M4500) running Windows 7 and custom LabVIEW software. The software controls the OPO and analyzes signals in real time to allow for unattended monitoring once the probe is secured.

In this study, we attempted to isolate the effects of primary and tertiary blast injury while minimizing all other effects. The custom-made Vandenberg blast device (Fig. 2) uses nail gun cartridges inserted into a detachable barrel [21]. To prevent accidental activation, the device only fires when an operator simultaneously presses two switches, which requires both hands. A solenoid drives a metal bar to strike the firing pin against the cartridge (Fig. 2, bottom). In this study we used Remington nail gun cartridges of .27 caliber with power level 5, which in our unpublished study were shown to induce moderate brain injury in the animal model. Under these conditions, the Vandenberg blast device produces a combined blast over/underpressure that is followed by a blunt impact caused by the venting gas jet.

The rat model is the most commonly used model in the TBI research due to the similarities between TBI in humans and rats and knowledge obtained in numerous rat TBI studies. To demonstrate the feasibility of the blast-induced hematoma formation monitoring with this technique, the rat model was best because the blast-induced TBI has been studied in rats by many groups. Blast-induced TBI in large animals is less reproducible and practical and is not well established yet. All experimental procedures were approved by the Institutional Animal Care and Use Committee of the UTMB. Adult, male Sprague-Dawley rats (N = 15, weight = 370–520 g) were anesthetized with 4% isoflurane in an induction chamber, intubated and mechanically ventilated with a mixture of 70:30 air:oxygen and 1.5-3% isoflurane (2-3% during surgery, 1.5-2% during monitoring). A tail artery was cannulated for continuous measurement of arterial blood pressure via a Biopac MP-100 system (Biopac Systems, Inc., Goleta, CA). The instantaneous blood pressure (with a sampling interval of 50 ms) was acquired, displayed and logged by AcqKnowledge 3.9 software running on a Macintosh PC. The current mean arterial pressure (MAP, averaged over the last 30 sec) was periodically calculated and entered in the AcqKnowledge journal. Body temperature was monitored with a rectal probe. The dorsal surface of the head was shaved, and any remaining hair removed with Nair lotion, to minimize the attenuation of the blast wave, the laser pulses, and the acoustic signals during oxygenation monitoring.

The optoacoustic probe was placed on the dorsal midline of the head with a layer of ultrasound gel for acoustic coupling and aligned for maximum SSS signal. The SSS oxygenation (SSS) was measured continuously and in real time for ~10 min to provide a baseline. Then the optoacoustic probe was removed and the rat was transferred onto a ~5 cm thick foam pad to minimize tertiary blast injuries. Using high-speed video recordings, we had previously confirmed that the force of the blast presses the rat into the foam pad. To block both debris (e.g. unburned powder) and heat from reaching the animal, a ~1.5 mm thick silicone rubber pad was placed on the head over a layer of ultrasound gel (which conducted the blast wave). Earplugs were inserted to protect the eardrums. The rat was positioned under the Vandenberg device, with the opening of the barrel 15 mm above the protective pad and directly over the right hemisphere of the brain. A single blast was fired, the rat was returned to its original position, and optoacoustic monitoring was resumed as quickly as practical (typically within 5 min). The OA probe was placed as close as possible to its pre-blast location, while maximizing the SSS signal. MAP monitoring was continued during the blast. Whenever the MAP began to drop rapidly, or if it fell below 60 mmHg, the OA probe was lifted, proper ventilation was verified, and the arterial catheter was flushed with saline. To maintain body temperature in the 37-38 °C range, we placed a warm-water blanket under the rat and a 100 W halogen lamp above it; the lamp was raised or lowered as needed.

The blast injury often was followed by the formation of a hematoma (intracranial, extracranial, or both) over the right hemisphere, close to the SSS, which was revealed upon the completion of the experiment and animal euthanasia. Even though the probe was aligned over the SSS, in many cases it also picked up an optoacoustic signal from the hematoma. This allowed simultaneous

Fig. 2. Vandenberg blast device: external appearance (top); internal structure (bottom).

Fig. 3. Optoacoustic signals from a rat’s head before and after blast injury, acquired at a wavelength of 800 nm.
measurement of blood oxygenation in the SSS and in the hematoma, as well as monitoring the progression of the hematoma.

After 60-80 minutes of monitoring, the isoflurane anesthesia was increased to 4% for ~10 min in preparation for euthanasia. Then the rat was decapitated and the skull was opened for gross examination. The brain was immediately transferred to dry ice for further analysis.

3. Results & discussion

Fig. 3 shows the optoacoustic signals obtained from the intact head of one of the rats before the blast and at several time intervals after the blast, using the same probe position (except before the blast). Each of these waveforms was obtained by averaging 50 consecutive signals induced by 800 nm laser pulses. During the experiment, the operator selected the peaks of interest in the displayed signal, and their positions and amplitudes were automatically calculated and displayed in real time. The pre-blast signal contains only two distinct peaks, from the skin and the SSS. The very first peak was the signal from skin (at t = 0 µs) because the probe was in contact with skin and t = 0 µs corresponded the probe-skin interface. The SSS signal has a well-defined bipolar shape, with an initial maximum pressure (SSS+) followed by a minimum (SSS−). After the blast, a peak from the intracranial hematoma was immediately apparent and increased over time.

About 10 min after the blast, another peak appeared near the skin, indicating an extracranial (subcutaneous) hematoma. The ultrasonic time-of-flight measurements (from the skin peak to each of the other peaks) were converted into distance measurements using the fact that the speed of sound in most soft tissues is very close to the speed in water. The measured distances correlated well with the expected anatomical features. Also, the SSS signal was maximized when the probe was placed on the midline of the head, which is the actual location of the SSS.

Both hematoma peaks increased in amplitude over time as the volume of blood in the hematomas increased. Higher vascularization and blood flow in brain compared to that in skin explain faster formation of intracranial hematomas compared to extracranial hematomas. The amplitude of the SSS peak declined over time because more light was absorbed by the intervening hematomas.

Table 1

| Layer             | d(start), mm | d(end), mm | Δd, mm | ΔS, % | ΔS/Δt, %/min |
|-------------------|--------------|------------|--------|-------|---------------|
| Pre-blast total   | 1.809        | 1.852      | 0.043  | 02.4  | 0.23          |
| Post-blast total  | 2.128        | 2.459      | 0.331  | 15.6  | 0.93          |
| Scalp             | 0.480        | 0.667      | 0.187  | 39.0  | 2.34          |
| Skull             | 0.905        | 0.955      | 0.050  | 05.5  | 0.33          |
| Intracranial      | 0.743        | 0.837      | 0.094  | 12.7  | 0.76          |

Fig. 4. Depths of the optoacoustic peaks, before and after blast, and definitions of swelling parameters.

Fig. 5. MAP and oxygenation of the SSS and hematomas, before and after blast.
Absorption by the extracranial hematoma also reduced the amount of light scattered back to the skin, resulting in lower skin signal amplitude. Moreover, it is highly likely that blast-induced trauma may result in vasoconstriction and decreased blood flow in skin, further reducing the skin signal due to lower hemoglobin content. As the blast-induced swelling progressed, the distance from the skin to the hematoma and SSS peaks increased steadily (Fig. 4). Here we divide the tissue into 3 hypothetical layers, separated by the peaks in the optoacoustic signal. Layer “skull” extends from the skin peak to the extracranial hematoma, layer “scalp”–from extracranial hematoma to SSS. Let $d_{sk}(t)$ denote the thickness of layer X, and $d_{total}$–the total thickness of all layers (the distance from the skin to the SSS). To calculate $d_{sk}$, we multiply the time difference between the optoacoustic peaks by 1.5 mm/μs, the speed of sound in soft tissue. (This will underestimate the skull thickness, but will not affect the % swelling result.) Let $t_{p}$ be the time (in minutes) of the start of the monitoring period, and $t_{end}$–the end of the period. Fig. 4 defines $S_{sk}(t)$ (the percent swelling in layer X, relative to initial thickness), $\Delta d_{sk}$ (the absolute change in thickness from start to end of monitoring), and $\Delta S_{sk}$ (total % swelling). The average rate of swelling (in %/min) is given by $\Delta S_{sk}/\Delta t$. All results are presented in Table 1. We can see that the rate of swelling before the blast was within measurement error (considering only the total tissue thickness since there were no hematomas yet), but after the blast it was significant. As expected, the skull layer experienced almost no swelling. The intracranial layer did not swell as much as the scalp, probably because it was constrained by the cranium.

Fig. 5 shows the blood oxygenation measured in the SSS, extracranial hematoma, and extracranial hematoma of the above mentioned rat. Cerebral blood flow, and hence SSS oxygenation, increases with cerebral perfusion pressure (CPP = MAP – ICP), where ICP is intracranial pressure. Before the blast, the ICP was constant but MAP decreased quickly before stabilizing, leading to a gradual decrease in SSS oxygenation. It should be noted that even before the blast MAP was often unstable in the rats due to anesthesia. After the blast, the SSS blood oxygenation gradually decreased from ~70% to ~60%. While the MAP increased gradually, the ICP increased greatly due to hematoma formation, leading to lower CPP and slowly declining SSS oxygenation. The oxygenation of both hematomas started low and rapidly increased (from ~40% to 68% in the intracranial hematoma, and from ~40% to 83% in the extracranial hematoma). The decrease of SSS blood oxygenation was due to the blast-induced brain injury resulting in insufficient cerebral blood flow. The higher increase of oxygenation in the extracranial hematoma was due to low oxygen consumption in skin tissue compared to that in brain tissue. In general, skin tissues have higher oxygenation compared to that of other tissues (including brain tissues) because of the lower oxygen consumption. We speculate that the hematoma oxygenation was low (~40%) immediately after the blast because: 1) the fresh hematomas consisted primarily of venous blood (~70% of cerebral blood is venous) and 2) the blast may result in rapid and transient vasoconstriction within a few minutes after blast. Over time, the pressure gradient between arterial and venous blood resulted in leakage of more highly saturated arterial and capillary blood into the hematoma. The strong correlation between the MAP and the oxygenation of each hematoma supports this hypothesis.

After blast MAP measurements can be resumed quicker (in 2 minutes) than the optoacoustic measurements (in 5 minutes) due to limitations in the animal study. Most likely, that within a few minutes after the blast the SSS blood oxygenation closely followed the MAP (namely, sharply decreased and then quickly recover). The short transition period was followed by the steady increase in the MAP, while the SSS blood oxygenation decreased due to intracranial pressure rise.

The presence of extracranial hematoma was visually confirmed after the scalp over the blast site was removed (Fig. 6, left). On the excised brain, the intracranial hematoma was easily identified (Fig. 6, right). Similar hematomas were observed in five other rats.

The experiments were designed to induce moderate traumatic brain injury in rats. In 6 out of 15 rats the blasts resulted in hematoma formation. After blasts, the additional peaks were detected in all the rats with hematomas and these peaks had similar trends in these animals.

4. Conclusions

In this pre-clinical study, we have demonstrated the feasibility of optoacoustic detection of blast-induced hematomas in a small animal model. Detection of hematomas through an adult human skull requires a separate clinical study, which is beyond the scope of the present work but is planned for the near future. However, we have already measured SSS oxygenation in adult human volunteers [17] and TBI patients [18] through intact scalp and skull. The penetration depth of our technique in these cases is ~2 cm (up to 1 cm scalp and 1 cm skull). In real clinical settings an optoacoustic array may be used to provide real-time, fast data acquisition for hematoma detection.

Our observations have clinical diagnostic significance. Not only will the optoacoustic device detect intracranial hematomas, it also will provide valuable information regarding hemoglobin saturation in the hematoma. Most subdural hematomas result from venous bleeding and would be expected to have relatively low hemoglobin saturation. In contrast, many epidural hematomas result from arterial disruption and would be expected to be associated with hemoglobin saturation similar to arterial saturation. Diagnosis of hematomas with an apparent arterial source could prompt more
rapid intervention because arterial bleeding may be more rapidly progressive.

Conflicts of interest statement

The authors declare that there are no conflicts of interest.

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