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Blast-induced neurotrauma in whales

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Abstract

A majority of investigations on primary blast injuries have focused on gas-containing organs, while the likelihood of blast-induced neurotrauma remains underrated. In Norway minke whales (*Balaenoptera acutorostrata*) are hunted using small fishing boats rigged with harpoon guns, which fire harpoons tipped with a grenade containing a charge of 30-g penthrite. The grenade detonates 60–70 cm inside the animal. The present study was undertaken to characterize the neuropathological changes caused by the penthrite blast and evaluate its role in the loss of consciousness and death in hunted whales. The study included 37 minke whales that were examined shipboard. The brains were later subjected to gross and light microscopy examination. The results showed that intra-body detonation of the grenade in near vicinity of the brain resulted in trauma similar to severe traumatic brain injury associated with a direct blow to the head. Detonation in more distant areas of the body resulted in injuries resembling acceleration-induced diffuse traumatic brain injury. The authors conclude that even if several vital organs were fatally injured in most whales, the neurotrauma induced by the blast-generated pressure waves were the primary cause for the immediate or very rapid loss of consciousness and death.

Keywords: Blast injury; Brain; Penthrite; Explosives; Traumatic brain injury; Minke whale; *Balaenoptera acutorostrata*; *Cetacea*

1. Introduction

'Blast injury' refers to the events that occur when a living body is exposed to a blast of any origin. The injuries may be caused by shock wave-induced pressure differentials (primary blast injury), by fragments that are propelled into the body (secondary blast injury), and/or by bodily displacement (tertiary blast injury) (Phillips and Richmond, 1990; Stuhmiller et al., 1990). A vast majority of the reports on primary blast injuries have been focusing on damages in gas-containing organ systems (Phillips, 1986; Sharpnack et al., 1990; Rosen and Pillgram-Larsen, 1992; Cernak et al., 1996; Ylverton, 1996; Elsayed, 1997; Mayorga, 1997; Irwin et al., 1997). However, the likelihood of blast-induced neurotrauma is still underrated. This is somewhat surprising as observations of traumatic brain injury (TBI) in blast exposed combatants were reported already during World War I (Mott, 1916), generally known as 'shell shock', where the causalities characteristically had no marks of external violence but nevertheless sustained an instantaneous loss of consciousness and/or post-traumatic persistence anxiety and confusion (Mott, 1917; Myers, 1940; Fulton, 1942). During and after World War II, the term 'blast concussion' began to appear more frequently. Cerebral edema, contusions and lacerations, skull fractures, as well as hemorrhages in the meninges, brain substance, nerve roots and ventricles were reported in a large number of combatants. The clinical states varied from immediate unconsciousness and death to persistent confusion, intractable headache, amnesia and anxiety (Fulton, 1942; Abbott et al., 1943; Ascroft, 1943; Pollock, 1943; Rogers, 1945; Aita and Kerman, 1946; Wood and Sweetzer, 1946; Fabing, 1947; Cramer et al., 1949; Jacobs and Berg, 1968; Murthy et al., 1979). Such results were also reported from experimental and observational studies on animals (Stewart et al., 1941; Krohn et al., 1942). However, there was much controversy whether 'blast concussion' was a definite pathological entity causing organic brain damage or simply a state of psychogenic war neurosis, and the

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controversy concerning blast-induced neurotrauma has continued up till today (Denny-Brown, 1943; Clemedson and Hultman, 1954; Clemedson, 1956; Phillips, 1986; Demann and Leisman, 1990; Sharpnack et al., 1990; Mayorga, 1997; Guy et al., 1998). The extensive increase in the worldwide use of explosive munitions (Coupland and Meddings, 1999) has again raised this question and initiated new research on the subject. Experimental studies on animals (rats, pigs, rabbits and dogs) have demonstrated that exposure to blast waves and overpressure energy induces changes in neuronal as well as non-neuronal cells in the central nervous system (CNS) (Suneson et al., 1987, 1988, 1989, 1990; Kaur et al., 1995, 1996, 1997a,b; Cernak et al., 1999; Axelsson et al., 2000; Guy et al., 2000; Säljö et al., 2000; Cernak et al., 2001a; Li et al., 2001; Säljö et al., 2001, 2002a,b). Such findings have also recently been reported in man (Trudeau et al., 1998; Sylvia et al., 2001). Cernak et al. (2001b) have indicated that blast-induced neurotrauma in rats resemble diffuse traumatic brain injury (dTBI). However, the patho-physiology of the brain response and the injury mechanisms causing neurotrauma after blasts are still poorly characterized.

Annually, Norwegian fishermen are harvesting about 500 minke whales (*Balaenoptera acutorostrata*) from the Northeast Atlantic stock, which consists of about 112 000 individuals (Anonymous, 1996). The minke whale is a small baleen whale that in the North Atlantic may achieve a length of about 10 m and a weight of approximately 8500 kg (Jonsgård, 1992). The hunt is carried out from small fishing boats (average length about 20 m) rigged for whaling in the hunting season. They carry harpoon guns of caliber 50 or 60 mm, which fire harpoons, weighing between 12–18 kg, tipped with a grenade containing a charge of 30 g penthrite (pentaerythritol tetranitrate or PETN) (Øen, 1995a,b, 1999). Penthrite is a supersonic explosive that detonates at a speed of 6500–8400 m/s, producing a gas volume of more than 6000 times the original mass (Baytos, 1980; Dobratz, 1981). The current Norwegian grenade ('Whalegrenade-99') is 23 cm long and weighs 1750 g. The pressed penthrite charge, which is formed like a ring and placed around the grenade body in its housing made of poly ethylene and protected from humidity by a thin (0.7 mm) aluminum tube, is set off by a mechanical trigger system that ignites the grenade when it has penetrated to a depth of 60–70 cm inside the animal. As in many other forms of large game hunting, the gunner is usually aiming at the thorax of the whale. Animals that survive the first shot, or show movements in the flippers or tail, are either re-shot with a new grenade or shot in the brain with a heavy caliber rifle (minimum 9.3 mm), using round-nosed, full-jacketed bullets. In the 2001 hunting season 79.8% of the animals were recorded instantaneously dead (Øen, 2002). The dead whale is hauled transversely across the deck, bled and butchered (flensed) and the meat and blubber are stored in ice in the hold before being brought back to port.

Due to the size of the whale relative to the boat size it is difficult to carry out extensive post-mortem examinations on whales caught during regular hunting. However, during a research program on marine mammals in Norwegian waters in the early 1990s (Haug et al., 1998) such examinations were conducted on 19 minke whales caught using grenades with 22 g penthrite (Øen, 1995a). These demonstrated that the pathological changes in internal organs resembled those described from man and experimental animals exposed to blast overpressure (Øen, 1995a,b,c). As fresh brains were difficult to remove without damaging the brain tissue, brains from two whales that stopped moving and instantly sank after the detonation and brains from two other whales which moved their tails uncoordinatedly for several minutes, were fixed in situ and later examined for pathological changes. In all four brains, both hemorrhages in the meninges, brainstem, ventricles and the white matter of cerebellum were demonstrated, and it was concluded that these four whales had lost consciousness and died instantaneously or very quickly (Øen, 1995a; Øen and Mørk, 1999). The question then arose, and the aim of the present study has been to answer: depending on where the grenade detonates which pathological changes do the penthrite blast cause in the CNS and what is the role of blast-induced neurotrauma in loss of consciousness and death of hunted minke whales?

2. Materials and methods

Sampling took place on two different Norwegian whaling boats during the hunting seasons of 1998, 1999 and 2000. Both boats were using 60-mm harpoon canons and 'Whalegrenade-99'. The study includes 37 whales killed by a single penthrite grenade detonation.

In the field, the animals' reaction to the detonation were observed and recorded. During the flensing, the whales were examined and data including body length, harpoon and grenade performance and organ and tissue damage were collected. The brains were preserved as a whole by fixation in situ, and this method is reported in detail in Knudsen et al. (2002). After excision, the whole brain resting in its dural sack was put into a perforated plastic bag tagged with an identification number (in running order) and stored in 30-l containers with 8% neutral formalin for at least 2 months.

Before further examination all brains were randomized and given a new identification number, so further analyses were conducted blind. The code was not opened until all samples were analyzed. After weighing the brain without the dura mater and gross external examination, the cerebral hemispheres were separated from the brainstem and cerebellum by transecting the caudal midbrain, and cut into 1-cm thick coronal slices. The cerebellum was detached from the brainstem and the cerebellar hemispheres were separated in the vermis. The brainstem and first portion of the cervical spinal cord were sliced transversely into 5-mm thick disks. Altogether, about 40 specimens for histological examination including two tissue blocks from dura; 14 from cerebrum; three from cerebellum; and the whole brainstem and first portion of cervical spinal cord (20–25 tissue blocks) were collected from the same anatomical sites in all brains. Additional tissue blocks were sampled when pathological findings were evident in areas that were not covered by the routine sections. The blocks were embedded in paraffin wax, sectioned at 5 μm with a microtome, and stained with hematoxylin/eosin (H&E) after standard protocols (Culling et al., 1985). Sections from the brainstem and spinal cord from the first three brains analyzed were additionally stained with Luxol-fast-blue/Cresyl Violet (LFB/CV) for myelin (Culling et al., 1985). Photomicrographs of the micro slides were taken and mounted using a Zeiss Axioscop (Carl Zeiss Vision GmbH, Hallbergmoos, Germany), a Sony Power HAD Camera Adapter (Sony Corporation, Tokyo, Japan), and Matrox Imaging Intellicam (Matrox Electronic System Ltd, Dorval, Canada). Adobe Photoshop 6.0 (Adobe System Inc., San Jose, USA), Corel Photo-Paint™ Select 5.0 (Corel Corporation, Ottawa, Canada) and Imaging for Windows® (Eastman Software Inc./Kodak, Billerica, USA) were used for digital processing of the illustrations (adjusting of contrast, brightness and sharpness).

3. Results

Brain damage attributable to the grenade detonation was evident in 35 of the 37 brains. The neuropathological alterations varied from very severe brain tissue laceration with concomitant skull fractures and regular decapitation, massive gross evident bleedings in meninges and brain substance, to histologically evident intracerebral hemorrhages in central brain areas. A summary of the results is presented in Table 1, where the whales have been grouped and ranged according to extent and severity of trauma in the skull and brain. The whales in Group I have the most violent damages.

Complete penetration of the body by the harpoon after the grenade had detonated was seen in 27 of the whales. All grenades functioned. The letters (A–E) in the right column of Table 1 refer to the region in the whales in which the grenade detonated, and these correlates with the letters given in Fig. 1. The shipboard examination of the whales revealed that the detonation in most cases had caused severe and fatal damage to several organs. At detonation in the thoracic cavity (region C) commonly the heart, lungs and major vessels (the aorta, the pulmonary veins and the vena cava) were injured. These injuries consisted of violent tearing and laceration, substantial hemorrhages and in many cases total destruction of the organs. Detonations in the interface between thorax and abdomen (region D and E) caused damage also to cranial abdominal organs (liver and stomach). In cases were the center of the detonation occurred in the lower back and damaged the spinal cord either in region C, D or E, additionally damages to thoracic and abdominal organs and vessels were registered in most cases. Detonations in muscles resulted in a jelly-like consistency in the vicinity of the detonation, and the affected musculature were transformed into a granular, pulped mass lacking normal tissue structure.

3.1. Group I ($n = 2$)

The two brains in Group I were almost totally damaged by the grenade detonation. They were lacerated to a degree that almost no normal brain structures and architecture remained, and they could not be fixed for further analysis. In both cases the grenade had detonated in the neck muscle and less than 30 cm behind the skull creating large craters and cracks in the skull bones.

3.2. Group II ($n = 5$)

All whales in Group II had several large diastatic and displaced skull fractures (5–20 cm) and damaged ear bones (*bulla tympani*). The grenade had detonated less than 1 m from the brain in all whales. Two whales were virtually decapitated after detonation in the upper cervical spine at the level of the 1st–2nd cervical vertebra.

The gross examination of the five brains revealed that the architecture was mainly intact, but very massive epidural, dural, and subdural bleedings, extensive subarachnoid hemorrhage (SAH) and cortical surface hemorrhages were covering large parts of the brains. On the slice surfaces, multiple punctuate intracerebral hemorrhages were seen in multiple brain regions as well as intraventricular hemorrhage (IVH) in the midbrain and brainstem. These findings were confirmed by microscopy.

3.3. Group III ($n = 11$)

The 11 brains in Group III showed massive epidural and dural bleedings, extensive acute subdural hematomas (ASDH) and substantial SAH. The ASDH and SAH were most extensive under the brainstem and cervical spinal cord (Fig. 2). The prepontine and peduncular cisterns were commonly filled with blood and the nerve roots were embedded in clots. In seven of

Table 1
Blast-induced brain and head injury in minke whales associated with the detonation of the penthrite grenade

Group	No. of whales (N)	Gross	Microscopy	Detonation region A–E (n/N)[a]
I	2	Large skull fractures and almost total disintegration of gross architecture	Histology not possible	A (2/2)
II	5	Multiple skull fractures. Massive intracerebral haemorrhages	Multiple haemorrhages in most brain regions	A (1/5), B (4/5)
III	11	Massive intracerebral haemorrhages, especially under the base of the brain	Multiple haemorrhages in several brain regions	C (5/11), D (3/11), E (3/11)
IV	17	Minor haemorrhages or no findings	Multiple haemorrhages in central brain regions (especially brainstem and spinal cord)	B (1/17), C (12/17), D (3/17), E (2/17)
V	2	Small localized menigeal haemorrhage on top of cerebrum	Inconclusive findings	C, D
Total	37			A (3/37), B (5/37), C (17/37), D (7/37), E (5/37)

The whales have been grouped and ranged according to extent of neurotrauma after gross and microscopy examination. The letters A–E in the last column correlates with the letters given in Fig. 1 and refers to the region in the whales in which the grenade detonated.
[a] n/N, Number of whales (n) hit in this region in relation to the total number of whales in the group (N).

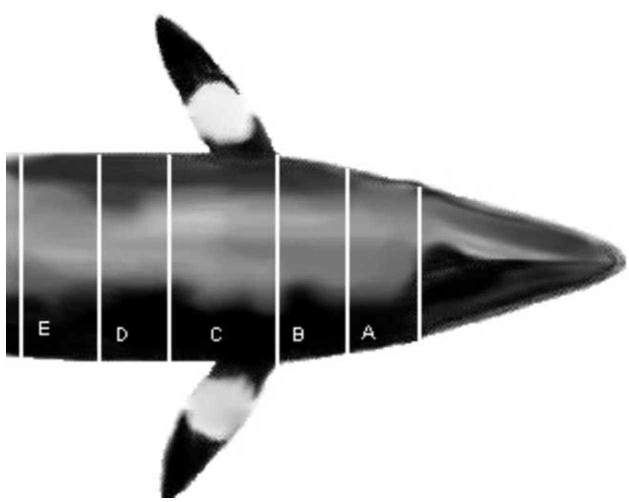

Fig. 1. A drawing of the cranial part of a minke whale viewed dorsally from the upper abdomen to the tip of rostrum. The letters (A–E) indicates the different detonation regions. The brain is situated in the middle of zone A. The diaphragm is situated between region D and E.

the brains petechial hemorrhages were visible on the slice surfaces, especially in thalamus, brainstem and spinal cord at gross examination, and IVH was evident in the 3rd and 4th ventricle in four of the seven brains.

During microscopy it was found that the hemorrhages in thalamus, pons, brainstem and spinal cord were especially evident around the aqueduct, under the 4th ventricle and in the gray substance around the central canal (Fig. 3A–C). These acute bleedings were in some cases symmetrically bilateral. In nine of the brains, additional deep white matter bleedings in cerebrum, cerebellar vermis, peduncles and spinal cord were detected, as well as hemorrhages in the cranial nerves (Fig. 3D–F). Cortical hemorrhages were presented in rostral and ventral parts of cerebrum and in different parts of cerebellum in five of these, and a generalized engorgement of blood vessels throughout the brain were seen in two.

The detonation additionally caused severe damage to the heart, lungs, major vessels and/or cranial abdominal organs in all animals.

3.4. Group IV ($n = 17$)

Of the 17 whales in this group, 12 had a few (1–4) and minor (1–5 cm) localized menigeal bleedings on cerebrum and/or cerebellum. In six of these also sparse petechial hemorrhages were seen in the brainstem and spinal cord at gross examination. In the remaining five whales in Group IV no macroscopic changes were registered. Microscopy, however, revealed similar changes in all 17 whales, appearing as hemorrhages in the midbrain, brainstem and spinal cord. In 15 these acute bleedings were seen in multiple central brain areas, while in two they were found mainly in the medullar–spinal junction in the obex area. In conformity with the findings in Group III, the hemorrhages were predominantly located in the peri-aqueductal gray matter, under the base of the 4th ventricle, and in the central gray matter of the cervical spinal cord (Fig. 3A–C). Deep white matter hemorrhages especially in corpus callosum, hippocampus, thalamus, cerebellar vermis and peduncles were found in all, but two brains (Fig. 3D and F). A generalized engorgement of blood vessels and perivascular spaces were seen in five brains. Cortical hemorrhages were revealed both in rostral and ventrocaudal parts of cerebrum in five of the whales.

The detonation had occurred in region B, C, D and E (Table 1), and in 15 whales severe damage to heart, lungs and major vessels were recorded. In two whales the grenade detonated in muscles (in region B and C),

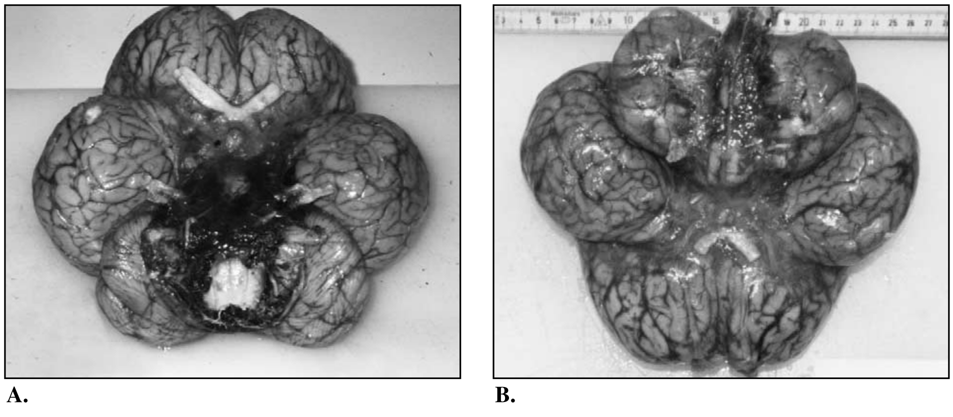

Fig. 2. (A) Brain from a minke whale in Group III viewed ventrally. The brainstem and spinal cord has been transected. There are massive subarachnoid under the base of the brain from chiasma and backwards. (B) Brain from another minke whale in Group III viewed ventrally. There are subarachnoid hemorrhages under the cervical spinal cord, brainstem and cerebellum. Scale bar for A = 3.1 cm, B = 3.0 cm.

and in one of these (region B) the ear bones were damaged.

3.5. Group V ($n = 2$)

In both whales the grenade had detonated in the lungs. The gross examination of the brains revealed a single localized dural and subdural bleeding on cerebrum, as well as hypostasis in rete mirabile, dura and pia-arachnoid ventrally, especially under the brainstem and around the cranial nerves. One brain was compressed laterally on the left cerebral hemisphere, which made evaluation of any possible asymmetry difficult. At microscopy a generalized vacuolation of the brain substance appeared in several brain regions as well as enlargement of the perivascular spaces. A few hemorrhages were found near the aqueduct in thalamus, under the 4th ventricle in the brainstem and in white matter ventrally in the cerebellar vermis.

3.6. Myelin staining

The LFB/CV staining for myelin, which was performed on three brains, one from Group II, IV and V, respectively, did not reveal any additional changes beyond those registered by H&E.

3.7. Body length and brain weight

The mean body length of the whales was 764 cm (95% KI: 736–791; Range: 485–885; $n = 37$). The mean fixed brain weight (without dura mater) was 2775 g (S.D.: 212, Range: 2322–3265 g; $n = 34$). Two of the brains were so lacerated that they could not be weighed whilst technical failure during gross examination prevented measurement of one brain.

4. Discussion

The results demonstrate that intra-body detonation of 30 g penthrite causes severe and fatal neurotrauma in minke whales. Not unexpectedly, the extent of damage to the brain and adjacent tissue increases the closer to the brain the detonation occurs. However, the study also demonstrates that detonations even as far back as the rostral abdomen may cause fatal neurotrauma. Pollock (1943) described three spherical zones about the point of a detonation: (1) the 'zone of brisance', in which everything is disintegrated and all life is destroyed, (2) a more distant zone where the shock wave still has great traumatic effect, and death without external evidence of injury occurs with capillary hemorrhages in the nervous system, and (3) a remote zone in which the shock wave loses its force and becomes merely a sound wave. Even though this description relates to an extra-body detonation, it appears to be applicable also to characterize the injury pattern of neurotrauma caused by the intra-body detonation of penthrite in the present study. In all cases where skull fractures were registered (Group I and II) the detonation occurred less than 1 m from the brain, i.e., in region A and B. The brains of Group I were severely lacerated, while the brain damage in Group II resembled the TBI observed after a direct blow or impact to the head (Demann and Leisman, 1990). It is reasonable to conclude, that for these animals the brain was in, or just beyond, the 'zone of brisance' of the detonation. There was, however, one case were detonation in region B (Group IV) did not cause skull fracture, except for the ear bones. This whale was hit obliquely from behind and the harpoon went in and out on the same side causing the grenade to detonate in muscular tissue at the exit wound in front of one of the flippers, and some of the energy from the detonation was probably lost to the water.

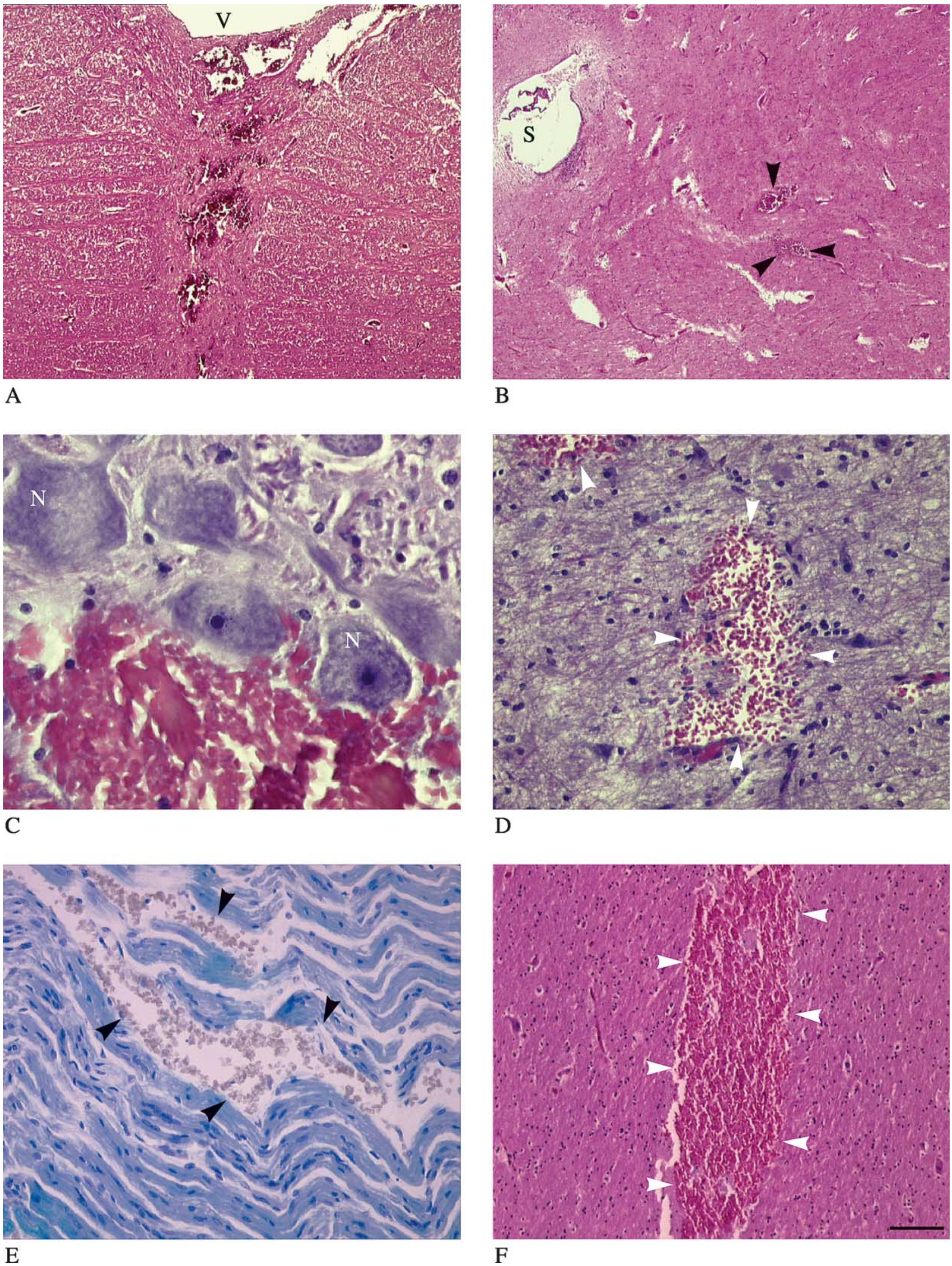

Fig. 3

When the distance between the detonation site and the brain increased (region C–E) the most striking features were ASDH and SAH under the base of cerebellum, midbrain, brainstem and spinal cord (Group III), histological evident hemorrhages sub-ependymal in the same brain areas (Group III and IV) and deep white matter injuries (Group III and IV). The acute and massive ASDH and SAH will probably, as in humans, have a severe effect and result in high mortality (Crooks, 1991; Zhao et al., 1999; Tomita et al., 2000), especially with the extensive hemorrhages underneath the base of the pons, cerebellum and medulla oblongata (Boström and Helander, 1986; Boström et al., 1992). The authors therefore consider it highly likely that these whales immediately lost consciousness and subsequently died of the neurotrauma. The presence of sub-ependymal and white matter hemorrhage is concurrent with recent experimental studies on blast-induced neurotrauma in rats (Kaur et al., 1996; Cernak et al., 2001b). These damages share many similarities with the typical findings at deceleration and acceleration-induced dTBI (De Girolani et al., 1994). Primary dTBI occurs from shearing or impulsive forces, and has in humans commonly been associated with instantaneous loss of consciousness without a lucid interval and the vegetative state (Boström and Helander, 1986; McIntosh et al., 1996; Smith et al., 2000). We consider it likely that such damages will have the same effect also in whales. Morphologically, dTBI is characterized by few findings at gross examination, except for the presence of petechial hemorrhages in the white matter of cerebrum, in the basal ganglia, in the mesencephalon, in the pontine region, around the aqueduct, in the cerebellar peduncles, and rostral brainstem, and it is common that many more hemorrhages are seen microscopically than macroscopically (Boström and Helander, 1986; De Girolani et al., 1994; Ng et al., 1994). Another typical feature of dTBI is diffuse axonal injury (DAI), characterized by rupture of axons in the cerebral white matter, in the brainstem and in cerebellum (McIntosh et al., 1996). Such axonal damage was not demonstrated in this study. However, DAI may be present, but not detectable, even with refined staining techniques, if the survival is less than 1–2 h (Boström and Helander, 1986; McIntosh et al., 1996; Meythaler et al., 2001). Even if none of the whales in this study survived for a long time, LFB/CV staining was applied to detect possible myelin globoids and axonal retraction balls in three brains, but no DAI was visible. However, the predilection sites for the intracerebral hemorrhages are strong indicators of a dTBI-like condition and DAI may be present, but not easily identifiable, due to the extremely short survival time in the whales.

There have been several theories regarding the mechanism of direct organic brain damage following primary extra-body blast exposure, especially on how shock waves may travel and impact the CNS. One is that shock waves are transmitted to the brain via viscera, muscles, bones, blood vessels and cerebrospinal fluid (CSF) resulting in increased pressure on the brain cells and capillaries, especially in the brainstem (Mott, 1916; Stewart et al., 1941; Cramer et al., 1949; Cernak et al., 2001b). It has also been considered that the blast may cause compression of thorax and abdomen resulting in intense venous backpressure to the brain, which leads to intracerebral hemorrhages (Rogers, 1945; Wood and Sweetzer, 1946). Clemedson (1956), though, suggested that the blast may create an increased pressure in the body except for in the craniovertabral space that is protected by a strong bony capsule, and hence the blood from the largest vessels may literally be squeezed into this low-pressure area. All these theories relates to shock wave-induced damage after an extra-body detonation. The detonation of penthrite intra-bodily results in a very rapid gas expansion, which probably gives rise to oscillating pressure waves that propagates supersonically in all directions in the body. When these reach the CNS it is likely that they generate high-pressure differentials within the blood vessels and a very rapid change in the intracranial pressure, resulting in rupture of the blood vessels in the meninges and brain tissue. The waves most likely choose the road of less resistance and therefore travel via fluids (CSF and blood), rather than through the tissue. This is supported by the presence of hemorrhages sub-ependymal, which was evident especially when the detonation occurred more peripherally from the brain. The reason why many more hemorrhages were seen in the brains during microscopy than at gross examination, may be due to the fact that the capillaries and post-capillary venules in the brain have less supporting connective tissue compared to the rest of the vascular system within the brain, and are therefore more likely to be damaged by pressure waves (Suneson et al., 1987). Other mechanisms may also explain the intracerebral hemorrhages in the whales, including rapid blast-induced acceleration of the torso

Fig. 3. Montages of microphotographs showing representative intracerebral hemorrhages in Group III and IV using H&E and LFB-CV staining techniques. (A) Multiple hemorrhages centrally under the 4th ventricle (V) in the upper brainstem (H&E). (B) Acute hemorrhages (arrows) near the central canal (S) in the medullary-spinal junction (H&E). (C) Extravasal erythrocyte accumulation in close vicinity of motor neurons in a nucleus just below the 4th ventricle in obex (H&E). (D) Hemorrhages (arrows) in the cerebellar peduncle in the mid brainstem (H&E). (E) Hemorrhage (arrows) in a cranial nerve in the lower brainstem (LFB-CV). (F) Hemorrhage (arrows) in the sub-cortical white matter of the frontal lobe (H&E). Scale bar for A = 0.4 mm; B = 0.4 mm; C = 29.4 μm; D = 55.6 μm; E = 62.5 μm; F = 100 μm.

that cause displacement of the brain resulting in deep intracerebral hemorrhages and tearing of the many bridging veins in the meninges.

The examination of the two brains in Group V only revealed minor changes and intracerebral hemorrhages were very sparse. The hypostasis and compression are probably fixation artifacts (Knudsen et al., 2002). This might also be the case with respect to the histological evident tissue vacuolation and increased perivascular spaces or these maybe indicative of brain edema, or a combination of brain edema and poor fixation as diffusion of fixing fluid in an edematous brain is less effective than in an unaffected one (Esiri, 1996). However, in the field it was observed and recorded that these whales died very rapidly. This course should not have been expected solely from lung damage (Øen, 1995b). Stewart et al. (1941) suggested the blast exposure might cause cessation of nerve cell activity without producing macroscopic or microscopic evidence of injury, i.e., the clinical picture of brain concussion, and experiments by Axelsson et al. (2000) have shown that shock waves may have a direct effect on vital centers in the brainstem or higher controlling units appearing as depression of cortical activity (flattening of the EEG). The rapid loss of consciousness in the two whales in Group V may be explained by concussion although no firm conclusions should be drawn.

The damaging effect of the approximately 15 kg harpoon is obviously also contributing to the killing effect. In general, the lethality of a non-explosive projectile is directly related to the damage it causes to the organs and tissues it hits. The weight, shape, speed and the degree to which the projectile expands when it passes through the tissue are all relevant (Berlin et al., 1976). Øen (1983, 1995a,d) found that in earlier catch when only harpoons without explosives (cold harpoons) were used, both the 50 and 60 mm harpoons did not cause as much tissue damage and effect as might be expected of projectiles of such size and weight probably due to the low velocity the harpoon (<90 m/s). The harpoon only contributes to direct damage of the organ it hits, and the pressure differentials produced when the harpoon strikes the body at some distance from the brain is most likely too low to contribute to any distant effects on the CNS. Also, no grenade fragments of significance are produced at detonation as the only splinters come from the aluminum tube. These are usually small (1–4 mm) and very light and do not travel more than a few centimeters from the detonation site (Øen, 1995a).

5. Conclusions

The present study has demonstrated that depending on the detonation site, 30 g of penthrite inside the body of a minke whale induces neurotrauma similar to either severe TBI associated with a direct impact to the head or acceleration-induced dTBI, in which the cardinal symptoms are immediate loss of consciousness without any lucid interval and very high mortality rate. The detonation also caused severe damages to other vital organs that obviously were fatal for the whales, but in some animals these injuries were not so extensive that an immediate or very rapid death should be expected. The authors therefore consider neurotrauma caused by the blast-generated pressure waves as being the primary cause of the very rapid loss of consciousness.

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