

The time to loss of brain responsiveness following exsanguination in adult cattle

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Introduction: The humane slaughter of meat animals requires that they be rendered immediately unconscious by stunning, and that this should be followed by exsanguination to produce cerebral anoxia and death. Except where cardiac fibrillation is induced, insensibility following stunning is, at least potentially, reversible. Therefore the time needed for brain failure to occur following severance of the circulation to the brain needs to be defined.

Based on the study of the electroencephalogram, Newhook & Blackmore (1982) have reported that insensibility can be delayed for up to 85 sec in calves, and that resurgence of cerebral activity can occur for up to 300 sec. This prolonged activity in the EEG has been attributed to the greater contribution of the vertebral arteries to cerebral circulation in bovines compared to other domestic species, due to supply of the rostral rete by the vertebral artery (Baldwin & Bell 1963).

This study contrasted however with previous, similar work with calves (Nangeroni & Kennet 1963; Schultze et al 1978; Levinger 1979). More recently, Gregory & Wotton (1984) used visual evoked responses (VERs) to determine the onset of brain failure, and found it occurred in 17 sec. Also, the electrocorticogram became isoelectric in 23 sec, and no resurgence of activity was noted thereafter.

A potential limitation of the use of VERs in the context of measuring brain function during slaughter has been suggested by Blackman et al (1986). Since cutting the blood vessels in the neck will, in addition to severing the circulation to the brain, also prevent circulation to the retina of the eye, it could be argued that the loss of VERs is due initially to loss of function in the retina rather than at the cortical level.

This study therefore addressed this possibility by comparing the duration of VERs following exsanguination with that of somatosensory evoked responses (SERs). Also, since previous work has been limited to calves, this study was carried out in adult cattle in order to identify any possible age related effects.

Materials and Methods:

Eight cattle of Friesian x Hereford breed, ranging in weight from 285 to 355 kg (mean 328 kg) were used. They were tranquilised with 1 ml/100kg xylazine (Rompun, 5% solution) and sufficient intravenous sodium thiopentone (100 mg/ml, approx 25 ml) to induce anaesthesia and permit intubation. Anaesthesia was then maintained by mechanical ventilation (Draeger) with oxygen enriched air and halothane throughout the experiment.

Electrocorticograms were recorded using 5mm silver-silver chloride electrodes. The positive and negative electrodes were implanted epidurally over the visual and somatosensory cortices respectively. Two earth electrodes, one of which was used for AC suppression, were implanted midway between the recording electrodes.

The signal was amplified using a 0.1 sec time constant and 70 Hz upper frequency filter (Mingograph EEG 10; Siemens). The signal was then stored on FM tape (TEAC R-71). The evoked responses were averaged and quantified from their excursion distances as described by Gregory & Wotton (1983b) VERs were produced using a flash of light 1m in front of the contralateral eye, at a frequency of 2 flashes/sec. The SER was produced by stimulation of the contralateral tibial nerve, with a stimulation duration of 2 msec and a voltage sufficient to produce a pronounced leg twitch. SER stimulation frequency was generated at a randomly variable frequency of 1 stimulus per 1-1.5 sec. Spontaneous cortical activity was quantified by integration of the positive going signal over 10 sec epochs.

Following a 5 min control recording period, the animals were exsanguinated under anaesthesia by a transverse cut which severed the carotid and jugular vessels and all soft tissue ventral to the spinal cord. Recording was then continued for a further 5 min.

Results:

Spontaneous activity recorded from the cortex fell to less than 10% of control, pre-stick, values in a mean time of 214 sec (S.E. ± 2). Following onset of the isoelectric state, there was no evidence of resurgence of activity for the remaining 5 min of the experiment.

Cortical responsiveness to visual stimuli was abolished in a mean time of 18 sec (S.E. ± 3), at which time VERs were less than 10% of control levels. Both values agree closely with those found by Gregory & Wotton (1984) in calves using a similar experimental protocol. They found that the cortex became isoelectric in a time of 23 sec, and VERs were abolished in 17 sec.

SERs were successfully recorded in 5 animals, simultaneously with the recording of VERs. Following sticking, SERs disappeared in a mean time of 20 sec (S.E. ± 2).

Time to loss of spontaneous cortical activity (sec)	Time to loss of VERs (sec)	Time to loss of SERs (sec)
24 S.E. ± 2 n=8	18 S.E. ± 3 n=8	20 S.E. ± 2 n=5

Conclusion:

In view of the similarity between the times to loss of VERs and SERs following severing the carotid and jugular vessels, it seems unlikely that ischaemia of the retina will have distorted the value for loss of cortical responsiveness to visual stimuli. This conclusion is in accordance with that of Noell & Chinn (1950), who examined the effects of anoxia on the visual system in the rabbits: rapid induction of anoxia was found to abolish cortical VERs minutes before loss of retinal VERs.

The unusual anatomy of the cerebral blood supply in the bovine first described by Baldwin & Bell (1963) and since confirmed by Blackman et al (1986) could in principle provide an explanation for the delay in onset of insensibility reported by Newhook & Blackmore (1982). However, while Blackman et al have demonstrated the continuation of blood flow to the brain via the vertebral arteries after sticking, the question of rate of perfusion must also be considered. Cerebral perfusion must be a minimum of 15 ml/100mg/min in the cortex and 10 ml/100mg/min in subcortical structures to sustain SER and spontaneous activity in primates (Gregory et al., 1979; Branston et al., 1984): this amounts to at least 20% of normal flow rate. Assuming similar figures are valid in bovines, the present results suggest that such flow as reaches the brain via the vertebral is insufficient to sustain functional activity.

A further disparity between these results and those of Newhook and Blackmore involves the different times to loss of spontaneous cortical activity. The use of barbiturate and halothane anaesthesia in the present work may be a contributing factor since such anaesthetics will reduce blood pressure. On the other hand, anaesthesia will also reduce metabolic activity in the brain, and through this, prolong the time to loss of activity (Mayevsky, 1978).

In view of the minimal differences between loss of SERs, VERs and spontaneous cortical activity, the present evidence suggests two things: firstly, the duration of VERs following exsanguination is not defined by retinal function, but rather by cortical ischaemia. Secondly, the vertebral arteries in the bovine do not seem to supply sufficient cerebral blood flow to substantially alter the time to loss of brain function compared to other domestic species.

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