PRE-SLAUGHTER CO_2-ANAESTHESIA FOR SWINE

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ABSTRACT

The effects of one min inhalation of 80% CO_2 in air upon the electrical brain activity (EEG), acid/base balance, oxygen tension and stress hormone release were studied. EEG changes typical for the second stage of anaesthesia developed before the swine exhibited the brief Period of muscular jerks usually occurring during pre-slaughter CO_2-anaesthesia. At this stage pronounced arterial hypercapnia, hypoxemia and acidemia had developed. Any of these distortions per se may have caused the EEG changes.

Determinations of Plasma cortisol, adrenalin and noradrenalin did not provide any direct evidence that the inhalation of CO_2 imposed any emotional strain in addition to that induced by the mere transport of the swine to the immediate pre-exposure situation.

INTRODUCTION

During the present century, ethical aspects on pre-slaughter stunning have actualized the demand for methods of anaesthesia, which are satisfactory from the humanitarian point of view. This debate has stimulated research on the behavioural, neurophysiological and systemic effects of high concentration CO_2-inhalation in swine. The aim of the experiments reported here was to provide answers to the following questions:

A. Are the delayed, transient motor reactions a manifestation of emotional stress?

B. How deep and how long-lasting is the CO_2-induced anaesthesia?

C. To what extent are the swine subjected to hypoxia during conventional pre-slaughter CO_2-anaesthesia?

METHODS

EEG registration

Six Yorkshire swine (b.wt. 40-50 kg) were supplied with recording electrodes placed in different brain regions (frontal neocortex and dorsal hippocampus). With a 48 h interval the swine were exposed twice to 80% CO_2 in air for one min under continuous recording of the EEG.

Blood gases and stress hormones

Twelve Yorkshire swine (b.wt. 40-50 kg) were used. Permanent implantation of venous (n = 12) and arterial (n = 7) catheters was performed 5-7 days before the CO_2-exposure.

Experimental Procedure

A control venous blood sample was taken in the stable before the animal was placed in a cage, resting on wheels, and transferred to the experimental room. Additional venous blood samples were taken about 30 s before the exposure, at approximately 45 s of CO_2-exposure and at 5, respectively 10 min post CO_2. These samples were used for determination of cortisol-, adrenalin- and noradrenalin as well as potassium and calcium concentrations. Venous and arterial blood samples for determination of acid/base parameters were taken immediately before the descent, at approximately 15 and 45 s of exposure and at 1.5 and 10 min post CO_2. For further information concerning methods and material see Forslid 1987, Forslid et al 1986 and Forslid & Augustinsson 1988.

RESULTS

Since the device developed to mimic pre-slaughter CO-anaesthesia (see Forslid 1987) allowed free inspection of the animals, it was possible to relate the transient motor reactions, seen in most pigs during the CO_2-inhalation, to changes occurring in the continuously recorded EEG.

Neocortical EEG

During inhalation of 80% CO_2 in air, a change in the neocortical EEG was seen already after 11-15 s (Fig.1). Then, an increasing amount of intermittent slow waves became dominating 10-15 s later. The muscular jerks

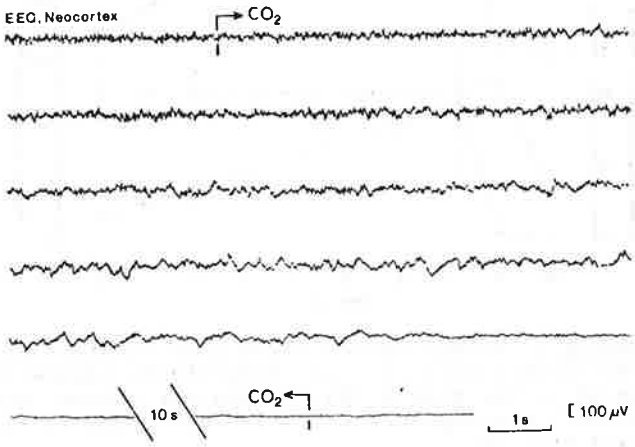

Figure 1. Continuous EEG recording from right frontal neocortex immediately before and during 1 min exposure to approximately 80% CO_2. An intersection of 10 s has been made during the EEG silence (Forslid 1987).

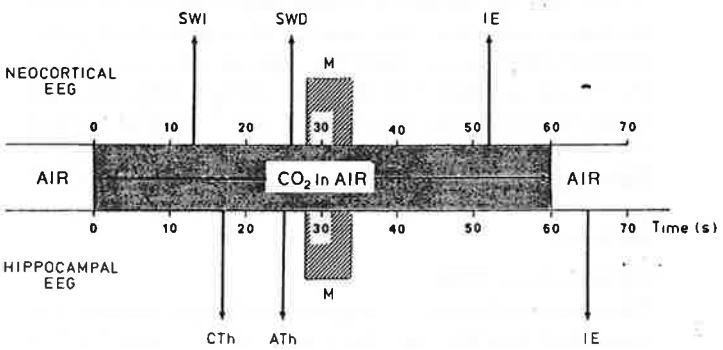

Figure 2. Illustration of the time relations (mean values) between the period of motor reactions (M) exhibited by the swine during 60 s of inhalation (horizontally hatched area) and obvious changes in the simultaneously recorded neocortical, respectively hippocampal EEG. SWI=slow wave increase SWD=slow wave dominance IE=apparent isoelectricity CTh=continuous 5-7 Hz activity ATh=accentuated 5-7 Hz activity.

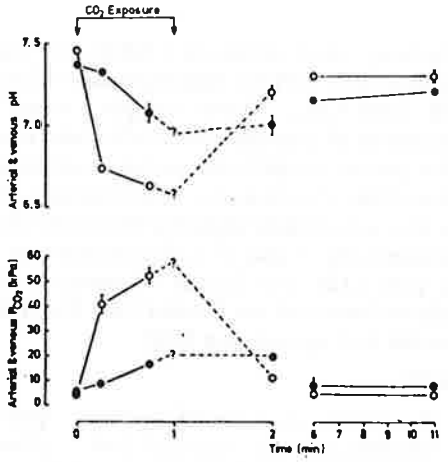

Figure 3. Influence of 1 min inhalation of 80% CO_2 in air on arterial (o——o) and venous (•——•) pH, respectively CO_2 tension (PCO_2) in 7 swine. Vertical bars show SE.

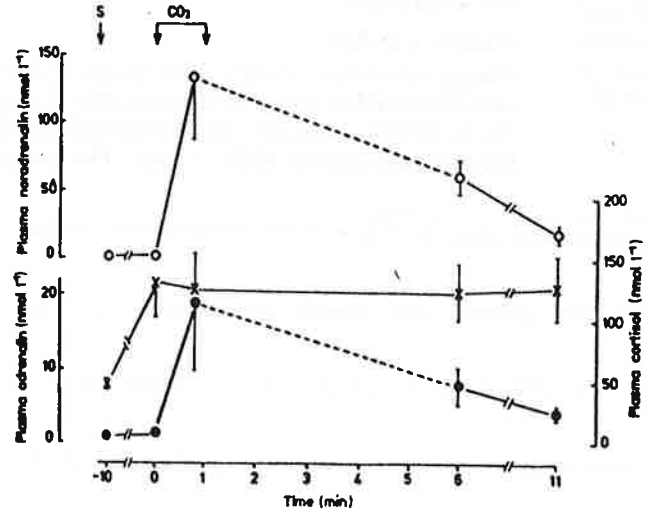

Figure 4. Plasma concentrations of cortisol (x——x) (n=10) and noradrenalin (o——o) respectively adrenalin (•——•) (n=5) in the stable (S) immediately before, during and after 1 min exposure of swine to 80% CO_2 in air. Vertical bars show SE.

started 23-36 s following the onset of CO_2-inhalation and lasted for 2-9 s. Thus, a clearly visible change in the neocortical EEG always preceded the muscular jerks by ± 8 s, and the neocortical slow waves had in all cases become dominating at the onset of these jerks. During the second half of the one min CO_2-exposure, the neocortical electrical activity faded away completely, and an apparent isoelectric ("flat") EEG was recorded around the termination of the CO_2-exposure. This EEG flattening lasted for 19-113 s. A neocortical EEG of pre-CO_2 pattern was not obtained until 3-5 min post exposure.

Hippocampal EEG

The onset of clear-cut changes in the hippocampal EEG coincided roughly with the onset of neocortical EEG changes. Dominating 5-7 Hz activity had always developed when the swine exhibited muscular jerks. After 35-45 s of CO_2-inhalation the amplitude of the hippocampal activity gradually became reduced, and apparent hippocampal isoelectricity developed in 8 out of 11 experiments, lasting for 23-60 s after the end of the CO_2-exposure. The time relation (mean values) between the period of muscle jerks and visible changes in the EEG are illustrated schematically in Fig. 2.

Induction of hypercapnia and acidosis

Already 15 s following the onset of the CO_2-inhalation the arterial CO_2 tension ($PaCO_2$) had risen from an initial value (5.3 kPa) to -40 kPa, and exceeded 50 kPa 30 s later. An extrapolation of the rising curve (Fig. 3 hatched line) indicates that the $PaCO_2$ might have approached 60 kpa at the end of the inhalation period. The hypercapnia became considerably reduced during the first post-inhalation minute (to a $PaCO_2$ -11 kPa) and was, within the subsequent 4 min converted into slight hypercapnia ($PaCO_2$ -4.8 kPa). The steep rise in $PaCO_2$ was accompanied by a fall in arterial pH from 7.45 to 6.7 at 15 s, and a further drop to 6.6 at 45 s of the CO_2-inhalation. A re-elevation to an arterial pH of 7.2 had occurred 1 min post CO_2. However, the animals remained acidotic (arterial pH 7.3) in the presence of hypercapnia at 5 and 10 min after the end of the CO_2-inhalation.

Influence on arterial oxygen tension

Within 15 s of CO_2-inhalation, the arterial oxygen tension (PaO_2) fell from 13.5 to 7.3 kPa, and a further reduction to 3.9 kPa was observed half a minute later. An extrapolation of the oxygen curve indicates that the PaO_2 might have been reduced to < 3 kPa at the termination of the exposure. The PaO_2 became almost normalized during the first post-inhalation minute, and a 1.6 kPa rise above initial level had occurred 4 min later.

Stress hormones

The initial blood sample was taken on the unrestrained swine. The second sample was drawn after the transportation of the swine to the experimental room (Fig. 4). This pre-exposure proceeding induced a 3-fold increase in plasma cortisol concentration (PC), but only minor, statistically non-significant, rises in plasma adrenalin (A) respectively noradrenalin (NA) concentrations (Fig.4). Unexpectedly, the one min CO_2-inhalation did not induce any further increase in PC, which remained at the high immediate level during the exposure, and for 10 min pre-CO_2 thereafter. In contrast, 15, respectively 50 fold rises in plasma A and NA occurred within 45 s of exposure (Fig. 4).

DISCUSSION

The fact that both the neocortical and the hippocampal EEG exhibited changes characteristic of the second stage of anaesthesia (Stumpf 1965; Pichlmayr et al. 1984) before the appearance of muscular jerks indicates that the swine were anaesthetized and unconscious at the onset of the motor reactions. During slaughterhouse CO_2-anaesthesia the muscular jerks have been reported to occur earlier (after about 15 s of CO_2-inhalation) (Blomquist 1957; Hoenderken et al. 1979). Possibly, therefore, CO_2-independent stress/arousal factors present in an abattoir may facilitate the development of motor reactions with the result that they will become manifest before the EEG has attained an anaesthesia pattern. It is also possible that early motor reactions in abattoirs are of another ("voluntary") type, emanating from a still active neocortex. Using CO_2 concentrations hither than 80% will probably hasten the development of severe hypercapnia/hypoxemia, and consequently also the CNS events leading to the development of muscular jerks. Obviously, the animals remained depressed below the stage of surgical anaesthesia as long as neocortical EEG silence persisted, i.e. for about one minute following the end of the CO_2-inhalation. This finding implies that, from the ethical point of view, exsanguination can be performed safely within the first minute following end of exposure. Neocortical EEG changes became apparent after 13 s of CO_2-inhalation (Fig. 1). At approximately this stage (at 15 s) pronounced arterial hypercapnia, hypoxemia and acidemia had already developed. Thus, any of these distortions per se may have elicited the EEG changes. However, it appears more likely that all the three factors acted jointly as depressors of the neocortical neurons. ACTH-mediated glucocorticoid release can be elicited by a variety of emotional factors, for instance restraint of conscious animals (see Guyton 1986). It explains the 3-fold increase in PC seen here in response to the pre-exposure proceeding. No corresponding increase occurred in plasma A and NA, which suggests that PC is a more sensitive index of emotional stress than are plasma A and NA concentrations in the pig. The fact that no further increase in PC occurred during, and after the CO_2-inhalation may be regarded as evidence that the CO_2-exposure as such did not mean any additional emotional strain to the pigs. However, the response maximum for cortisol release may have been reached before the descent into the CO_2. The sympathetic nervous system is activated by principally the same physical and emotional stress factors as the pituitary-adrenocortical axis (Axelrod and Reisine 1984). It can no' be excluded, therefore, that the increases in plasma A and NA seen during the CO_2-inhalation to some extent might have been manifestations of emotional stress. However, among physical stress factors, acidosis constitutes a powerful sympathicoadrenal stimulus (Barton et al. 1982). It is likely, therefore that the severe respiratory acidosis developed at 45 s of CO_2-inhalation was the main cause of the conspicuous A/NA elevation observed at that stage.

ACKNOWLEDGEMENT

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