Changes in Electrical Activity of the Cerebral Cortex During Amylobarbitone Anaesthesia in Rabbits

A.T. Elsa, 'U.A. Osunkwo and 'P.A. Onyeyi
Department of Veterinary Medicine, Surgery and Theriogenology, Department of Pharmacology, Usmanu Danfodiyo University, Sokoto, Nigeria

Abstract: The changes in electrical activity of the cerebral cortex during amylobarbitone anaesthesia were studied in rabbits. Electrical activity of the rabbit cerebral cortex recorded (using an oscilloscope), sixty minutes after induction of amylobarbitone (30 mg kg⁻¹, body weight, i.p.) anaesthesia did not significantly differ from control values obtained before anaesthesia or 24 h after recovery from anaesthesia. Amylobarbitone administered @ 60, 90 and 120 mg kg⁻¹ body weight significantly (P< 0.05) and dose-dependently decreased the cerebral electrical activity. Therefore, amylobarbitone at the dose of 60 mg kg⁻¹ body weight or higher may cause depression in the electrical activity of the rabbit brain.

Key words: Amylobarbitone, cerebral cortex, rabbits, electrical activity, anaesthesia

Introduction
The single most important discovery in the exploration of nervous mechanism was that the nerve impulse is identical with an electrical change (Darnaud, 1984; Abbott and Howarth, 1973). The electrical sign of activity has given the investigator a means of studying the function of nervous system in the living organisms through the complexity of structure that the microscope can reveal only in dead tissue (Gasanov, 1987). Recording instruments have evolved through the capillary electrometer, the cathode ray oscilloscope and electromagnetic recorders to the cathode-ray oscilloscope. The cathode ray oscilloscope device with its inelastically electron beam has proved to be the instrument of choice for recording the exceedingly brief and feeble currents of nerve activity and has become the display unit for modern laboratory computers (Coombs et al., 1955; Mize, 1984). The measurement of nerve conduction velocity is now a standard radiodiagnostic procedure in clinical neurology (De Jesus et al., 1973). The most commonly used techniques estimate the conduction rates of fast fibers only (Dawson, 1956), but new methods allow for the study of slow conducting sensory (Butchthal and Rosenthal, 1971) and motor fibers (Butchthal and Rosenthal, 1971; Thomas et al., 1985). Several investigations have been performed using motor evoked potential monitoring as a diagnostic tool for detecting the impending damage of central nervous system descending pathways in an early still reversible state during anesthesia and neurosurgery (Thees et al., 1997). In many fields of neurophysiology it is often of interest to observe the firing pattern of single neuron on an oscilloscope. It is convenient at the same time to observe the rate of cell firing and to record it on a chart recorder (Graysone et al., 1967; Gasanov, 1967). General anesthetic gains access to the brain rapidly inducing a stable level of anesthesia and providing an accurate index of potency for every class of anesthetic agents (Finsente et al., 1986). Amylobarbitone is an intermediate acting barbiturate widely used in laboratory animals for organ resection purposes (Gootz et al., 1987; Osunkwo et al., 1989). It was therefore of interest to develop a technique to record accurately the discharges in the cerebral cortex in the amylobarbitone anaesthetized rabbits.

Materials and Methods
Twenty New Zealand rabbits of either sex weighing 1.0–2.3 kg and aged 6 to 12 months were used for the study. The rabbits were divided into four groups each of five and housed in research laboratory, Department of Pharmacology, Usmanu Danfodiyo University Sokoto. The rabbits were fed with green leaves and rabbits pellet. Water was provided ad libitum. All the rabbits were groomed with cinnamophyll (Asuntol 16%, Bayer Pharmaceuticals, Switzerland) and daworn with Levamisole (Citarin-L 2.5% solution, Bayer Pharmaceuticals, Switzerland) at a subcutaneous dose of 5 mg kg⁻¹ body weight. The animals were kept for observation for four weeks period of acclimatization before the experiment was commenced.

Rabbits in group A, received amylobarbitone (30 mg kg⁻¹, body weight, i.p.) and the electrical activity of the cerebral cortex was observed after 60 min of anaesthesia induction by placing the oscilloscope (Hitachi Denki Ltd., Tokyo, Japan) probe on the cerebral cortex through a hole, drilled in the skull with spinal needle. The electrical signals of the cortex were fed into the digital recorder (Model WR7700, Graphite Corporation, Japan) and recorded on the chart paper.

Animals in groups B, C and D were treated with amylobarbitone intraperitoneally at a dose of 60, 90 and 120 mg kg⁻¹ body weight respectively. A dorsal craniotomy was performed on all the rabbits in three groups. The head was clipped, shaved and disinfected. A cranial bony midline skin incision was made on the skull. The scalp was cut along the midline and retracted with the temporal muscle in order to reveal nearly all the frontal and parietal bone and as much of the temporal bone as possible. All the subcutaneous tissues were carefully scraped away. The skull was cut with a scalpel blade along with the tissues attaching parietal, maxillary and frontal and occipital bones. The part of the cut skull was reflected cranioly to expose the cerebral cortex. The probe of the oscilloscope was placed on the cortex in situ. The signals of the electrical activity of the cortex was fed into the digital recorder and recorded on the chart paper. The reflected bone of the skull was placed into its anatomical position. The skin was sutured with silt in simple interrupted suture pattern. The surgical site was dressed with gentian violet. Postoperatively, 400,000 IU, procaine penicillin was injected intramuscularly once daily for three days. On postoperative day one, the interrupted skin sutures were removed and the signals of the electrical activity of the cortex were recorded. The reflected part of the skull and skin was returned and re-sutured. Gentian violet was applied on the surgical site to prevent flies and other insects and to enhance the healing process. Data obtained from the study was presented as means standard deviation and differences between means were analyzed using analysis of variances (ANOVA).

Results
The results of the oscilloscope recordings of the electrical activity of the cerebral cortex at four different doses of amylobarbitone (30, 60, 90 and 120 mg kg⁻¹) indicated that the electrical activity of the rabbits cerebral cortex was recorded 60 min after induction of anaesthesia (Table 1) and 30 mg kg⁻¹ amylobarbitone differed
Table 1: Electrical activity of the cerebral cortex during treatment with

\[
\begin{array}{cccc}
\text{Groups of rabbits} & \text{Before} & \text{During} & \text{After} \\
& \text{anaesthesia} & \text{anaesthesia} & \text{anaesthesia} \\
& (24 h) & (60 min) & (24 h) \\
A (30) & 36.1 \pm 0.1 & 27.6 \pm 0.9^* & 26.8 \pm 1.1 \\
B (60) & 34.0 \pm 1.6 & 5.8 \pm 1.9^* & 33.8 \pm 1.5 \\
C (90) & 35.7 \pm 1.4 & 5.8 \pm 1.9^* & 35.2 \pm 1.6 \\
D (120) & 36.0 \pm 1.4 & 4.2 \pm 1.4^* & 36.1 \pm 1.3 \\
\end{array}
\]

* P < 0.05 when compared with values before anaesthesia and 60 min.
during anaesthesia using analysis of variance.

non-significantly from the control values recorded before
anaesthesia or values obtained 24 h after recovery from
anaesthesia. Although amylobarbitone at 60 mg kg^{-1} body weight,
significantly (P < 0.05) decreased cerebral cortex electrical activity
after 60 min of anaesthesia, induction of higher doses of
amylobarbitone (90 or 120 mg kg^{-1} body weight) significantly
(P < 0.05) and dose-dependently decreased the cerebral electrical
activity of rabbit brain.

Discussion
The oscilloscope was used to study the electrical activity of the
rabbit brain because it has proved to be the instrument of choice
for recording the exceeding brief and feeble currents of nerves
and has become the display unit for modern laboratory work
(Grayston et al., 1969; Evans, 1985). When amylobarbitone was
administered at a lower dose, all the rabbits so treated showed
insomnia with non-significant decrease in electrical activity of the
cerebral cortex, 60 min after induction of anaesthesia. The slight
decrease in the electrical activity of cortex suggests that the
central effect of amylobarbitone did not largely affect the cortex
to produce significant changes in the rabbit brain.

However, amylobarbitone when administered at a higher dose,
caused a significant decrease in electrical activity of the cerebral
cortex of rabbits. Also much higher doses of amylobarbitones,
significantly and dose-dependently further reduced the cerebral
electrical activity. These observations can be explained by the fact
that changes in electrical activity of the cortex at the normal
metabolic state could be affected by the degree of depolarization
of the nerves produced by the action of anesthetic agent. As
reported by Delmonte (1984) and Chen and Tothe (1984), if the
nerve is impaired by a narcotic agent its excitability will be
reduced. It therefore follows that amylobarbitone may produce
central effects consistent with an inhibition of these brain areas
causing narcosis and subsequent decrease in electrical activity
of cortex. Thus a possible cortico-inhibitory mechanism of central
origin might be operating during amylobarbitone anaesthesia.

Another possible explanation for the mechanism of
amylobarbitone produced reduction in the electrical activity of
the cerebral cortex is that many nerve cells are affected by the
anaesthetic agent that could be responsible for adding their
activity in such a way as to produce waves of amplitude having
their major electrical characteristics in common (Thomas et al.,
1985; Gowanov, 1987). The exact neuronal mechanisms at the
membrane level are still to be defined to suit all the available data
but there is now general agreement that neocortex potentials are
generated by postsynaptic activity and that their rhythms are
paced from the thalamus (Brazier, 1961; Nwanwakwa and
Ezemuzie, 1995). However measurement of the electrical activity
of the cerebral cortex of rabbits using an oscilloscope at 24 h
before and after anaesthesia did not produce any significant
change. This is probably due to the unavailability of anesthetic
agent in the body system. It is also possible that at least parts of
the inhibitory actions at amylobarbitone on the cerebral cortex
could be explained by cortical mechanism (Storlade and Lines,
1988). For example the electrical activity of the cortex was
reduced by amylobarbitone at both lower and higher doses but
remains remarkably unchanged in unanaesthetized rabbit. These
findings suggest that dose of amylobarbitone equal or greater
than 60 mg kg^{-1} body weight was adequate to cause decrease in
the electrical activity of the rabbits' brain.

Acknowledgments
The authors gratefully acknowledge Usmanu Danfodiyo University
Sokoto for partial funding of this work.

References
Brain: The First Half-Century, 1st Edn., Pitman Medical
Butchhal, F. and A. Rosental, 1971. Sensory condition from
digit to palm and palm to wrist in the carpel tunnel syndrome.
Chen and Z. A.L. Tothe, 1984. Cerebral response to medullary
pyramids stimulation in the rabbits. Brain Behav. Evol., 26:
175-186.
Coombes, J.S., J.C. Eccle and P. Fatt, 1955. The electrical
properties of the motor neuron membrane. J. Physiol. (Lond.),
130: 291-325.
Dawson, G.D., 1956. The relative acetylatability and conduction
velocity of sensory and nerve fibers in man. J. Physiol. (Lond.),
phenomena associated with medication practice. A literature
effect of cold on nerve conduction of human slow and fast
Evans, M.H., 1985. An oscilloscope spot intensities to improve
Firestono, L.L., J.C. Miller and K.W. Miller, 1986. Tables of
physiological and pharmacological properties of
anaesthetics. Roth S.H. and Miller K.W. (Eds.). Plenum Medical
amylobarbitone and psychosocial withdrawal. Psychiatr. J.
Univ. Ottawa, 12: 51-52.
meter and oscilloscope intensity modulator for single cell
Nwanwakwa, R.N.P. and F.L. Ezemuzie, 1995. Lecture notes and
Port Harcourt, Nigeria, pp: 125-130.
Differential effects of amylobarbitone and urethane on
cardiovascular effects of chloroquine in rats. Nig. J. Physiol.
Sci., 8: 11-17.
the thalamus and the associated neural interplay. Physiol. Rev.,
68: 649-742.
C. Frankel and J. Wurker, 1997. Dose-dependent suppression
of motor evoked potentials by nitrous oxide. Br. J. Anaesth.,
78: 169-172.
Stefanoson, 1985. Electrical activity of cerebral cortex during
induced hypotension in man. A comparison of sodium