ELECTROCARDIOGRAMS AND ELECTROENCEPHALOGRAMS OF SOME NIGERIAN DOMESTIC ANIMALS: NORMAL PATTERNS AND EFFECTS OF SOME DRUGS AND DISEASE CONDITIONS

BY

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A

THESIS

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DECLARATION

This is to certify that I carried out the work reported in this thesis in the Departments of veterinary Physiology and Pharmacology, and Pharmacognosy and Drug Development, Ahmadu Bello University, Zaria, under the supervision of Prof. Y.O. Aliu, Dr. E.N. Sokomba and Prof. C.D. Ezeokoli.

The work of other investigators in the areas covered in the thesis is duly acknowledged and referred to appropriately. No part of this thesis has been submitted elsewhere for a degree or diploma.

DIGITIZED

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CERTIFICATION

This thesis entitled " Electrocardiograms and Electroencephalograms of some Nigerian Domestic Animals: Normal Patterns and Effects of some Drugs and Disease Conditions" by Mohammed Mamman meets the regulations governing the award of Master of Science of Ahmadu Bello University and is approved for its contribution to scientific knowledge and literary presentation.

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DEDICATION

This thesis is dedicated to those who honourably seek and do right.

And those who strive
In Our (cause) - We will
Certainly guide them
To our paths:
For verily Allah
Is with those
Who do right

-Q.XXIX:69

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ABSTRACT

The present study was conducted to establish baseline electrocardiographic and electroencephalographic patterns and data for Nigerian indigenous domestic animals which were, until now, not documented. Generally, the findings of this study are similar to those established for the respective exotic species.

The electrocardiographic data determined were in respect of the patterns, voltages and durations of conventional waves of the electrocardiogram (ECG) of clinically healthy indigenous domestic cat, cow, dog, donkey, goat, horse and sheep. In these species, the normal ECG pattern consisted of the P, Q, R, S, T, QS and R' wave forms that were variably shown among 6 (standard and augmented) limb leads, and 5 selected precordial leads. The shortest and longest mean durations obtained for these wave forms were respectively 27.50 ± 5.58 msec (P wave in cat lead CV6LL) and 443.33 ± 28.75 msec (QT interval in horse lead rV2). The lowest and highest absolute mean deflection potentials were respectively recorded in the sheep (0.003 ± 0.040 mV, P wave in lead III) and donkey (1.03 ± 0.067 mV, R wave in lead V10).

While several related previous studies were conducted in anaesthetized exotic breeds of dogs and cats, the data established from the present study were derived from conscious indigenous animals.

From experimentally- induced myocardial ischaemia and infarction studies in dogs, characteristic abnormal electrocardiographic changes observed included QS complex and deep Q waves in standard limb leads, ST segment elevation, arrow- head T waves, and paroxysmal ventricular tachycardia.

Standard drug- induced electrocardiographic alterations that included decreased QT interval (carbachol), increased QT interval (atropine), reflex tachycardia (chlorpromazine), paroxysmal ventricular tachycardia (digoxin), and increased R wave amplitude and T wave depth (pentobarbitone) were reproduced in dogs and cats.

Also established are the chief features of the scalp electroencephalogram (EEG) of clinically healthy indigenous domestic cat, dog, goat and sheep. The EEG frequency was slowest (2 waves per second, right temporal- right occipital), in xylazine- sedated sheep and fastest (11 waves per second, left frontal- right frontal) in dogs under

pentobarbitone general anaesthesia. The EEG deflections were minimal (5 uV, right occipital-left occipital) in cats under barbiturate general anaesthesia, and maximal (120 uV, right frontal-left frontal) in xylazine- sedated goats. As reported by other authors, the present study also concludes that frequency change may be the most reliable indicator of EEG abnormality.

The patterns of depth EEG of chickens were studied. The normal activity was slowed and accompanied by high voltage waveforms following administration of halothane, pentobarbitone and xylazine, and accelerated with low voltage waveforms with apomorphine and leptazol.

The use of the ECG and EEG data established through this study for clinical and research purposes in relevant Nigerian indigenous domestic animals is therefore suggested.

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GLOSSARY

AH	Accessory Hyperstriatum
AULL	Augmented Unipolar Limb Lead
AVN	Atrio-Ventricular Node
Ca++	Calcium ions
CM	Cell membrane
CNS	Central Nervous System
CTZ	Chemosensitive Trigger Zone
CVS	Cardiovascular system
ECF	Extracellular Fluid
ECG	Electrocardiogram
EEG	Electroencephalogram
EMG	Electromyogram
ECGy	Electrocardiography
EEGy	Electroencephalography
Hz	Hertz
HVFA	High Voltage Fast activity

HVSA	High Voltage Slow Activity
K+	Potassium ions
LVFA	Low Voltage Fast Activity
mcV	Microvolt
mg/kg	Milligrams per kilograms
mm	Millimetre
mV	Millivolt
msec	Milliseconds
M	Metre
n	Number
N	Number
Na+	Sodium ions
NS	Nervous System
OT	Optic Tectum
RF	Reticular Formation
sec	seconds

SAN	Sino-Atrial Node
SLLs	Standard Limb Leads
SPCM	Semipermeable Cell Membrane
SUPLs	Semiunipolar Precordial Leads
uV	Microvolt
ULLs	Unipolar Limb Leads
UPLs	Unipolar Precordial Leads
WCT	Wilson's Central Terminal
wps	Waves per second

CHAPTER ONE:

INTRODUCTION TO VETERINARY ELECTROCARDIOGRAPHY AND ELECTROENCEPHALOGRAPHY

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INTRODUCTION

The normal functions of the body depend essentially on the integrity of cardiovascular and central nervous systems which respectively determine systemic circulatory and nervous regulatory control. The critical organ in the cardiovascular system (CVS) is the heart, and in the nervous system (NS), the brain. A feature esential for activity of both organs is the inherent capacity to generate, conduct and spread electrical activity. Any factor that will cause significant pathologic alteration in the electrical activity of the heart and/or the brain may cause dysfunction of either or both organs.

Such factors include disorders of generation and/or transmission of impulses (Klemm, 1976; Schamroth, 1971); reduction in blood flow (Guyton, 1981); tissue dysfunction and inflammation (Hamlin, 1976); anatomical abnormalities and toxic conditions (Ettinger and Suter, 1970); as well as pressure-induced mechanical dysfunction due to parasites and other causes (Klemm, 1976). Depending on their severity, some of these disorders can be detected through studies of several functional activities of the heart and brain.

Except in acute dysfunction of the heart (heart attack) or the brain (stroke), congenital or

acquired pathological disorders in these two organs usually progress from mild reversible stages to chronic irreversible forms. Therapeutic reversal of such conditions can usually be implemented following early electrodiagnosis.

There are several methods for detailed studies of the qualitative and quantitative aspects of the CVS and NS including their electrical activities. However, the best means of studying the electrical activities of the heart and brain are, respectively the electrocardiogram (ECG) and electroencephalogram (EEG).

Since their introduction in 1875 and 1903, electroencephalography (EEGy) and electrocardiography (ECGy) have assumed strategic roles in the electrodiagnosis of disorders of the brain (Klemm, 1976) and heart (Ettinger and Suter, 1970) respectively.

An electrocardiographic lead is a hypothetical line drawn between two sites on the body where electrodes have been positioned (Ettinger and Suter, 1970). Advances in ECGy have enabled the development of several new leads from the original standard limb leads (SLLs). These "new" leads include unipolar limb leads (ULLs), augmented

unipolar limb leads (AULLs), semiunipolar precordial leads (SUPLs), unipolar precordial leads (UPLs), oesophageal and orthogonal leads. Today through the use of radiotelemetry, it is possible to monitor the ECG of animals in exercise (Hill et al., 1977). In humans, twelve leads are routinely recorded. These include three each of SLLs and AULLs, and six UPLs. In domestic animals however, the principal leads recorded are the SLLs and AULLs plus usually one or more UPLs.

Modern electrocardiographs are multichannel enabling improved efficiency and reduced recording time. Coupled with microprocessors they provide on-the-spot analysis and summary outputs.

Several reports reflect the importance of ECGy in healthy and in diseased domestic animals. The normal ECG has been documented for the exotic dog, cat, horse, cow, (Clark et al., 1966), and goat (Upudhyay and Sud, 1977). Electrocardiographic alterations of diagnostic importance have been documented in such disorders as aortic stenosis in the dog (Shirai, 1979), arrhythmia in the horse (Hilwig, 1977), hypomagnesaemia in calves (Hovarth, 1971), lantana poisoning in sheep (Uppal and Paul, 1972) and atrial fibrillation in a cat (Washizu,

1979). There is an increasing growth in the number of reports of pregnancy diagnosis using the ECG (Parkes and Colles, 1977).

The historical development of veterinary EEGy in the strictly applied sense is very brief (Klemm, 1976). In its early periods a book was published on the studies of cerebral functions in normal and abnormal conditions (Pampiglione, 1963). Subsequently, notable contributions from several authors (Croft, 1963; Klemm and Mallo, 1966) through clinical and experimental research reports aided in developing animal EEGy to its present state.

The most commonly used montages, ie electroencephalographic electrode arrangements, are based on methods proposed by Redding (1965), Klemm (1968), and Beaver and Klemm (1973). Modern electroencephalographs are very costly to install, run and maintain. Nevertheless, clinical EEGy in domestic animals is rapidly gaining place as a routine diagnostic facility in brain disorders of animals (Klemm, 1976). Through its uses the EEG of animals in both normal and abnormal states has also been documented.

For most clinically useful and research drugs, there are extensive reviews on how they affect the animal ECG and EEG. In one report, the general anaesthetic, thiopental was used in high doses to produce isoelectric EEG in the dog (Steen and Michenfelder, 1979). On the heart this drug in the same species causes arrhythmia less often than thiamylal- 40 and 85% incidence respectively (Booth and McDonald, 1982). While doxapram in therapeutic doses does not cause alteration in the EEG pattern; increasing depth of anaesthesia with halothane causes progressive replacement of fast, low voltage EEG waves by slow waves of greater voltages.

Tricyclic antidepressants cause synchronisation and suppression of alpha rhythms in the EEG at the same time causing ECG changes manifested by T wave inversion and prolonged myocardial conduction time. The anti- arrhythmic agent quinidine causes increased QRS duration, PR and QT intervals. Bretylium causes similar effects but the QRS duration does not change. Hypercalcaemia and hyperkalaemia cause decreased QT interval and tall, peaked T waves respectively. In lead poisoning, EEG changes precede clinical signs of illness and changes in myocardial electrolytes occur before ECG alterations (Asokan, 1974). The ECG and EEG,

therefore, play important roles in the establishment of the safety of clinically useful therapeutic agents.

Normal EEG tracings from several domestic animals in varying degrees of consciousness have been determined (Klemm, 1976), as well as in some disorders that include canine encephalitis and hydrocephalus (Klemm and Hall, 1971), and sheep coenurosis (Bagedda, 1968). However, significant variations in the ECG and EEG patterns occur due to differences in species, breed, physiologic status and disposition during recording. Since differences between the normal and abnormal (though often slight) are significant, direct transfer of data derived from one breed to another for any reliable assessment is unjustifiable (Hilwig, 1976). This means that the ECG and EEG data established for exotic breeds may not be applicable directly to our indigenous domestic animals. This research project was therefore conducted to establish baseline ECG and EEG patterns for Nigerian indigenous domestic animals, as well as the effects of some drugs and diseases on the wave characteristics.

CHAPTER TWO:
ELECTROCARDIOGRAPHY

Literature review

Willem Einthoven (1860-1927) is considered as the father of ECGy. He invented the string galvanometer, the basic component of the electrocardiograph, and was the first to use it to record the ECG of man in 1903. Veterinary clinical ECGy began early this century when the first documented case of abnormal equine ECG characterised by auricular fibrillation was reported (Lewis, 1911). Some notable contributors to the development of veterinary ECGy are Lannek (1949); Horowitz et al., (1953); Broojmans (1957); Detweiler et al., (1960; 1965); and Crawley and Swenson (1966).

Electrophysiology of the ECG

The ECG is a graphic record of the voltage produced by the myocardium during the course of depolarisation and repolarisation plotted against time. Normally, each heartbeat is initiated by a spontaneous electrical discharge in the sino-atrial node (SAN). The electrical impulse then passes across the atria to the atrio-ventricular node (AVN), through the bundle of His and down the purkinje fibres to the ventricular myocardium. The sum of all these electrical events that occur in the atria, ventricles and their specialised conduction tissues can be recorded from the skin surface as the ECG.

The resting heart muscle cell has an electrical potential of -90 millivolt (mV) across the cell membrane, the interior of the cell being negative with respect to the exterior.

When the cell is adequately stimulated to depolarise, positively charged sodium ions (Na+) flow into the cell across the semipermeable cell membrane (SPCM) reversing the polarity of the stimulated portion of the cell membrane (CM). The electrical gradient across the CM becomes +30 mV. Calcium (Ca++) probably also diffuses along with Na+ into the muscle to catalyse excitation-contraction coupling (Winegrad, 1961). The change in potential that occurs when the electrical gradient across the CM is reversed is registered as a voltage deflection on the ECG. During depolarisation, potassium (K+) begins to diffuse to the outside of the cell and when the rate of diffusion exceeds that of Na+ influx, repolarisation begins. This leads ultimately to a high concentration of extracellular K+ and intracellular Na+. During repolarisation of the myocardium another series of voltage deflections is reproduced and recorded in the ECG. Sodium and K+ diffuse into the extracellular fluid (ECF) and the myocardial cell, respectively, during diastole.

The electrical activity of the heart is relatively more organised than that of any other

excitable tissue in the body because the myocardial cells behave as a "functional syncytium". This enables excitation of the entire atrial and/or ventricular mass following adequate stimulation of a single myocardial cell.

Some specialised cardiac tissues namely the SAN, some atrial fibres, the bundle of His and its branches, and the purkinje fibres are capable of spontaneous discharge. Under abnormal circumstances other atrial and ventricular muscle cells can assume automaticity (Hoffman et al., 1966); in the AVN however, there are no automatic fibres, and conduction is both slow and decremental. The normal pacemaker of the heart is the SAN; it normally has the most rapid rate of spontaneous depolarisation. Of the several theories evolved to define the basis of clinical ECG, the single dipole is considered valid and is used in its interpretation (Ettinger and Suter, 1970).

The theory assumes that the multiple dipoles generated by the heart at a given instant are effectively resolved into a single electrical force before reaching the body surface.

Electrocardiographic recording techniques

The heart is considered as a single mass of cardiac muscle placed within the body which is filled with ECF. Consequently, recording electrodes

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attached to the body surface permit detection of alterations in electrical potential created by the depolarisation and repolarisation of the myocardium.

In obtaining the ECG, electrodes in specifically attached positions on the subject provide the means by which the electrical activities of the heart are transmitted to the electrocardiograph for processing followed by display of corresponding wave forms constituting the ECG on the ECG-paper. An electrocardiographic lead could either be unipolar or bipolar. In bipolar recordings both the recording (positive) and the reference (negative) electrodes reflect changes in potentials which could be additive or subtractive to each other. The recording electrode in unipolar recordings varies in potential with changes in the electrical state of the myocardium, while the other electrode(s) in the lead maintain(s) a zero potential during the course of depolarisation and repolarisation (Breazile, 1971). Depending on the lead being recorded, electrodes attached to specific body positions acquire positive or negative polarity.

In recording SLL I, the positive and negative terminals of the electrocardiograph are connected to the region just proximal to the olecranon on the caudal aspects of the left and right forelimbs respectively. The position of the positive

electrode in lead II is the region just proximal to the patella on the cranial aspect of the left hindlimb, the position of the negative electrode in leads I and II is the same. In recording lead III the positive terminal used in deriving lead I, in its same position, becomes negative while the positive terminal used in recording lead II retains its position and polarity.

In all animal ECG recordings an electrode attached on the cranial aspect of the right thigh just above the patella connects the animal to the ground. Different leads enable the viewing of the net electrical activities occurring in the heart from the specific inclinations of the arrangements of the leads in relation to the heart. The occurrence, amplitude and polarity of the wave forms in the ECG could therefore vary with the lead. Additional technical modifications, typified by the Wilson's central terminal (WCT), permit increase in wave amplitudes of some leads in relation to others (Breazile, 1971).

The WCT consists of electrode series, one placed on each of the right and left forelimbs, as well as the left hindlimb in the specific positions mentioned above. Each of these electrodes is in turn connected to three separate 500 ohm resistors and finally to a single lead which connects to the recording device. The terminal serves as a

reference electrode which, theoretically, remains zero during the course of depolarisation and repolarisation of the myocardium. The WCT is utilised in some, but not all, electrocardiographic leads.

The three ULLs in veterinary ECGy are derived through the use of the WCT. They are named in accordance with the limb on which the recording electrode is attached. With the recording electrode placed on the right and left forelimbs, and on the left hindlimb the ULLs VR, VL and VF are obtained respectively. About 50% increase in amplitude is however achieved when the same electrode arrangements mentioned above are slightly modified. The AULLs aVR, aVL and aVF are, respectively, similar to VR, VL and VF. In the augmented leads, however, when the recording electrode is placed on a limb, the electrode on that limb which is being utilised for the WCT is removed. A similar modification has been adopted for the precordial (chest) leads.

In the past, SUPLs have been greatly used. One of those, CR, utilised a recording electrode on the thoracic wall and a reference electrode attached to the right(R) or left(L) forelimbs, or left hindlimb(F). The location of the recording electrode on the thoracic wall is indicated by different subscripts that describe the side and

level of the thorax in which the placement is made.

In the dog lead CR5RL utilizes a recording electrode placed on the right 5th intercostal space immediately lateral to the sternum; in lead CR6LU the placement of the recording electrode is over the left 6th intercostal space at the costo-chondral junction. For higher amplitude recordings the UPLs are more extensively used than the SUPLs.

Like the AULLs, all UPLs utilize the WCT thereby providing higher amplitudes of similar wave forms obtained with the SUPLs. The five commonly used AUPLs in domestic animals are:

(1) CV5RL (rV2) - in this lead the recording electrode is placed over the low right 5th intercostal space immediately lateral to the sternum

(2) CV6RL (rV4)- the 6th low right intercostal space is the position over which the recording electrode in this lead is placed

(3) CV6LL (V2)- the location of the recording electrode in this lead is the left, low 6th intercostal space

(4) CV6LU (V4)- the only difference between this lead and CV6LL is the placement of the recording electrode in the upper left 6th intercostal space at the costochondral junction

(5) V10 - a widely recorded lead in domestic species. The position of attachment of the recording electrode in this lead is over the 7th thoracic vertebra on the dorsal midline.

Electrocardiographic equipment

Unlike older machines, modern electrocardiographs are much easier to operate since the operator does not have to attach, remove and reattach electrodes for each different lead recording. Following a single appropriate placement of the electrodes in the specified positions, the various leads are recorded on the standard electrocardiograph by moving the selector switch to the lead desired. The machine automatically establishes correct polarities for the lead chosen. Alligator clips and hypodermic needles are the most commonly used electrodes in veterinary ECGy.

Animal preparation for ECG recording

With the four limbs extended perpendicular to the body, the right lateral recumbent position is recommended for the dog because much of the empirical data available for this species had been determined using the same position (Ettinger and Suter, 1970). However, some authors advocated the use of other positions, notably the sternal in the dog and cat (Gonin, 1962; Rubin, 1968). In other domestic animals, the standing position with the four limbs perpendicular to the torso is recommended. Regardless of the methods used, the relationship of the animal's torso and the

extremities must not vary from one examination to another if consistent and reproducible recordings are to be obtained to make comparison between recordings (made at different times or places) possible.

Even when the machine and animal are fully grounded, there may still be a need for the placement of an insulator between the animal and the lying or standing surface. In addition adequate contact of electrodes and the subject's skin must be ensured. This may not necessarily involve shaving as solution of alcohol or soap applied over the electrodes and the specific skin attachment areas is usually satisfactory (Hamlin, 1976).

Established ECG wave patterns

The wave forms in the ECG are generated during the course of the depolarisation and repolarisation of the heart. Mechanisms underlying the polarity, configuration and rhythm of these wave forms have been adequately described (Ettinger and Suter, 1970; Breazile, 1971; Schamroth, 1971; Hamlin, 1976). Activation (depolarisation) pattern of the myocardium differs among the species. Atrial activation and spread are respectively 80 milliseconds(msec) and 1 metre(M)/second(sec) in the dog, and 130 msec and

2M/sec in the horse. However, little is known about atrial and ventricular repolarisation pathways in animals (Hamlin, 1976). Variation in the distribution of the purkinje fibres appears to be the major factor determining the different ventricular depolarisation pathways and therefore, the corresponding wave forms. In the dog, the endocardial quarter purkinje fibres, in an endocardial to epicardial direction, penetrate and terminate in the endocardial third of the ventricular free walls. However, in the goat the purkinje fibres penetrate the entire endocardial to epicardial distance. These differences are reflected in the manner of activation and its spread.

In both the dog and goat ventricular activation begins with the apical third of the interventricular septum, predominantly from left to right. This event, in the goat is followed by the activation of the ventricular free wall which occurs rapidly and with a single burst (Hamlin, 1976). Activation of the middle third of the interventricular septum follows immediately. In the dog the activation wave first spreads to the end-points of the purkinje fibres in the subendocardium and then in an endocardial to epicardial direction through the ventricular free walls. And in a similar manner the middle third of the septum is also activated. A little later the

wave of activation reaches the epicardium of the right and then left ventricles and also the basilar third of the septum. While the last portion to be activated in the goat is the basilar third of the interventricular septum, in the dog it comprises the bases of both ventricular free walls and that of the interventricular septum in an apicobasilar direction.

The most prominent wave forms established in the mammalian ECG (Fig. 2a) are:

P-representing combined right and left atrial activation

Q-which indicates initial depolarisation of the interventricular septum; its forces are directed rightward (with respect to the midline)

R-is most often the major positive deflection especially in SLLs.It represents depolarisation of the ventricular free walls; its forces are predominantly leftward

S-the terminal depolarisation of the basal portions of the ventricles and septum are represented by this wave

T-is inscribed in association with the more rapid period of ventricular repolarisation. Atrial repolarisation occurs simultaneously with ventricular depolarisation and the corresponding wave, Ta, is normally masked by the more prominent QRS complex. However, in right atrial enlargement the Ta appears and may cause the depression of the PQ segment.

The PQ(PR) segment is the isoelectric period that succeeds the P wave and is measured from the beginning of the P wave to the beginning of the first deflection away from the baseline indicating ventricular activity. During the PQ segment the impulse is being slowly conducted

through the AVN (Guyton, 1981).

PQ interval is measured from the onset of the P wave to the onset of the QRS complex.

The ST segment represents the slower phase of ventricular repolarisation and is measured from the end of the S wave to the beginning of the T wave.

The duration of the QRS complex is measured from the beginning of the Q to the end of the S waves.

The QT interval is measured from the onset of the Q wave to the end of the T wave.

There is marked variation in the amplitude, duration, configuration and polarity of the different ECG wave forms due to differences in species, age, purkinje fibre distribution, posture, etc. Clinically, normal characteristics (Table 2a) of these wave forms solely or in combination provide guidelines for the diagnosis of some cardiac disorders (Hamlin, 1976). For example, in ventricular hypertrophy the duration of QRS complex may be significantly prolonged and the potential of the accompanying R wave may be greater than 3 mV (Hamlin, 1976). Left atrial enlargement as occurs in mitral valvular insufficiency is characterised by a notched and prolonged P wave (Lannek, 1949). Inversion of the T wave may occur in myocardial ischaemia (Schamroth, 1971). Normal sinus rhythm is altered in arrhythmias (Huisman and Teunissen, 1963). The interpretation and significance of ST-T changes in relation to canine ECG have been reviewed.

Aids for recording the ECG

Standard electrocardiographs record on a paper speed of 25 mm/sec. The wave voltage deflections below or above the isoelectric (zero) line are measured in mV. The conventional calibration value is 10 mm to a mV. Most machines however have selector switch for paper speeds faster or slower than the conventional speeds. Similarly, the sensitivity could be increased beyond or decreased below 10 mm to 1 mV.

Restraint during ECG recording

Adequate restraint in recording the ECG is essential. Significant changes in the ECG could result from sudden bodily motion wholly or in part (Hill, 1968). With the aid of an assistant (or owner) comfortably but adequately preventing motion of a dog in a laterally recumbent position, the ECG is usually recorded smoothly, all other conditions being satisfactory. Occasionally, a dog may have to be muzzled or even sedated. Local anaesthetics have been used to eliminate pain sensation at points of electrode attachments.

This aspect of the research was therefore conducted to establish baseline ECG data for Nigerian indigenous domestic animals, and to reproduce the characteristic effects of some drugs and myocardial disorders.

Materials and methods

Indigenous breeds of dogs, cats, sheep, goats, cattle, horses and donkeys were used for this study. They were clinically normal, but for comparative purposes a few dogs and cats that were given atropine (Scanpharm A/S, Copenhagen, Denmark), carbachol (Norden Labs, Nebraska, USA), Chlorpromazine (Lab D-Delmars, France), Digoxin (JAM Pharmaceuticals, UK) or Pentobarbitone (May & Baker Ltd, England). For each animal species, both sexes were included varying in age from two weeks to twelve years. The number used for each species is summarised in Table 2b.

A total of fifteen clinically healthy dogs, four as controls, were used for myocardial ischaemia, injury and infarction studies as described by Hess et al. (1985).

Restraint for ECG recording

Dogs and cats were placed on formica or wood insulated tables comfortably immobile in right lateral recumbency with the aid of an assistant as shown (Fig 2b).

The fore- and hindlimbs were kept perpendicular to the torso, in one position and separate to prevent contact between adjacent electrodes throughout each recording procedure. In such a position the animal was therefore kept adequately restrained allowing smooth, artefact-free recording. While most dogs remained satisfactorily calm throughout the recording period some did prove uncooperative. Consequently, it was occasionally necessary to suspend recordings during shivers or other bodily motion severe enough to cause significant effects on the ECG tracings.

On a few instances dogs had to be muzzled and reassured by petting. Sometimes electrodes had to be removed and reattached, or the animal allowed to return to a sternal position to rest briefly followed by resumption of recording. Because cats were generally uncooperative tracings were taken (with the cats) under pentobarbitone general anaesthesia.

All ECG tracings from other species were taken with the animals in the standing position (Fig. 2c). Special restraint facilities were used to ensure limited motion in the extremities and therefore, the torso.

For cattle, horses and donkeys a metal crush was used. This physical device allowed an animal to enter at one end in between side metals, strongly built into the ground and walls, thus limiting lateral motion. The neck of the animal coming out at another end was comfortably compressed laterally by metals, but firmly enough, to discourage lateral movement of the cranial cervical area including the head. In that restrained position most animals remained calm, long enough to complete recording. Recording was suspended when the animal struggled and resumed after it calmed down. The standing area and, occasionally, the sides of the "crush" had to be covered with a rubber or wooden insulator. In sheep and goats restraint was achieved using a small wooden "crush" working on similar principles with the larger metal "crush". During each recording procedure, significant distractive sound, motion and similar activities were avoided.

Electrocardiographic electrodes

Grease-free alligator clips coupled securely to cable tips were used as electrodes.

Electrocardiograph

The recorder used was a portable electrocardiograph (Fukuda Denshi, Model FJC-7110) with automatic selector switch for recording SLLs, AULLS and UPLs.

Recording procedure for ECG

With an animal appropriately positioned for ECG recording each specified electrode attachment area was moistened with a weak soap or 70% alcohol solution followed by the attachment of the electrode specific to the area. Accordingly, all the electrode attachment areas were sequentially moistened and appropriately connected with their respective electrodes. After animal preparation the electrocardiograph was then switched on, sensitivity adjusted to 10 mm (to 1 mV) and central placement of the stylus was ensured. As soon as the stylus was adequately hot a mV calibration voltage was recorded on the ECG heat-sensitive paper running at a speed of 25 mm/sec. Such calibration necessarily preceded and succeeded every recording procedure. Subsequently, the SLLs and AULLs as well as some UPLs were

recorded in all animals. In some tracings a few leads especially SLL II and UPLs were recorded at 50 mm/sec.

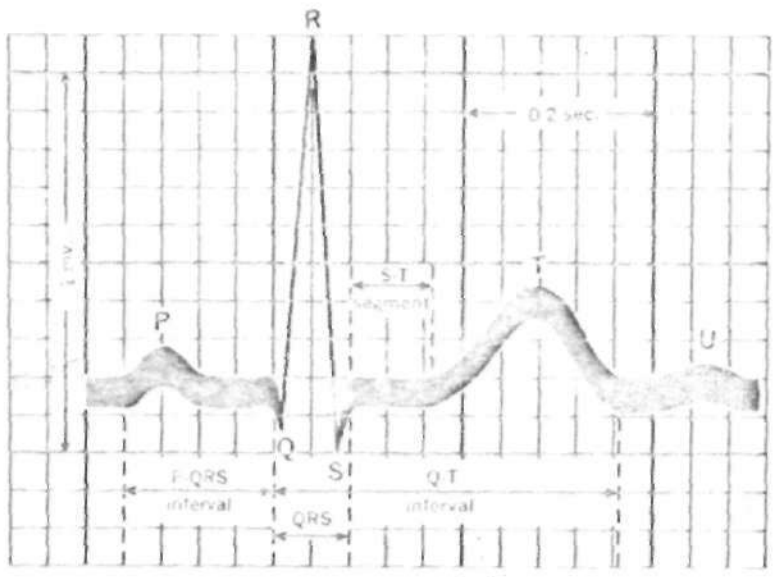


Fig. 2a: Conventional terms for electrocardiographic deflections and durations. (From Breazile, J. E.: In: Veterinary Physiology. Lea & Febiger, Philadelphia, 1971).

Table 2a: Criteria for determining the normal canine electrocardiogram*

Heart rate: 70 to 160 beats/min for adult dogs; up to 180 beats/min in toy breeds, and 220 beats/min for puppies.

Heart rhythm: Normal sinus rhythm; sinus arrhythmia; and wandering sinoatrial pacemaker.

Pwave: Up to 0.4 mV in amplitude; up to 0.04 sec in duration; always positive in leads II and aVF; positive or isoelectric in lead I.

P-Q interval: 0.06 to 0.13 sec duration.

QRS complex: Mean electrical axis, frontal plane, 40° to 100°

Amplitude - Maximum amplitude of R wave 2.5 to 3.0 mV in leads II, III and aVF.

Complex-positive in leads II, III and aVF.

Duration - To 0.05 sec (0.06 sec in large breeds)

S-T segment and T wave: S-T segment free of marked coving

*Source: Ettinger, S. J. and Suter, P. F. (1970). Canine cardiology. Saunders and Co., Philadelphia.

Results

The voltages and durations of the various ECG wave forms as recorded in the different species studied are summarised in Tables 2c to 2p. The value zero, as shown in Tables 2c through 2i, is indicated for those waves that failed to appear in some leads. For example the QS or R' wave in lead II was absent in healthy dogs but present in apparently healthy cattle. The P wave was variably positive and negative in some leads. However, it was consistently positive in leads I in dogs and horses ; II in dogs, cats, cattle, horses, donkeys; III in cats, cattle; aVF in cats, goats, cattle, donkeys; CV6LL in dogs, sheep, cattle, horses, donkeys; V2 in dogs, goats, cattle, donkeys, and CV5RL in cattle and horses.

Similarly, the T wave was either negative or positive in all leads recorded except in the donkey in which the T wave was consistently positive in leads III and V10.

In all species except for the R wave all other waves may or may not appear regardless of age or sex. However, the R wave was usually present in some leads in the following animal species: dog- I, II, III, aVR, aVF, and the precordial leads; cat- II, III, aVR, aVF, CV6LL, CV5RL, rV2; sheep- I, III, aVR, aVF, V10; goat- III, aVR, V10;

cattle- V10, CV6LL, CV5RL, rV2; horse- aVR, CV5RL, rV2; and the donkey- III, aVR, V10, CV5RL, rV2 .

Of the durations computed (Tables 2j to 2p), except in cattle, horses and donkeys, P wave duration was the shortest, QT interval was the longest, and QRS duration was intermediate between these two. The QRS duration was usually longer than that of the P wave but shorter than the PQ interval. In cattle and horses, QRS duration was the shortest; in the donkey, the P or QRS duration manifested as the shortest. A general pattern of progressive increase was evident with respect to mean duration and range from the smaller animals (dogs, cats) through sheep and goats to the larger animals (cattle, donkeys and horses). The voltages did not show a corresponding pattern.

Typical ECG patterns for each species studied as recorded in the different leads have been reproduced in Figs 2e to 2j . Generally, in dogs and cats the major positive deflections occurred in aVF as well as the SLLs and UPLs. In the other species because of the more frequent occurrence of the QS complex in several leads, the major deflections were quite often negative.

ECG tracings from dogs with induced myocardial

disorders featured such wave abnormalities as changes in P and R amplitudes, and Q depth; QS complexes in SLLs; and T inversion (Fig. 2k) and arrow- headed T; ST segment elevation; paroxysmal ventricular tachycardia (Fig. 2l).

Pentobarbitone caused increased amplitude in P and R waves, decreased T wave depth, and slight tachycardia (Fig 2n). Prolonged QRS duration and QT interval as well as marked bradycardia due to carbachol were normalized following atropine antidotal therapy (Fig. 2m). Reflex tachycardia due to chlorpromazine (Fig. 2o), and paroxysmal ventricular tachycardia due to digoxin (Fig. 2p) were also reproduced.

Discussion

In normal dogs QS complexes with or without R'wave were not observed except in leads aVL and V10. The appearance of such complex in other leads could therefore be of clinical significance in the recognition of cardiac disease in dog. In the ruminants and equines the frequently recorded QS complex in several leads (Fig. 2i) is normal and may be due to the mechanism of ventricular activation and topographical anatomy of the heart within the thorax (Breazile, 1971). Low voltages were commonly recorded in the adult ruminant and equines but not necessarily so in adult dogs and cats. Such low voltages (in adult ruminants and horses) are attributed to the highly synchronised ventricular depolarisation and the fact that the average direction of depolarisation is almost perpendicular to the planes of the leads. Conspicuously high voltages in any lead from all species may signal cardiac disorder. In dogs and cats abnormally high voltages (>3.0 mV) in any lead are considered pathologic, and are usually found in association with cadiac hypertrophy. Unusually low voltages recorded from all leads indicate pericarditis, extracardiac or thoracic masses (Ettinger and Suter, 1970).

The Data summarised in Tables 2c through 2p were obtained from clinically normal animals in the conscious state except in the cat where general anaesthesia was necessary. The use of conscious animals in the study has advantage over the previous studies by Burman et al.(1966), Crawley and Swenson (1966), McCrady et al.(1966) using chemical restraint in form of sedatives or anaesthetics, some of which are known to cause significant effects on the ECG (Ettinger and Suter, 1970; Hamlin, 1976). Nevertheless, the ECG voltages and durations established in this study are quite similar to those previously reported for the respective species (Ettinger and Suter, 1970; Breazile, 1971; Hamlin, 1976). Therefore, established ECG criteria for recognition of cardiac disorders (Ettinger and Suter, 1970; Hamlin, 1976) especially in relation to dogs and cats are not contradicted by this study and could cautiously be applied to Nigerian indigenous animals.

Currently, the criteria in use for electrocardiographic diagnosis of cardiac disorders include abnormal wave appearance or disappearance as in cardiac fibrillation (Fig. 21(d)); abnormality in wave form, voltage or duration exemplified by patterns obtained in myocardial

injury (Figs 2k & 2l), and digitalis-induced cardiac arrhythmias (Fig. 2p).

In this study the wide range in the voltages and durations obtained are regarded as normal because of wide variation in the ages, degree of calmness during recording and the incomplete breed characterisation of the indigenous animals used, especially dogs and cats.

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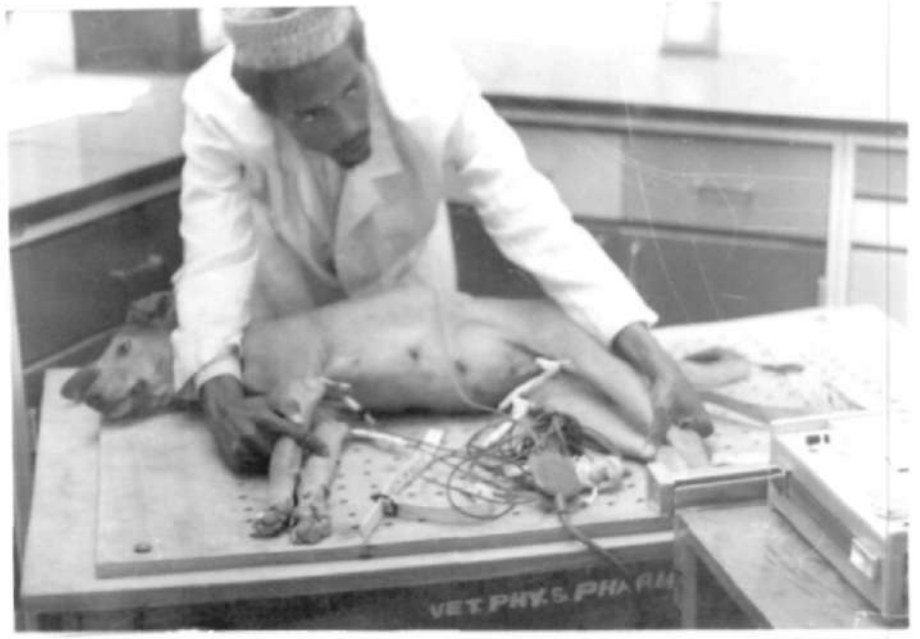


Fig. 2b: Positioning and restraint procedures in recording the electrocardiogram from a conscious dog.

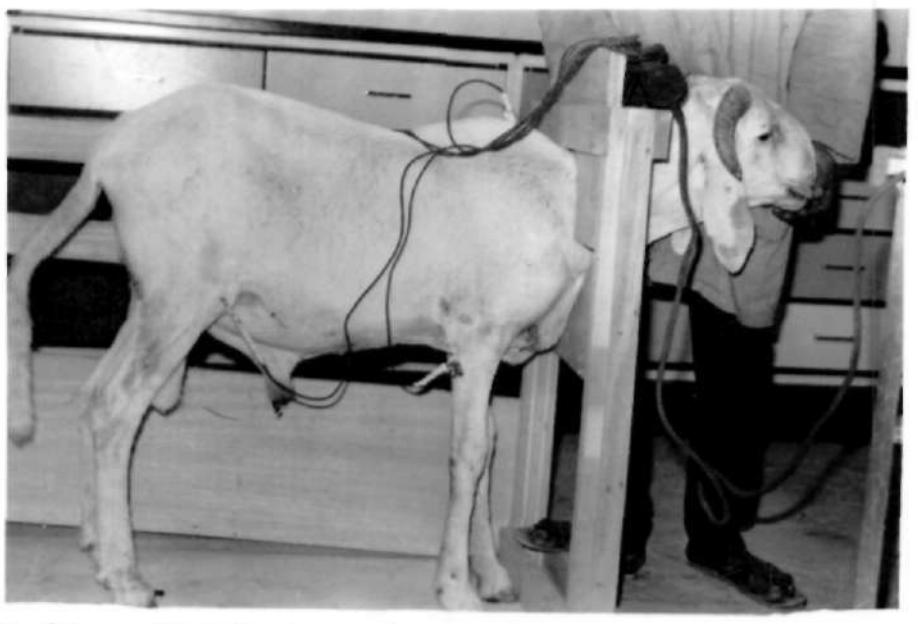


Fig. 2c: Positioning and restraint procedures in recording the electrocardiogram of a ram.

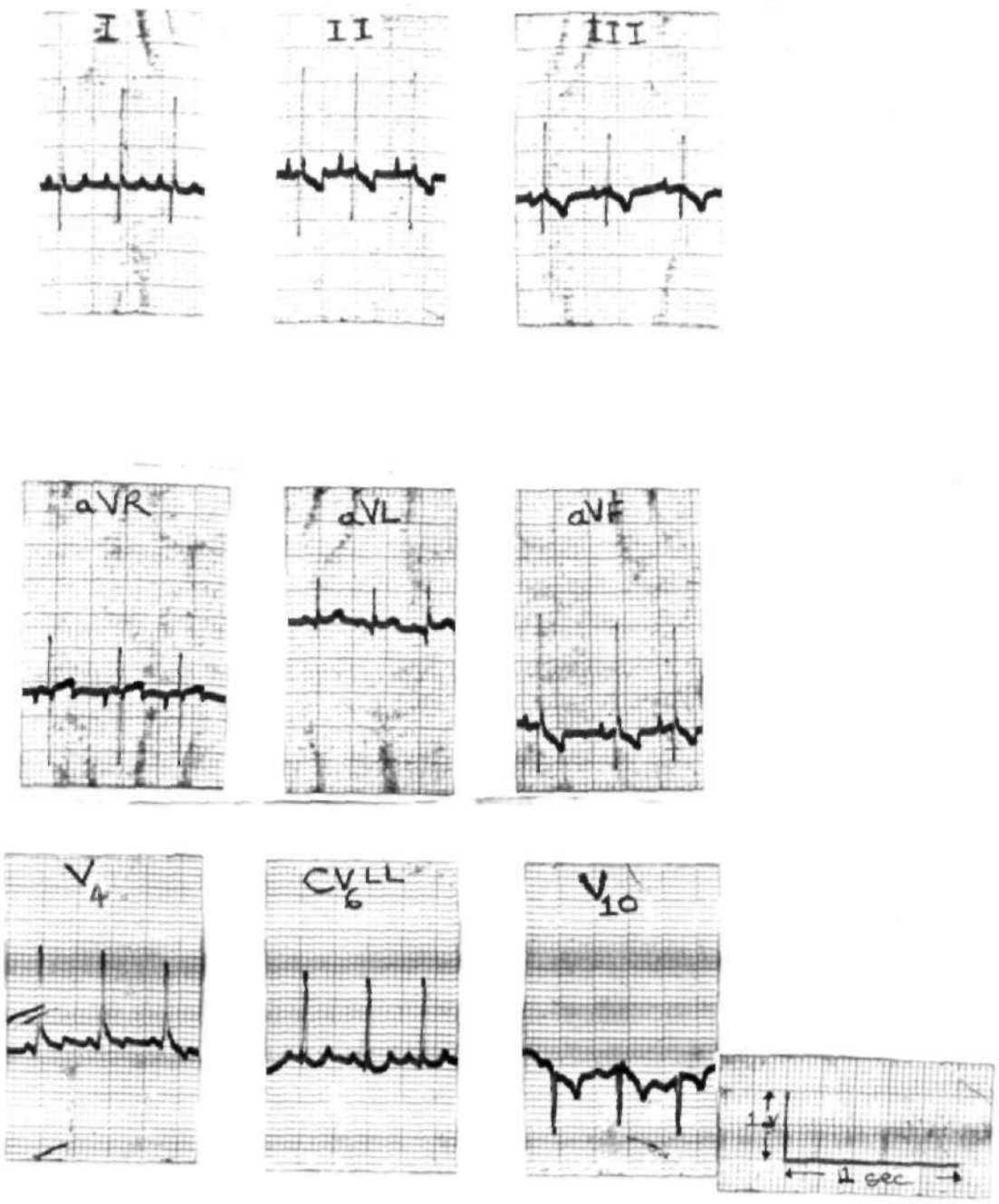


Fig. 2d: Electrocardiograms of a normal 2-year-old local dog. Mean electrical axis in the frontal plane is approximately 65 degrees. Heart rate (approximately 180 beats/min) and rhythm are normal.

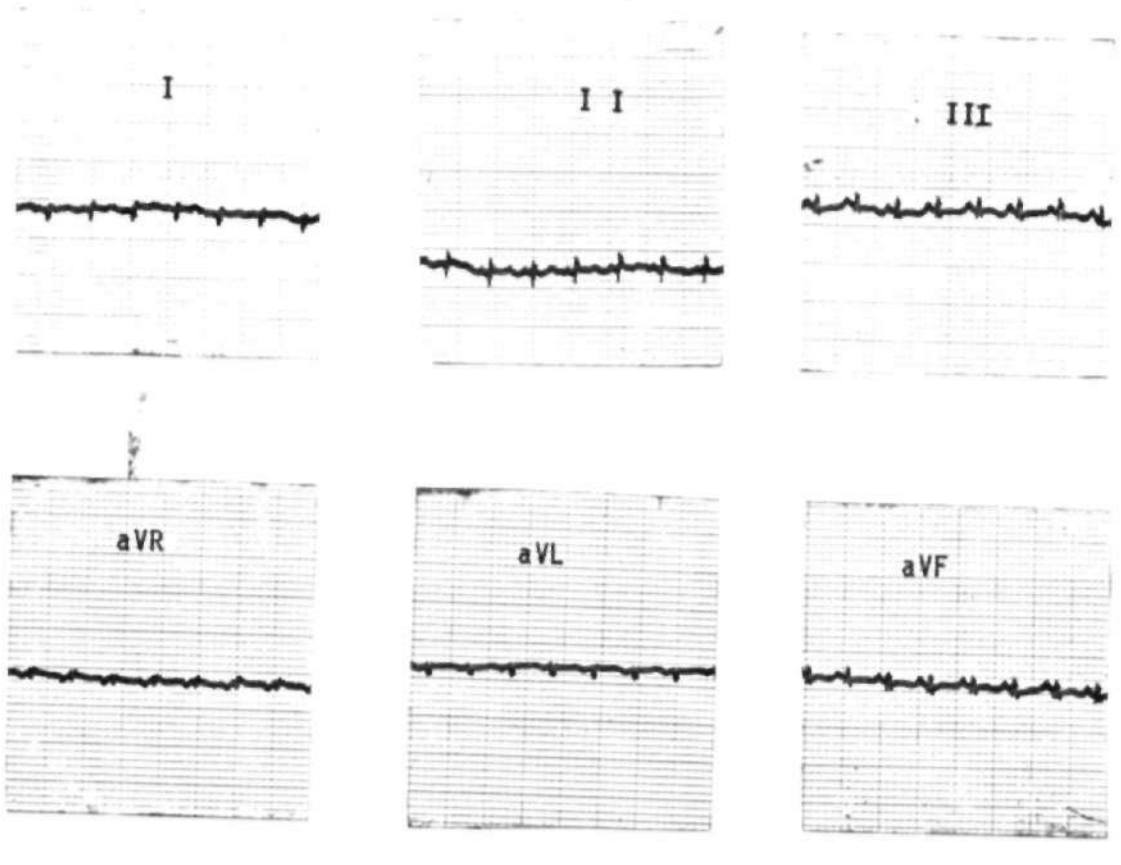


Fig. 2e: Electrocardiograms of the normal 1½-year old local tomcat. Mean electrical axis in the frontal plane is approximately 160 degrees. Heart rate (approximately 270 beats/ min) and rhythm are normal.

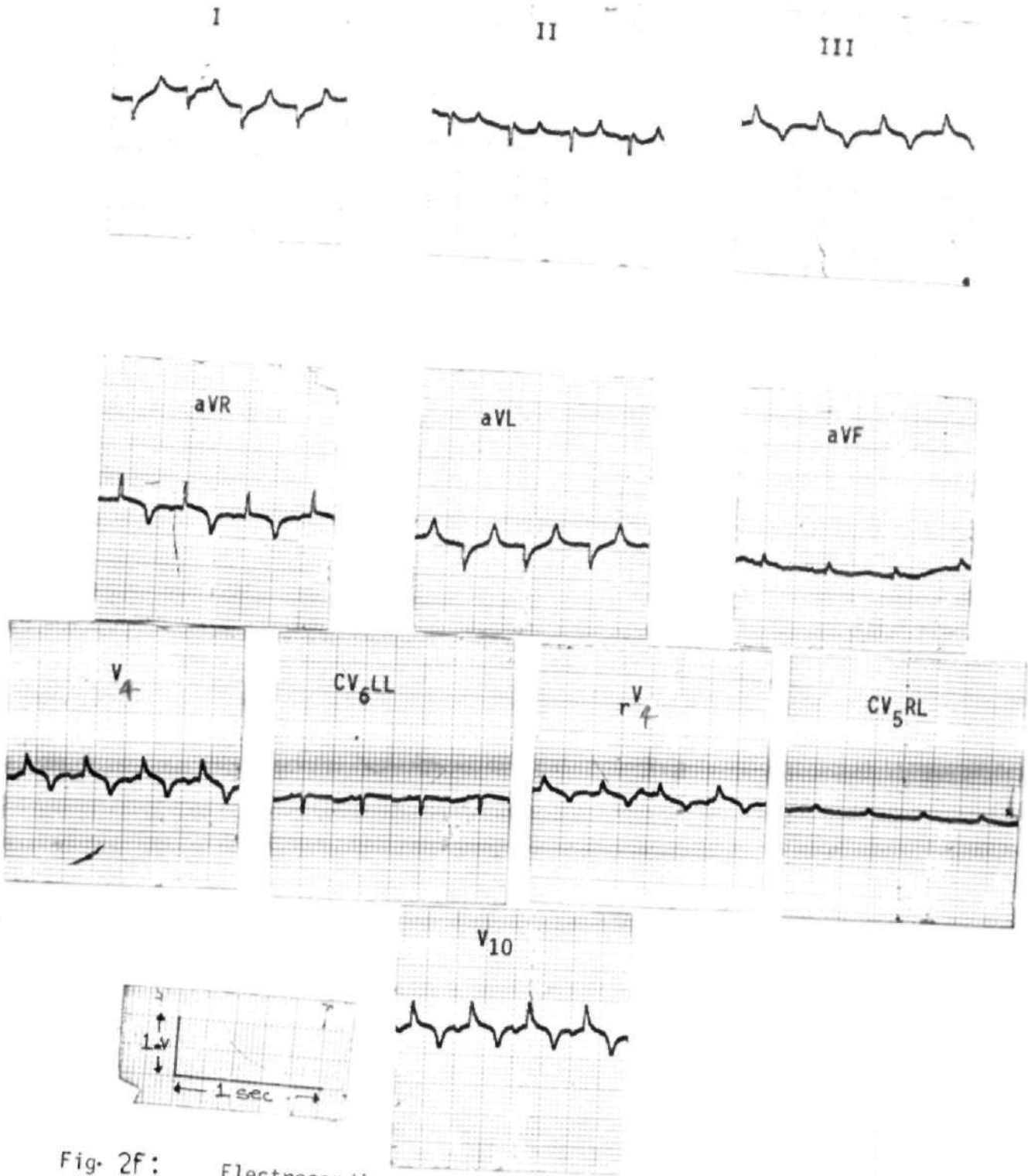


Fig. 2f: Electrocardiograms of a normal 1½-year-old Yankassa ram. Mean electrical axis in the frontal plane is approximately 145 degrees. Heart rate (approximately 180 beats/min) and rhythm are normal.

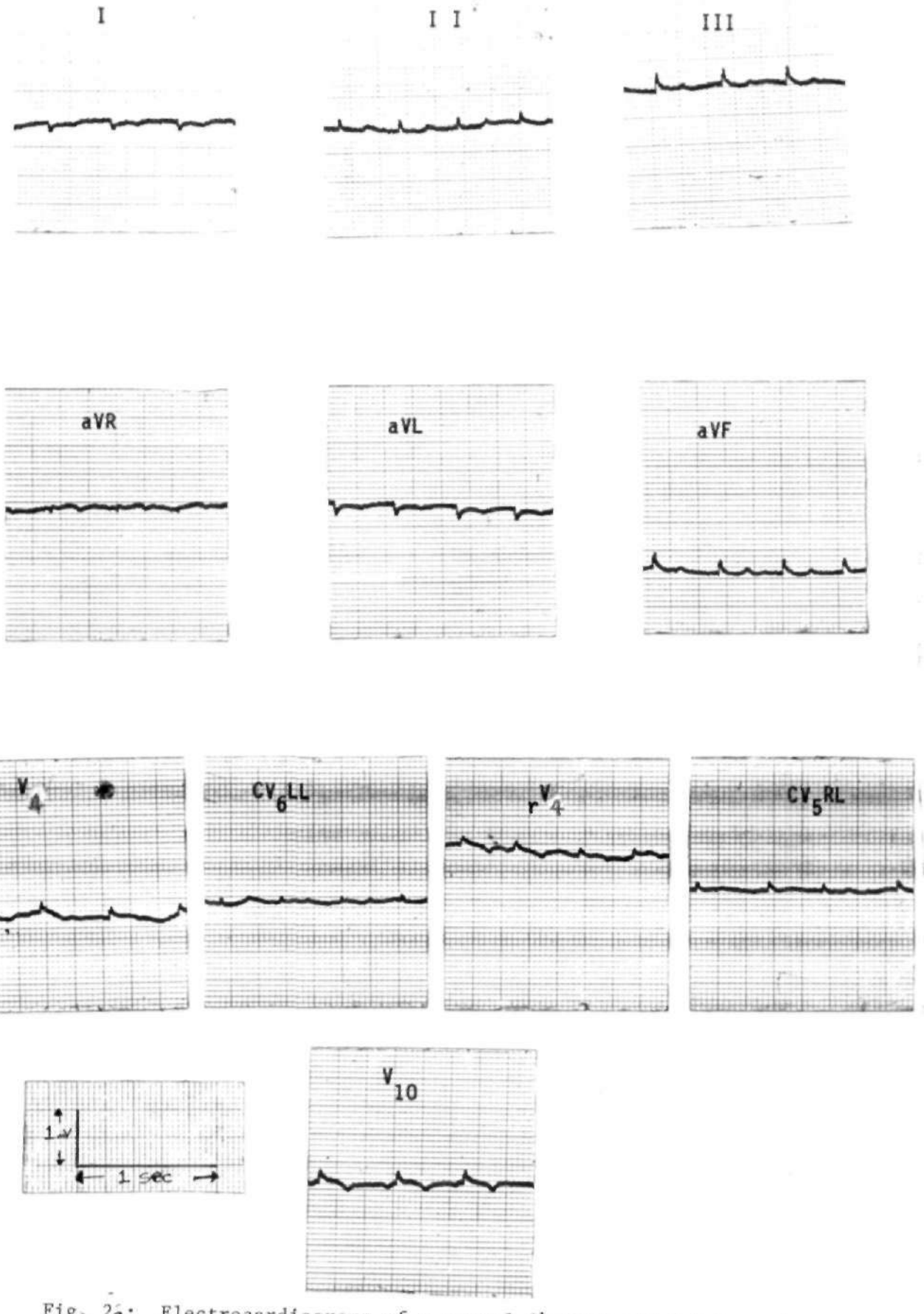


Fig. 29: Electrocardiograms of a normal 1½-year-old Sokoto red goat. Mean electrical axis in the frontal plane is approximately 115 degrees. Heart rate (approximately 110 beats/min) and rhythm are normal.

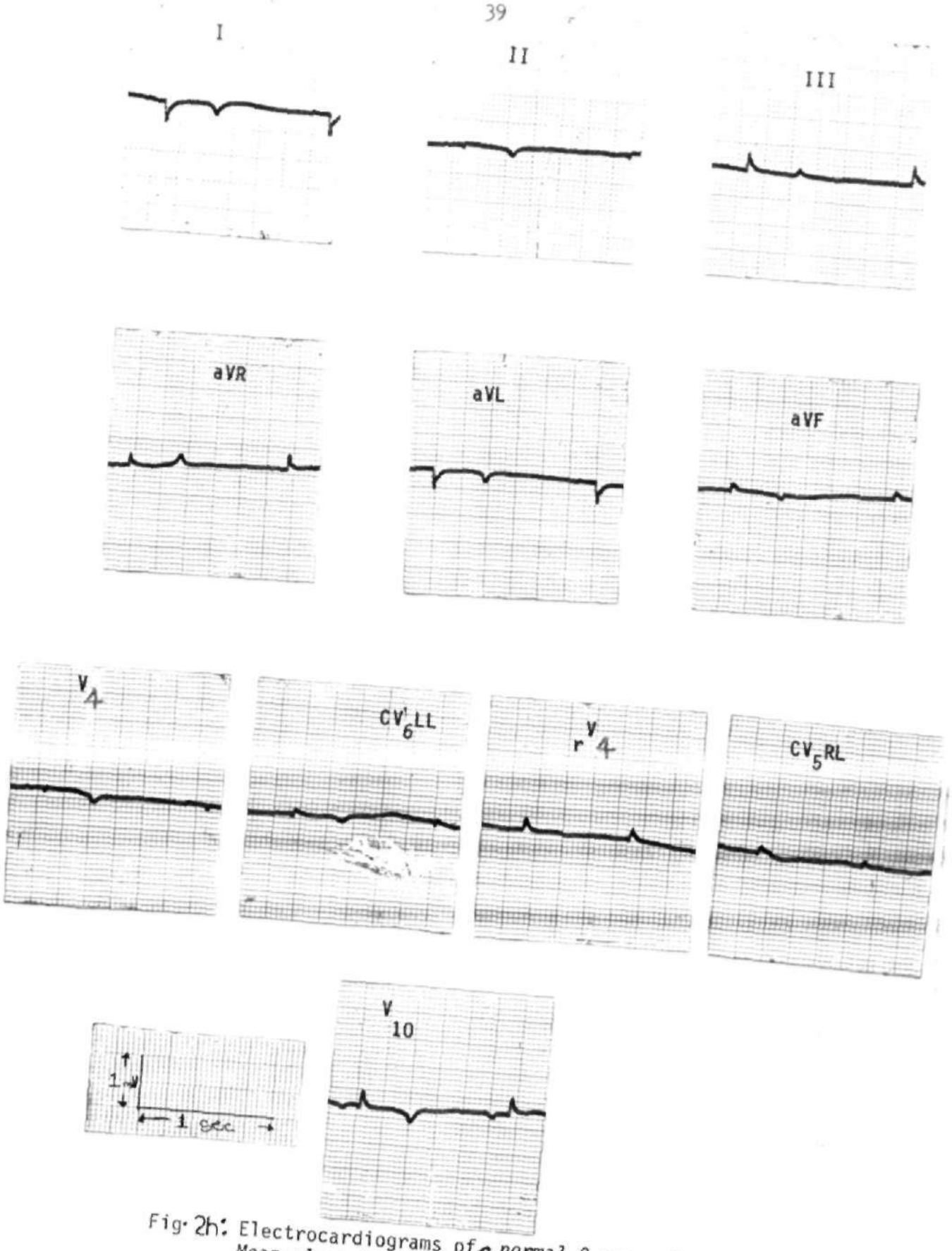


Fig. 2h: Electrocardiograms of a normal 2-year-old White fulani cow. Mean electrical axis in the frontal plane is approximately 165 degrees. Heart rate (approximately 30 beats/min) and rhythm are normal.

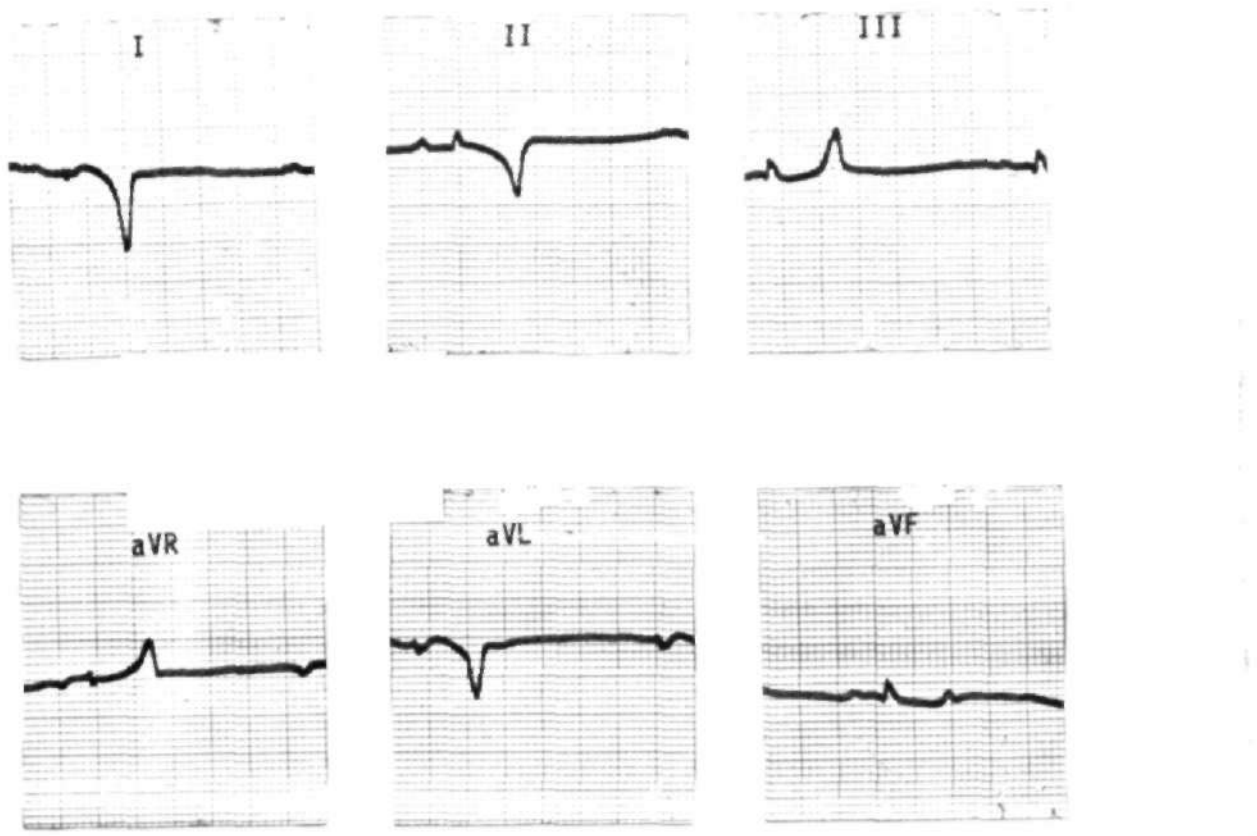




Fig. 2i: Electrocardiograms of normal 4-year-old local horse. Mean electrical axis in the frontal plane is approximately 120 degrees. Heart rate (approximately 20 beats/min) and rhythm are normal.

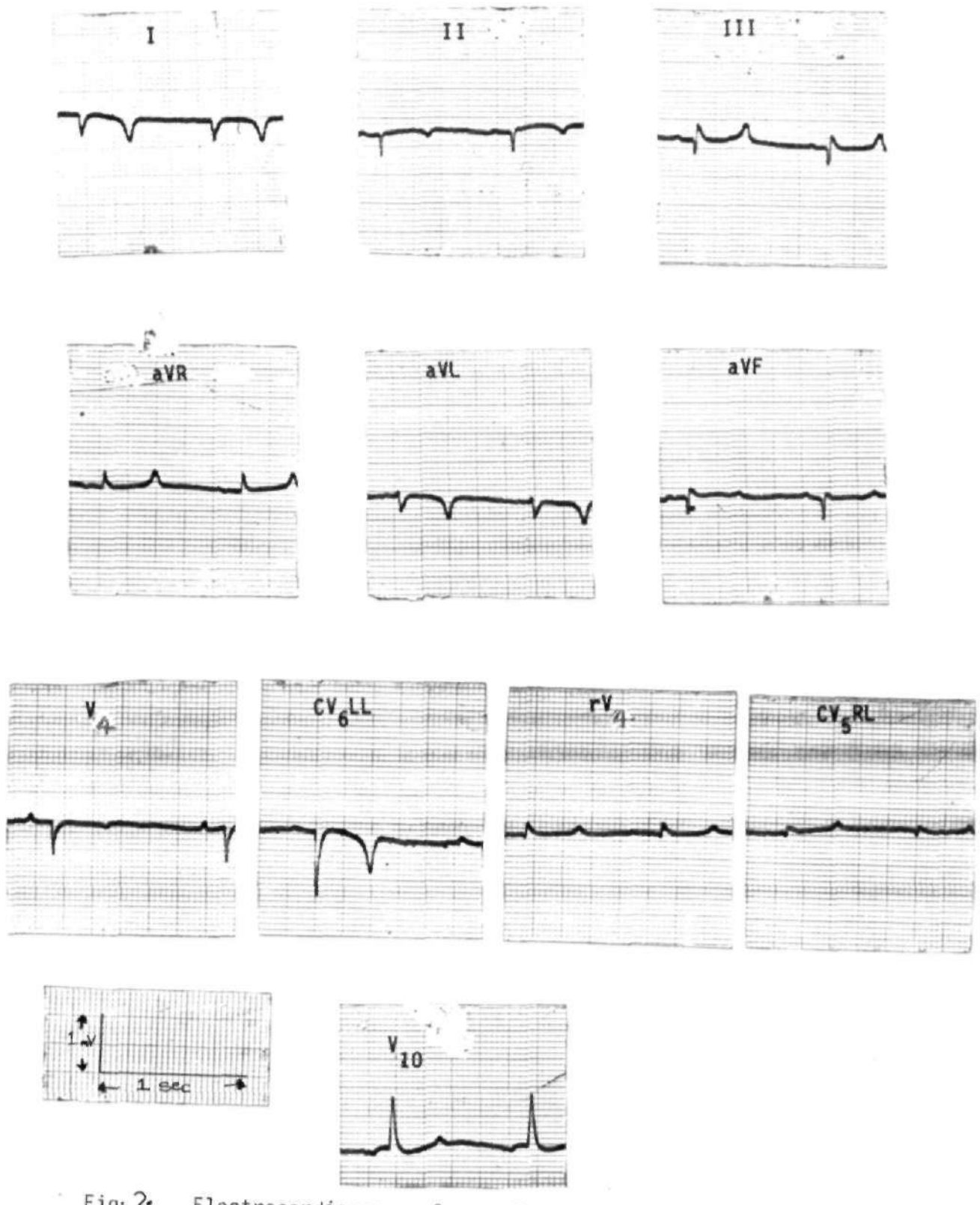


Fig. 2j Electrocardiograms of normal 1½-year-old local donkey.Mean electrical axis in the frontal plane is approximately 160 degrees. Heart rate (approximatelely 45 beats/min) and rhythm are normal.

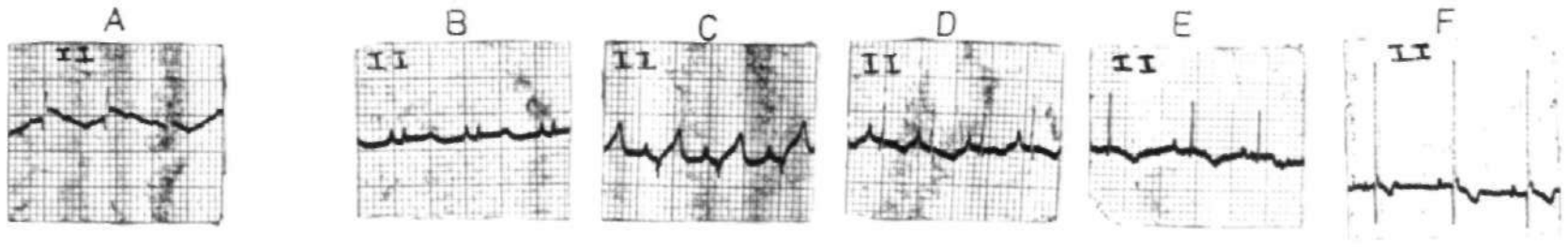


Fig. 2k: Sequential electrocardiographic changes of myocardial injury in the dog.

A: Normal tracing; preocclusion.

B First minute post-occlusion of middle coronary arterial branch. Note marked increase and decrease in P and R waves amplitude respectively. The Q wave amplitude is also markedly reduced and inversion of T wave is present.

C: First minute of reperfusion injury caused the appearance of QS complexes and tall, arrow-head T waves.

D: After an hour of reperfusion. Except for the unusually peaked P wave the record is fairly normal.

E: A day after reperfusion. Note increase in R amplitude.

F: Twenty days after reperfusion. Note distinct increase in R and Q waves amplitudes. Deep Q waves are highly suggestive of myocardial infarction.

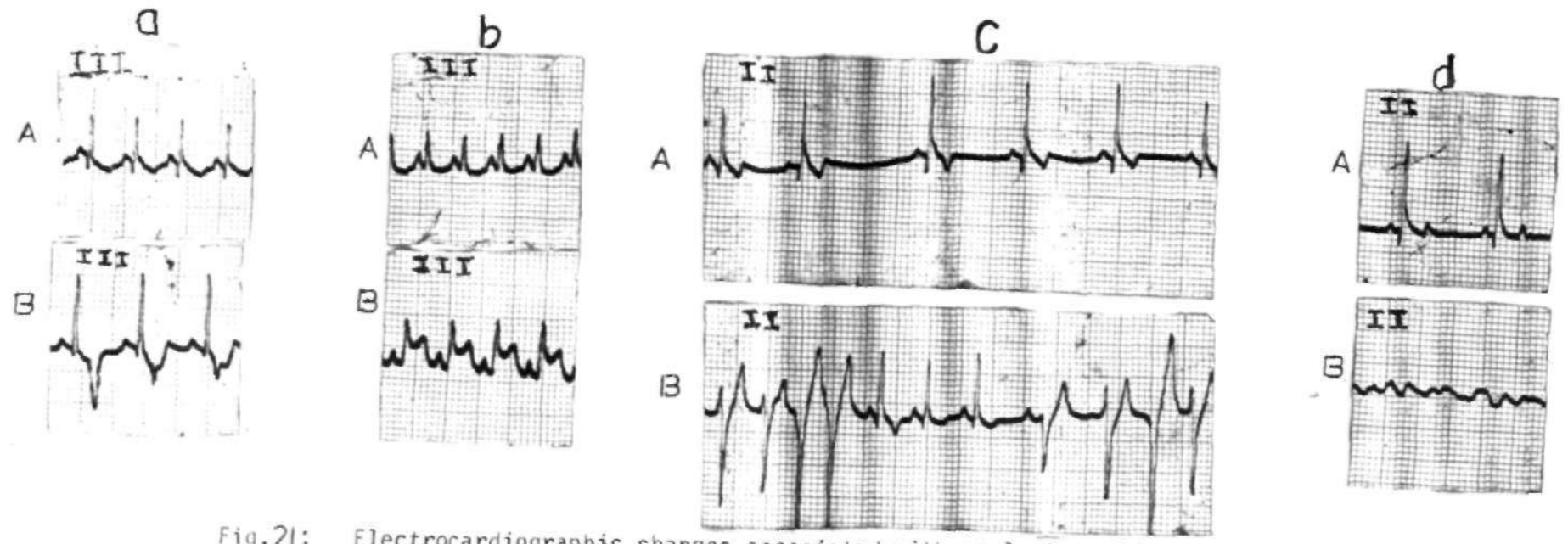


Fig.21: Electrocardiographic changes associated with occlusion of left anterior descending coronary artery in the dog.

a: Acute T wave changes. Tracing in the upper portion (A) is normal. The lower portion shows occurrence of deep, arrow-head T waves after five minutes of the occlusion. R wave amplitude is also increased in the presence of slight bradycardia.

b: ST segment changes. Tracing in the upper portion (A) is normal. The lower portion tracing (B) obtained five minutes after the occlusion shows ST segment elevation.

c: Ventricular tachycardia. Tracing in the upper portion (A) is normal. Twenty-four hours after the occlusion paroxysms of ventricular tachycardia interrupted the normal rhythm.

d: Ventricular fibrillation in the lower tracing (B) replaced the normal rhythm (A). This was acutely produced following occlusion in the most proximal region of the artery.

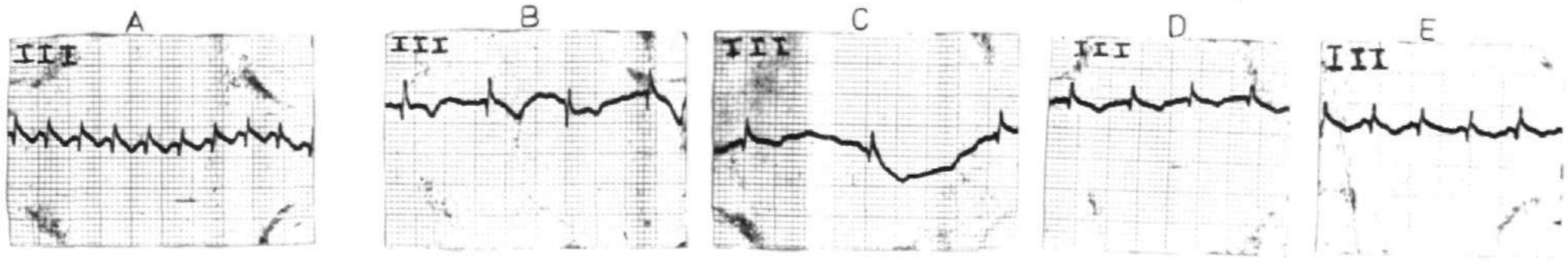


Fig. 2m: Electrocardiographic changes due to drugs in the cat.
A: Pre-carbachol electrocardiogram.

B: Ten minutes after carbachol (0.50 mg/kg, SC).
Note bradycardia, prolonged PQ and QT intervals as well as T wave changes.

C: Twenty-five minutes of carbachol administration. Note exaggeration of effects mentioned in Fig 2m(B). During this stage the cat urinated, salivated profusely and was severely dyspnoeic.

D: Antidotal therapy with atropine (0.50 mg/kg, IM) followed within a minute of abnormalities mentioned in Fig 2m(C). Note increased rate and decreased PQ and QT intervals.

E: An hour following antidotal therapy. Note further improvement in rate.

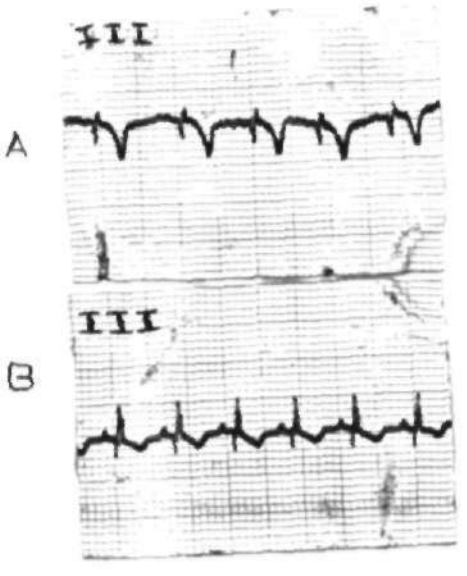


Fig. 20: Electrocardiographic changes due to pentobarbital sodium (30 mg/kg, IV). in a dog.. Upper tracing (A) is normal. The lower tracing (B) in anesthesia shows marked reduction in T wave depth, increased R wave amplitude and accelerated rate.

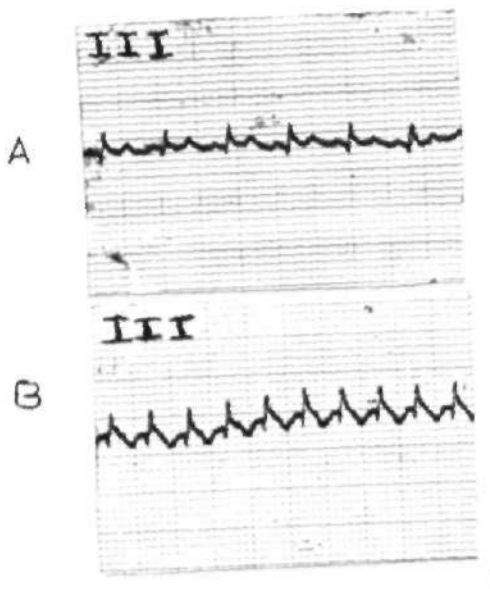


Fig. 20: Electrocardiographic changes due to chlorpromazine in the cat. In the upper tracing (A) is a normal rhythm. The lower tracing (B) shows changes recorded after five minutes of chlorpromazine (1 mg/kg, IM) administration. Reflex tachycardia and T wave inversion were evident.

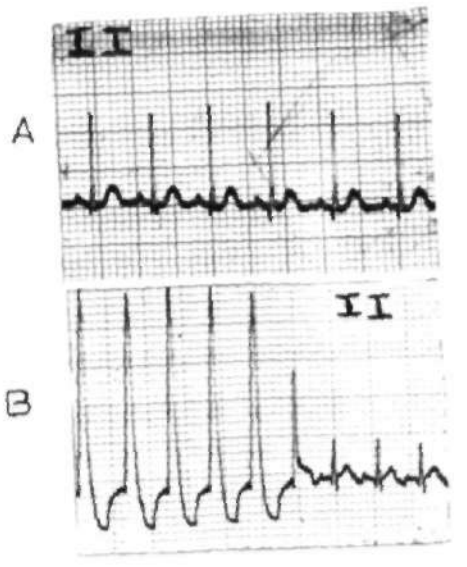


Fig. 2p: Electrocardiographic changes due to digitalis toxicity. In the upper tracing (A) is shown the electrocardiogram of a 3-year-old Dalmatian bitch with marked ascites and depression on presentation. It was immediately started on digoxin (0.20 mg/kg, POS), and on the third day of starting the maintenance dose (0.05 mg/kg, POS) paroxysmal ventricular tachycardia(B) was observed. Following withdrawal of digoxin, normal rhythm was re-established after 2 days.

Table 2b Total number and breed of animals used for each species in determining normal electrocardiographic patterns

Animal	Total No.
Dog (Local)	71
Cat (Local)	21
Sheep (Yankassa)	24
Goat (Sokoto red)	40
Cattle (White-Fulani)	28
Horse (Local)	22
Donkey (Local)	19

Table 2C: Electrocardiogram of clinically healthy Dogs: Amplitudes of the various wave forms in different leads.

Lead	Mean ± SD (Range) in millivolts; n = 71						
	P	Q	R	S	T	QS	R'
I	0.079 ± 0.041 (0.01 to 0.20)	0.180 ± 0.144 (0.00 to 0.70)	0.506 ± 0.304 (0.05 to 1.40)	0.140 ± 0.105 (0.00 to 0.250)	-0.049 ± 0.137 (-0.30 to 0.20)	0	0
II	0.146 ± 0.060 (0.000 to 0.300)	0.300 ±0. 210 (0.000 to 0.900)	0.944 ± 0.428 (0.300 to 2.000)	0.175 ± 0.101 (0.000 to 0.3000)	-0.082 ± 152 (-0.600 to 0.200)	0	0
III	0.099 ± 0.084 (-0.100 to 0.250)	0.162 ± 0.157 (0.000 to 0.630)	0.631 ± 0.353 (0.200 to 1.400)	0.207 ± 0.097 (0.000 to 0.400)	-0.059 ± 0.163 (-0.400 to 0.300)	0	0
aVR	-0.097 ± 0.047 (-0.200 to 0.150)	0	0.220 ± 0.164 (0.020 to 0.700)	0.672 ± 0.337 (0.200 to 1.700)	0.094 ± 0.160 (-0.200 to 0.400)	0	0
aVL	-0.019 ± 0.080 (-0.100 to 0.150)	0.076 ± 0.048 (0.000 to 0.200)	0.283 ± 0.244 (0.000 to 0.900)	0.280 ± 0.182 (0.000 to 0.650)	-0.016 ± 0.128 (-0.250 to 0.200)	0.307 ± 0.109 (0.000 to 0.500)	0.180 ± 0.135 (0.000 to 0.400)
aVF	0.120 ± 0.062 (-0.100 to 0.220)	0.260 ± 0.184 (0.000 to 0.800)	0.762 ± 0.399 (0.250 to 1.700)	0.210 ± 0.089 (0.000 to 0.350)	-0.076 ±0.195 (-0.600 to 0.250)	0	0
V_2	0.094 ± 0.035 (0.000 to 0.180)	0.139 ± 0.080 (0.000 to 0.300)	0.677 ± 0.404 (0.200 to 1.600)	0	-0.094 ± 0.098 (-0.200 to 0.130)	0	0
CV_6LL	0.090 ± 0.026 (0.000 to 0.120)	0.066 ± 0.032 (0.000 to 0.100)	0.687 ± 0.311 (0.350 to 1. 00)	0	-0.111 ± 0.095 (-0.200 to 0.100)	0	0
V_{10}	-0.065 ± 0.067 (-0.200 to 0.100)	0.050 ± 0.002 (0.000 to 0.072)	0.094 ± 0.035 (0.000 to 0.400)	0.542 ± 0.141 (0.000 to 0.700)	-0.191 ± 0.134 (-0.400 to 0.100)	0.676 ± 0.326 (0.000 to 1.500)	0.137 ± 0.091 (0.000 to 0.300

Table 2d: Electrocardiogram of clinically healthy Cats: Amplitudes of the various wave forms in different leads.

Lead	Mean ± SD (Range) in millivolts; n = 21						
	P	Q	R	S	T	QS	R'
I	0.101 ± 0.104 (-0.02 to 0.50)	0.035 ± 0.012 (0.00 to 0.50)	0.188 ± 0.180 (0.00 to 0.50)	0.072 ± 0.063 (0.00 to 0.150)	0.016 ± 0.082 (-0.20 to 0.10)	0.183 ± 0.022 (0.00 to 0.20)	0.035 ± 0.020 (0.00 to 0.050)
II	0.105 ± 0.016 (0.000 to 0.150)	0.052 ± 0.044 (0.000 to 0.150)	0.250 ± 0.141 (0.050 to 0.500)	0.032 ± 0.016 (0.000 to 0.050)	0.051 ± 0.162 (-0.300 to 0.250)	0	0
III	0.082 ± 0.016 (0.000 to 0.100)	0.046 ± 0.024 (0.000 to 0.100)	0.192 ± 0.080 (0.080 to 0.300)	0.065 ± 0.059 (0.000 to 0.150)	0.043 ± 0.109 (-0.200 to 0.180)	0	0
aVR	-0.078 ± 0.026 (-0.100 to 0.050)	0	0.057 ± 0.030 (0.020 to 0.150)	0.183 ± 0.159 (0.020 to 0.400)	-0.048 ± 0.101 (-0.150 to 0.200)	0	0
aVL	-0.013 ± 0.052 (-0.100 to 0.050)	0.036 ± 0.013 (0.000 to 0.050)	0.075 ± 0.046 (0.000 to 0.150)	0.112 ± 0.037 (0.000 to 0.150)	0.026 ± 0.075 (-0.060 to 0.120)	0.125 ± 0.025 (0.000 to 0.150)	0.085 ± 0.041 (0.00 to 0.150)
aVF	0.081 ± 0.018 (0.000 to 0.100)	0.053 ± 0.026 (0.000 to 0.100)	0.227 ± 0.114 (0.100 to 0.400)	0.059 ± 0.042 (0.000 to 0.120)	0.065 ± 0.112 (-0.150 to 0.250)	0	0
V_4	0.024 ± 0.49 (-0.050 to 0.100)	0.030 ± 0.017 (0.000 to 0.050)	0.268 ± 0.260 (0.000 to 0.700)	0.081 ± 0.064 (0.000 to 0.150)	0.050 ± 0.125 (-0.200 to 0.200)	0.150 ± 0.012 (0.000 to 0.200)	0
CV_6LL	0.010 ± 0.040 (-0.050 to 0.050)	0	0.125 ± 0.065 (0.020 to 0.200)	0.158 ± 0.091 (0.050 to 0.300)	0.078 ± 0.084 (-0.060 to 0.200)	0	0
rV_4	0.014 ± 0.075 (-0.080 to 0.100)	0.075 ± 0.024 (0.000 to 0.100)	0.125 ± 0.024 (0.050 to 0.150)	0	-0.048 ± 0.012 (-0.100 to 0.060)	0	0
CV_5RL	0.038 ± 0.054 (-0.050 to 0.100)	0.030 ± 0.026 (0.000 to 0.060)	0.130 ± 0.063 (0.050 to 0.200)	0.040 ± 0.010 (0.000 to 0.050)	-0.059 ± 0.038 (-0.100 to 0.050)	0	0
V_{10}	-0.071 ± 0.024 (-0.100 to 0.050)	0.100 ± 0.050 (0.000 to 0.180)	0.075 ± 0.035 (0.000 to 0.150)	0	-0.122 ± 0.053 (-0.200 to -0.030)	0.250 ± 0.050 (0.000 to 0.300)	0.105 ± 0.061 (0.000 to 0.200)

Table 20: Electrocardiogram of clinically healthy Sheep: Amplitudes of the various wave forms in different leads.

Lead	Mean ± SD (Range) in millivolts; n = 24						
	P	Q	R	S	T	QS	R'
I	0.014 ± 0.038 (-0.050 to 0.100)	0	0.030 ± 0.020 (0.010 to 0.060)	0.261 ± 0.112 (0.000 to 0.400)	0.051 ± 0.131 (-0.200 to 0.300)	0.304 ± 0.252 (0.010 to 0.700)	0
II	0.041 ± 0.058 (-0.100 to 0.100)	0.043 ± 0.010 (0.000 to 0.060)	0.200 ± 0.086 (0.000 to 0.300)	0	-0.045 ± 0.102 (-0.150 to 0.100)	0.138 ± 0.041 (0.000 to 0.200)	0.067 ± 0.31 (0.000 to 0.100)
III	0.003 ± 0.040 (-0.050 to 0.060)	0.022 ± 0.008 (0.000 to 0.040)	0.298 ± 0.142 (0.100 to 0.600)	0	-0.024 ± 0.124 (-0.200 to 0.120)	0	0
aVR	-0.007 ± 0.050 (-0.060 to 0.100)	0.021 ± 0.006 (0.000 to 0.030)	0.179 ± 0.112 (0.050 to 0.400)	0.043 ± 0.002 (0.040 to 0.050)	-0.018 ± 0.140 (-0.200 to 0.200)	0	0
aVL	0.017 ± 0.040 (-0.030 to 0.100)	0	0.024 ± 0.005 (0.000 to 0.030)	0.138 ± 0.060 (0.000 to 0.200)	0.023 ± 0.109 (-0.150 to 0.200)	0.321 ± 0.186 (0.000 to 0.700)	0
aVF	0.023 ± 0.025 (-0.030 to 0.050)	0.030 ± 0.018 (0.000 to 0.050)	0.211 ± 0.113 (0.000 to 0.400)	0	0.046 ± 0.021 (-0.200 to 0.070)	0.106 ± 0.011 (0.000 to 0.120)	0.080 ± 0.079 (0.000 to 0.17)
V_4	0.022 ± 0.031 (0.050 to 0.060)	0.023 ± 0.009 (0.000 to 0.040)	0.176 ± 0.136 (0.000 to 0.040)	0	0.048 ± 0.038 (-0.3000 to 0.100)	0.100 ± 0.40 (0.000 to 0.150)	0.066 ± 0.028 (0.000 to 0.100)
CV_6LL	0.030 ± 0.010 (0.000 to 0.040)	0	0	0	0.007 ± 0.052 (-0.070 to 0.060)	0.083 ± 0.027 (0.050 to 0.150)	0.030 ± 0.010 (0.020 to 0.040)
V_{10}	-0.071 ± 0.027 (-0.100 to 0.030)	0.027 ± 0.020 (0.000 to 0.080)	0.391 ± 0.136 (0.200 to 0.600)	0	-0.176 ± 0.103 (-0.400 to -0.100)	0	0

Table 2f: Electrocardiogram of clinically healthy Goats: Amplitudes of the various wave forms in different leads.

Lead	Mean ± SD (Range) in millivolts; n = 40						
	P	Q	R	S	T	QS	R'
I	0.042 ± 0.030 (-0.020 to 0.100)	0	0.060 ± 0.010 (0.000 to 0.100)	0.225 ± 0.150 (0.000 to 0.400)	0.097 ± 0.165 (-0.200 to 0.400)	0.276 ± 0.106 (0.000 to 0.500)	0
II	0.041 ± 0.031 (-0.050 to 0.100)	0.034 ± 0.017 (0.000 to 0.070)	0.083 ± 0.062 (0.000 to 0.200)	0	0.097 ± 0.135 (-0.150 to 0.300)	0.218 ± 0.064 (0.000 to 0.300)	0.070 ± 0.010 (0.000 to 0.080)
III	0.028 ± 0.031 (-0.050 to 0.100)	0.032 ± 0.021 (0.000 to 0.100)	0.236 ± 0.116 (0.020 to 0.450)	0.275 ± 0.076 (0.150 to 0.400)	0.013 ± 0.101 (-0.150 to 0.200)	0	0
aVR	-0.034 ± 0.048 (-0.100 to 0.040)	0.024 ± 0.009 (0.000 to 0.050)	0.238 ± 0.099 (0.060 to 0.450)	0	-0.074 ± 0.141 (-0.300 to 0.120)	0.120 ± 0.072 (0.000 to 0.200)	0
aVL	0.017 ± 0.036 (-0.050 to 0.100)	0	0.042 ± 0.028 (0.000 to 0.100)	0.118 ± 0.067 (0.000 to 0.200)	0.039 ± 0.094 (-0.200 to 0.200)	0.276 ± 0.118 (0.100 to 0.500)	0
aVF	0.042 ± 0.023 (0.000 to 0.100)	0.029 ± 0.010 (0.000 to 0.050)	0.134 ± 0.089 (0.000 to 0.300)	0	0.076 ± 0.113 (-0.200 to 0.300)	0.190 ± 0.054 (0.000 to 0.250)	0.085 ± 0.044 (0.000 to 0.120)
V_4	0.041 ± 0.027 (0.000 to 0.100)	0.048 ± 0.036 (0.000 to 0.120)	0.077 ± 0.047 (0.000 to 0.150)	0.533 ± 0.321 (0.000 to 0.900)	0.100 ± 0.111 (-0.100 to 0.300)	0.241 ± 0.190 (0.000 to 0.600)	0
V_{10}	-0.050 ± 0.047 (-0.100 to 0.060)	0.034 ± 0.025 (0.000 to 0.100)	0.265 ± 0.085 (0.100 to 0.400)	0	-0.091 ± 0.089 (-0.200 to 0.100)	0	0

Table 29: Electrocardiogram of clinically healthy Cattle: Amplitudes of the various wave forms in different leads.

Lead	Mean ± SD (Range) in millivolts; n = 28						
	P	Q	R	S	T	QS	R'
I	0.054 ± 0.047 (-0.050 to 0.150)	0.030 ± 0.020 (0.00 to 0.050)	0.078 ± 0.056 (0.000 to 0.150)	0.169 ± 0.157 (0.000 to 0.350)	-0.057 ± 0.107 (-0.200 to 0.100)	0.212 ± 0.144 (0.000 to 0.500)	0.030 ± 0.020 (0.000 to 0.050)
II	0.069 ± 0.039 (0.00 to 0.150)	0.036 ± 0.010 (0.000 to 0.050)	0.100 ± 0.070 (0.000 to 0.200)	0	-0.031 ± 0.142 (-0.200 to 0.300)	0.182 ± 0.147 (0.000 to 0.500)	0.082 ± 0.048 (0.000 to 0.150)
III	0.054 ± 0.035 (0.000 to 0.120)	0.019 ± 0.001 (0.000 to 0.020)	0.206 ± 0.154 (0.000 to 0.600)	0	0.030 ± 0.099 (-0.150 to 0.150)	0.282 ± 0.200 (0.000 to 0.700)	0.060 ± 0.014 (0.000 to 0.700)
aVR	-0.012 ± 0.085 (-0.150 to 0.160)	0.021 ± 0.012 (0.000 to 0.050)	0.176 ± 0.084 (0.000 to 0.300)	0.169 ± 0.149 (0.000 to 0.400)	0.113 ± 0.127 (-0.150 to 0.300)	0.080 ± 0.020 (0.000 to 0.100)	0
aVL	0.022 ± 0.054 (-0.100 to 0.100)	0.017 ± 0.003 (0.000 to 0.020)	0.133 ± 0.130 (0.000 to 0.400)	0.075 ± 0.061 (0.000 to 0.150)	-0.057 ± 0.118 (-0.200 to 0.150)	0.212 ± 0.205 (0.000 to 0.600)	0
aVF	0.052 ± 0.031 (0.000 to 0.100)	0.024 ± 0.011 (0.000 to 0.050)	0.148 ± 0.099 (0.000 to 0.400)	0	-0.035 ± 0.092 (-0.150 to 0.170)	0.174 ± 0.148 (0.000 to 0.400)	0.076 ± 0.020 (0.000 to 0.100)
V_4	0.070 ± 0.027 (0.000 to 0.100)	0	0.066 ± 0.028 (0.000 to 0.100)	0	-0.081 ± 0.118 (-0.200 to 0.070)	0.150 ± 0.040 (0.000 to 0.200)	0
CV_6LL	0.060 ± 0.036 (0.000 to 0.100)	0.057 ± 0.040 (0.000 to 0.100)	0.083 ± 0.008 (0.050 to 0.100)	0	-0.007 ± 0.100 (-0.100 to 0.100)	0	0
rV_4	0.012 ± 0.012 (-0.020 to 0.030)	0.019 ± 0.001 (0.000 to 0.020)	0.156 ± 0.043 (0.060 to 0.200)	0	-0.051 ± 0.068 (-0.120 to 0.080)	0	0
CV_5RL	0.100 ± 0.078 (0.000 to 0.200)	0	0.150 ± 0.020 (0.05 to 0.185)	0	0.100 ± 0.035 (0.05 to 0.160)	0	0
V_{10}	-0.086 ± 0.013 (-0.100 to -0.012)	0.048 ± 0.029 (0.000 to 0.100)	0.308 ± 0.066 (0.200 to 0.400)	0	-0.151 ± 0.049 (-0.200 to +0.080)	0	0

Table 2h: Electrocardiogram of clinically healthy Horses: Amplitudes of the various wave forms in different leads.

Lead	Mean ± SD (Range) in millivolts N = 22						
	P	Q	R	S	T	QS	R'
I	0.106 ± 0.057 (0.000 to 0.200)	0.032 ± 0.013 (0.000 to 0.050)	0.281 ± 0.166 (0.000 to 0.500)	0.319 ± 0.303 (0.000 to 1.400)	-0.378 ± 0.496 (-1.300 to 0.300)	0.266 ± 0.246 (0.000 to 0.550)	0
II	0.195 ± 0.068 (0.000 to 0.300)	0.087 ± 0.070 (0.000 to 0.200)	0.420 ± 0.248 (0.000 to 0.800)	0	-0.055 ± 0.549 (-0.800 to 0.700)	0.325 ± 0.064 (0.000 to 0.400)	0.130 ± 0.112 (0.000 to 0.250)
III	0.122 ± 0.068 (-0.150 to 0.200)	0.075 ± 0.057 (0.000 to 0.150)	0.592 ± 0.449 (0.000 to 1.500)	0	0.268 ± 0.458 (-0.400 to 1.150)	0.437 ± 0.137 (0.300 to 0.600)	0.150 ± 0.046 (0.000 to 0.200)
aVR	-0.110 ± 0.080 (-0.200 to 0.100)	0	0.129 ± 0.109 (0.020 to 0.400)	0.381 ± 0.331 (0.050 to 1.000)	0.063 ± 0.502 (-1.100 to 0.600)	0	0
aVL	-0.046 ± 0.103 (-0.150 to 0.200)	0.035 ± 0.011 (0.000 to 0.050)	0.184 ± 0.126 (0.000 to 0.400)	0.258 ± 0.201 (0.000 to 0.600)	-0.255 ± 0.265 (-0.800 to 0.200)	0.350 ± 0.050 (0.000 to 0.400)	0
aVF	0.154 ± 0.082 (-0.050 to 0.250)	0.078 ± 0.064 (0.000 to 0.200)	0.341 ± 0.142 (0.000 to 0.500)	0	0.081 ± 0.442 (-0.500 to 0.800)	0.369 ± 0.090 (0.000 to 0.500)	0.100 ± 0.040 (0.000 to 0.150)
V_4	0.150 ± 0.035 (-0.100 to 0.200)	0.133 ± 0.056 (0.000 to 0.200)	0.416 ± 0.160 (0.000 to 0.600)	0	0.100 ± 0.187 (-0.100 to 0.300)	0.574 ± 0.259 (0.000 to 0.900)	0
CV_6LL	0.160 ± 0.040 (0.000 to 0.200)	0.060 ± 0.036 (0.000 to 0.100)	0.500 ± 0.224 (0.000 to 0.800)	0	0.040 ± 0.403 (-0.400 to 0.600)	0.300 ± 0.100 (0.000 to 0.400)	0
rV_4	0.016 ± 0.082 (-0.150 to 0.100)	0.076 ± 0.072 (0.000 to 0.200)	0.438 ± 0.156 (0.030 to 0.700)	0	0.241 ± 0.295 (-0.300 to 0.550)	0	0
CV_5RL	0.100 ± 0.040 (0.000 to 0.150)	0.075 ± 0.025 (0.000 to 0.100)	0.190 ± 0.144 (0.040 to 0.500)	0.150 ± 0.004 (0.000 to 0.170)	0.128 ± 0.229 (-0.350 to 0.500)	0	0

Table 21: Electrocardiogram of clinically healthy Donkeys: Amplitudes of the various forms in different leads.

Lead	Mean ± SD (Range) in millivolts; n = 19						
	P	Q	R	S	T	QS	R'
I	0.044 ± 0.052 (-0.100 to 0.100)	0	0	0	-0.242 ± 0.435 (-0.800 to 1.000)	0.541 ± 0.109 (0.300 to 0.900)	0
II	0.125 ± 0.041 (0.000 to 0.200)	0.125 ± 0.020 (0.000 to 0.150)	0.524 ± 0.147 (0.000 to 0.700)	0	-0.192 ± 0.261 (-0.600 to 0.200)	0.500 ± 0.273 (0.000 to 0.800)	0.133 ± 0.010 (0.000 to 0.150)
III	0.063 ± 0.080 (-0.100 to 0.150)	0.086 ± 0.083 (0.000 to 0.200)	0.590 ± 0.203 (0.100 to 0.800)	0	0.391 ± 0.105 (0.200 to 0.500)	0	0
aVR	(0.079 ± 0.053) (-0.100 to 0.135)	(0.020 ± 0.011 (0.000 to 0.038)	0.286 ± 0.253 (0.020 to 0.700)	0.350 ± 0.350 (0.100 to 0.750)	0.283 ± 0.331 (-0.300 to 0.700)	0	0
aVL	-0.011 ± 0.087 (-0.100 to 0.100)	0	0.140 ± 0.136 (0.000 to 0.400)	0.316 ± 0.125 (0.000 to 0.450)	-0.434 ± 0.432 (-1.000 to 0.200)	0.450 ± 0.030 (0.000 to 0.500)	0
aVF	0.098 ± 0.034 (0.000 to 0.150)	0.090 ± 0.010 (0.000 to 0.100)	0.406 ± 0.349 (0.000 to 0.700)	0	0.083 ± 0.211 (-0.300 to 0.300)	0.366 ± 0.028 (0.000 to 0.400)	0.150 ± 0.050 (0.000 to 0.200)
V_4	0.125 ± 0.035 (0.000 to 0.180)	0	0	0	0.025 ± 0.116 (-0.100 to 0.150)	0.349 ± 0.112 (0.200 to 0.500)	0
CV_6LL	0.100 ± 0.003 (0.000 to 0.170)	0	0	0	-0.451 ± 0.700 (-0.500 to 0.400)	0	0
rV_4	0.004 ± 0.088 (-0.100 to 0.100)	0.037 ± 0.012 (0.020 to 0.070)	0.462 ± 0.118 (0.250 to 0.700)	0	0.250 ± 0.050 (0.200 to 0.400)	0	0
CV_5RL	0.010 ± 0.043 (-0.050 to 0.100)	0.030 ± 0.017 (0.000 to 0.050)	0.200 ± 0.100 (0.100 to 0.300)	0	0.287 ± 0.134 (0.150 to 0.500)	0	0
V_{10}	-0.150 ± 0.050 (-0.200 to 0.100)	0.050 ± 0.029 (0.000 to 0.100)	1.030 ± 0.067 (0.950 to 1.20)	0	0.330 ± 0.130 (0.200 to 0.500)	0	0

Table 2j : Duration of the P wave, PQ interval, QRS Complex and QT interval of apparently healthy Dogs.

Lead	Mean ± SD (Range) in milliseconds; n=71			
	P	PQ	QRS	QT
I	27.50 ± 7.02 (10 to 45)	84.86 ± 14.60 (60 to 120)	35.13 ± 9.61 (20 to 60)	178.38 ± 23.83 (120 to 240)
II	32.30 ± 6.62 (20 to 55)	88.11 ± 14.83 (65 to 120)	32.97 ± 9.24 (20 to 60)	173.51 ± 23.33 (110 to 220)
III	30.35 ± 7.39 (18 to 55)	87.43 ± 14.27 (60 to 120)	33.11 ± 10.02 (20 to 55)	174.05 ± 24.35 (100 to 220)
aVR	32.97 ± 8.45 (15 to 60)	86.49 ± 13.89 (65 to 120)	35.67 ± 11.00 (20 to 70)	172.43 ± 26.00 (110 to 220)
aVL	29.86 ± 8.37 (20 to 60)	88.38 ± 15.90 (65 to 120)	30.67 ± 8.75 (20 to 50)	171.35 ± 23.94 (110 to 210)
aVF	30.67 ± 6.47 (20 to 55)	86.62 ± 12.97 (60 to 120)	34.19 ± 9.39 (20 to 50)	174.59 ± 21.71 (100 to 210)
V_4	30.18 ± 6.86 (20 to 55)	84.63 ± 15.31 (60 to 130)	33.33 ± 10.56 (20 to 50)	182.04 ± 21.13 (140 to 220)
CV_6LL	30.00 ± 4.47 (25 to 35)	88.83 ± 18.35 (70 to 120)	31.67 ± 9.83 (20 to 45)	185.83 ± 20.59 (160 to 210)
V_{10}	44.21 ± 6.92 (35 to 60)	86.31 ± 11.28 (70 to 110)	31.58 ± 7.27 (20 to 40)	192.37 ± 22.38 (160 to 240)

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Table 2k: Durations of the P wave, PQ interval, QRS complex and QT interval of clinically healthy cats.

Lead	Mean ± SD (Range) in milliseconds; n=21			
	P	PQ	QRS	QT
I	28.87 ± 7.04 (20 to 40)	56.25 ± 13.02 (40 to 70)	36.67 ± 9.35 (20 to 55)	155.55 ± 39.09 (100 to 220)
II	28.89 ± 4.17 (20 to 35)	52.22 ± 10.02 (40 to 80)	35.00 ± 5.00 (30 to 40)	164.44 ± 44.47 (100 to 240)
III	29.44 ± 6.35 (20 to 40)	52.78 ± 13.02 (35 to 80)	33.89 ± 4.17 (25 to 40)	161.11 ± 41.36 (100 to 240)
aVR	31.11 ± 5.46 (25 to 40)	50.55 ± 10.74 (30 to 65)	36.11 ± 12.19 (20 to 60)	164.44 ± 41.87 (110 to 240)
aVL	29.44 ± 9.17 (20 to 50)	55.55 ± 14.24 (40 to 80)	31.67 ± 6.12 (20 to 40)	162.22 ± 39.62 (110 to 240)
aVF	29.44 ± 6.35 (20 to 40)	56.67 ± 15.81 (30 to 80)	30.55 ± 6.82 (20 to 40)	154.44 ± 43.91 (100 to 240)
V_4	30.00 ± 5.00 (25 to 40)	60.00 ± 15.27 (40 to 80)	33.12 ± 2.58 (30 to 40)	170.00 ± 46.90 (120 to 240)
CV_6LL	27.50 ± 5.58 (20 to 40)	56.67 ± 16.33 (40 to 80)	33.50 ± 2.74 (30 to 40)	161.67 ± 27.87 (130 to 210)
rV_4	34.0 ± 3.52 (25 to 40)	58.0 ± 11.23 (40 to 90)	35.0 ± 4.07 (25 to 40)	157.0 ± 21.68 (130 to 180)
CV_5RL	32.50 ± 5.00 (25 to 40)	53.00 ± 10.83 (40 to 75)	30.00 ± 5.48 (20 to 40)	153.33 ± 33.27 (110 to 200)
V_{10}	30.83 ± 4.81 (25 to 40)	51.67 ± 10.02 (40 to 80)	32.50 ± 6.12 (25 to 40)	156.67 ± 18.62 (120 to 180)

Table 21 : Duration of the P wave, PQ interval, QRS complex and QT interval of clinically healthy Sheep.

Lead	Mean ± SD (Range) in milliseconds; n=24			
	P	PQ	QRS	QT
I	33.33 ± 4.92 (25 to 40)	90.00 ± 21.32 (60 to 120)	44.17 ± 10.19 (25 to 60)	220.83 ± 32.60 (160 to 280)
II	33.33 ± 4.92 (25 to 40)	89.17 ± 16.76 (70 to 120)	37.50 ± 6.65 (30 to 60)	239.17 ± 30.59 (200 to 290)
III	30.00 ± 2.13 (25 to 35)	87.50 ± 17.34 (60 to 120)	53.75 ± 17.34 (35 to 80)	231.67 ± 23.29 (200 to 280)
aVR	32.27 ± 4.67 (25 to 40)	89.09 ± 16.40 (60 to 110)	35.91 ± 8.00 (20 to 50)	232.73 ± 31.33 (200 to 280)
aVL	33.00 ± 5.37 (25 to 40)	94.50 ± 21.14 (60 to 120)	45.91 ± 14.29 (20 to 70)	231.82 ± 30.60 (200 to 280)
aVF	29.00 ± 2.11 (25 to 40)	87.00 ± 16.36 (60 to 120)	48.75 ± 18.35 (25 to 100)	233.33 ± 22.70 (200 to 280)
V_4	31.36 ± 5.95 (20 to 40)	92.73 ± 16.79 (70 to 120)	40.00 ± 12.25 (25 to 70)	227.5 ± 20.64 (200 to 280)
CV_6LL	31.25 ± 5.29 (25 to 40)	105.00 ± 19.15 (80 to 130)	32.50 ± 1.89 (30 to 40)	235.00 ± 10.00 (220 to 280)
V_{10}	39.58 ± 9.40 (25 to 60)	95.83 ± 22.75 (60 to 130)	63.33 ± 16.14 (30 to 80)	250.83 ± 18.81 (230 to 280)

Table 2m: Durations of the P wave, PQ interval, QRS complex and QT interval of clinically healthy goats.

Lead	Mean ± SD (*Range*) *in milliseconds*; n=40			
	P	PQ	QRS	QT
I	27.63 ± 7.06 (20 to 40)	72.37 ± 18.44 (40 to 120)	48.5 ± 11.01 (20 to 70)	212.5 ± 37.96 (130 to 280)
II	30.00 ± 7.95 (20 to 40)	72.25 ± 19.23 (40 to 110)	39.5 ± 8.87 (30 to 60)	213.00 ± 31.47 (160 to 250)
III	29.50 ± 5.35 (20 to 40)	73.50 ± 15.31 (40 to 100)	49.75 ± 11.29 (30 to 70)	203.00 ± 31.30 (160 to 260)
aVR	31.31 ± 7.23 (20 to 40)	74.74 ± 13.07 (50 to 100)	40.00 ± 12.03 (20 to 65)	209.50 ± 36.34 (160 to 280)
aVL	28.42 ± 6.68 (20 to 40)	70.26 ± 16.37 (50 to 110)	46.75 ± 14.53 (20 to 70)	202.00 ± 31.72 (160 to 260)
aVF	29.75 ± 6.17 (20 to 40)	77.00 ± 20.29 (40 to 110)	41.25 ± 13.07 (20 to 65)	204.00 ± 36.48 (120 to 280)
V_4	28.5 ± 6.51 (20 to 40)	74.75 ± 17.43 (50 to 120)	36.05 ± 9.22 (20 to 55)	192.50 ± 36.83 (120 to 240)
V_{10}	36.00 ± 12.31 (20 to 70)	82.50 ± 17.13 (50 to 130)	49.50 ± 12.66 (20 to 80)	211.50 ± 32.33 (150 to 280)

Table 2n: Durations of the P wave, PQ interval, QRS complex and QT interval of clinically healthy Cattle.

Lead	Mean ± SD (Range) in milliseconds; n=28			
	P	PQ	QRS	QT
I	48.93 ± 15.71 (30 to 80)	134.28 ± 32.04 (80 to 200)	39.28 ± 17.30 (20 to 70)	327.14 ± 85.79 (220 to 480)
II	48.00 ± 15.06 (30 to 80)	135.71 ± 42.55 (80 to 240)	35.36 ± 11.17 (20 to 60)	334.28 ± 69.91 (200 to 440)
III	47.86 ± 12.05 (30 to 80)	140.71 ± 37.10 (80 to 240)	37.86 ± 13.26 (20 to 60)	300.00 ± 76.56 (180 to 440)
aVR	50.77 ± 15.52 (30 to 80)	133.08 ± 28.10 (80 to 180)	36.43 ± 10.08 (20 to 50)	324.64 ± 80.73 (200 to 440)
aVL	48.50 ± 10.92 (35 to 80)	140.00 ± 44.97 (80 to 240)	40.36 ± 15.50 (20 to 70)	313.33 ± 68.40 (200 to 420)
aVF	48.57 ± 17.48 (30 to 80)	132.86 ± 25.85 (80 to 160)	37.14 ± 13.26 (20 to 60)	305.00 ± 67.34 (200 to 410)
V_4	53.33 ± 10.65 (40 to 80)	132.00 ± 26.83 (100 to 160)	43.33 ± 2.16 (40 to 50)	330.00 ± 87.41 (200 to 440)
CV_6LL	40.00 ± 5.00 (35 to 50)	110.00 ± 4.14 (100 to 130)	40.00 ± 3.24 (35 to 45)	260.00 ± 54.85 (200 to 380)
rV_4	53.33 ± 12.09 (40 to 80)	133.33 ± 13.09 (120 to 160)	51.25 ± 16.52 (30 to 75)	347.5 ± 37.37 (280 to 400)
CV_5RL	60.00 ± 18.28 (40 to 100)	140.00 ± 18.28 (120 to 160)	50.00 ± 8.14 (40 to 60)	340.00 ± 18.28 (320 to 360)
V_{10}	52.86 ± 10.96 (40 to 80)	145.71 ± 34.09 (100 to 200)	47.14 ± 14.96 (20 to 70)	371.43 ± 66.17 (280 to 450)

Table 20: Durations of the P wave, PQ interval, QRS complex and QT interval of clinically healthy Horses.

Lead	Mean ± SD (Range) in milliseconds; n=22			
	P	PQ	QRS	QT
I	73.18 ± 23.69 (35 to 100)	232.73 ± 31.33 (200 to 280)	72.73 ± 20.04 (40 to 100)	421.82 ± 41.43 (360 to 520)
II	78.18 ± 24.83 (40 to 120)	236.36 ± 23.35 (200 to 280)	53.64 ± 12.01 (40 to 80)	436.36 ± 31.71 (400 to 480)
III	73.64 ± 12.06 (50 to 90)	238.18 ± 18.88 (200 to 280)	60.91 ± 16.41 (40 to 90)	430.91 ± 50.09 (360 to 520)
aVR	81.82 ± 20.40 (50 to 120)	241.82 ± 27.86 (200 to 280)	50.45 ± 14.57 (25 to 80)	419.09 ± 48.67 (320 to 500)
aVL	78.18 ± 17.21 (40 to 100)	230.91 ± 22.56 (200 to 280)	50.00 ± 17.32 (30 to 80)	436.36 ± 44.56 (360 to 520)
aVF	79.09 ± 12.21 (60 to 100)	236.36 ± 32.02 (200 to 280)	58.64 ± 18.72 (30 to 90)	434.54 ± 55.20 (360 to 560)
V_4	70.00 ± 20.00 (40 to 90)	240.00 ± 14.14 (220 to 260)	65.00 ± 13.23 (50 to 85)	416.00 ± 60.66 (340 to 520)
CV_6LL	68.00 ± 26.88 (40 to 100)	232.00 ± 30.47 (200 to 280)	54.00 ± 10.42 (40 to 80)	436.00 ± 30.90 (400 to 500)
rV_4	85.00 ± 3.37 (80 to 100)	253.33 ± 10.05 (240 to 270)	56.67 ± 13.66 (40 to 80)	443.33 ± 28.75 (400 to 490)
CV_5RL	67.14 ± 20.59 (40 to 90)	254.28 ± 11.02 (240 to 280)	61.43 ± 13.40 (40 to 100)	412.86 ± 19.04 (360 to 440)

Table 2P: Duration of the P wave, PQ interval, QRS complex and QT interval of clinically healthy Donkeys.

Lead	Mean ± SD (Range) in milliseconds; n=19			
	P	PQ	QRS	QT
I	51.67 ± 10.41 (40 to 90)	160.00 ± 35.82 (120 to 240)	72.50 ± 10.75 (60 to 100)	473.33 ± 64.08 (400 to 560)
II	66.67 ± 19.66 (40 to 80)	158.33 ± 34.88 (120 to 220)	58.33 ± 5.83 (40 to 70)	446.67 ± 58.88 (360 to 520)
III	46.67 ± 16.33 (40 to 80)	160.00 ± 39.50 (120 to 220)	60.00 ± 12.65 (40 to 80)	450.00 ± 53.29 (380 to 520)
aVR	65.00 ± 20.11 (40 to 100)	178.33 ± 38.17 (120 to 220)	48.33 ± 16.02 (30 to 80)	470.00 ± 56.21 (400 to 560)
aVL	55.00 ± 13.45 (40 to 90)	165.00 ± 34.50 (120 to 210)	68.33 ± 18.35 (40 to 90)	470.33 ± 53.17 (400 to 560)
aVF	59.67 ± 18.35 40 to 80	155.00 ± 30.74 120 to 200	55.00 ± 12.25 40 to 70	460.00 ± 55.73 400 to 560
V_4	50.00 ± 12.02 (30 to 65)	180.00 ± 35.00 (140 to 240)	60.00 ± 11.40 (40 to 80)	380.00 ± 46.00 (320 to 480)
CV_6LL	55.00 ± 16.25 (35 to 80)	160.00 ± 27.50 (130 to 240)	60.00 ± 12.54 (40 to 90)	450.00 ± 36.20 (400 to 520)
rV_4	52.00 ± 10.89 (40 to 80)	170.00 ± 44.72 (120 to 230)	61.00 ± 14.32 (40 to 80)	464.00 ± 60.66 (400 to 530)
CV_5RL	57.50 ± 10.61 (40 to 80)	160.00 ± 31.64 (120 to 240)	48.76 ± 10.31 (35 to 60)	470.00 ± 68.34 (400 to 560)
V_{10}	80.00 ± 14.14 (60 to 100)	186.00 ± 21.91 (150 to 250)	68.00 ± 7.37 (60 to 80)	464.00 ± 51.77 (380 to 530)

CHAPTER THREE:

ELECTROENCEPHALOGRAPHY

Literature review

Historical development

The earliest clinical EEG in animals was reported in dogs about the mid-sixties (Croft, 1963). However, as far back as 1890 animals were (and still are) used as experimental models for EEG and other brain related researches thereby making immense contribution to the development of both human and animal EEGy. Veterinary EEGy grew due to commendable contributions of several researchers that include Redding, et al., (1964); Croft (1963); Prynn and Redding (1968); and Klemm (1976). Table 3a summarizes data of scalp EEG of some domestic animals.

Physiologic basis of the EEG

The electrical activity of the brain is dissimilar to that of the heart; while the functional syncytium of the heart results in a highly organised electrophysiologic activity manifested by regular wave forms and periodicity, the same cannot be said of the EEG. The EEG is a random membrane fluctuations of all excitatory and inhibitory postsynaptic potentials within the pick-up range of the electrodes used in the recording. Though far from complete, evidence of

the genesis is sufficient to conclude that the EEG is a very sensitive indicator of the most fundamental functions of the brain tissue (Klemm, 1969).

It is now accepted that the cortex is the major source of EEG wave forms especially in scalp recordings. Studies by Li and Jasper (1953) involving microelectrode recordings of brain neurons revealed that single neurons in the cortex displayed similar EEG activity to that recorded from the scalp using gross electrodes, and also membrane potentials contribute greatly to the grossly recorded EEG. In 1964 Fujita and Sato correlated intracellular hippocampal activity with simultaneously grossly recorded hippocampal EEG. They also found that membrane potential fluctuated in synchrony with the EEG. In an earlier report it was shown that neurons do produce extracellular currents that are sufficient to permit the registration of EEG waves (Elul, 1962).

Practically, the EEG is a graphic plot of voltages as a function of time representing a sum of electrical activities of the brain regions in which the electrodes are located. These electrical activities are present irrespective of, but may change due to, ex[illegible] influences and could arise

from change in membrane impedance; intra- and extracellular chemical change; random and rhythmic synaptic transmission from cortical and extracortical neurons; and electrotonic spread from immediately adjacent active neurons.



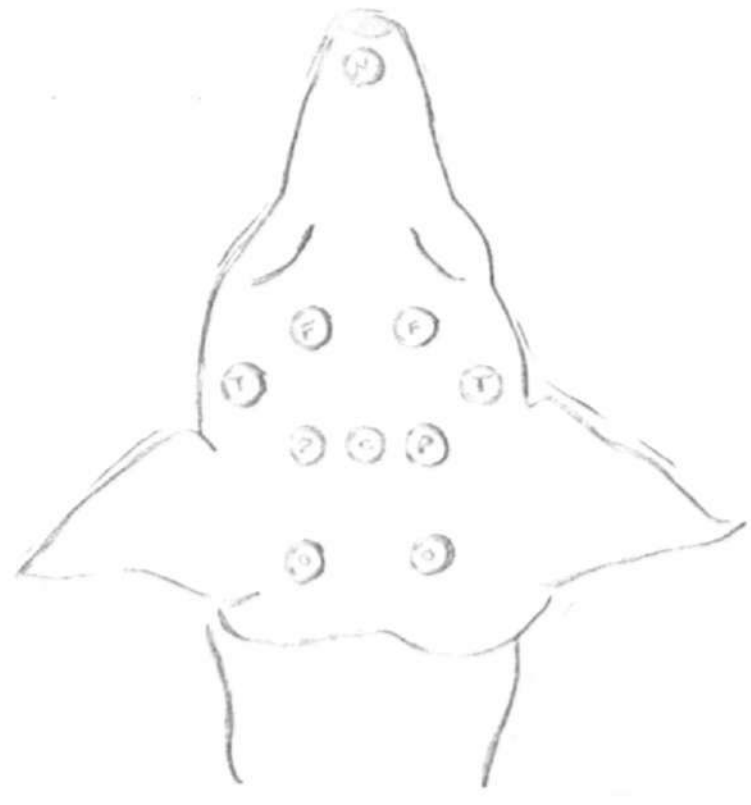


Fig. 3a: Proposed electrode position sites for scalp recording of electroencephalograms in the dog. Electrode positions: central (C), nasal (N), frontal (F), occipital (O), parietal (P) and temporal (T). (From Klemm, W. R.: In: Applied Electronics for Veterinary Medicine and Animal Physiology; Charles C Thomas, Illinois, 1976).

Electroencephalographic voltages

The wave forms in the EEG are biphasic and could therefore be negative or positive but, unlike those of the ECG, lack conspicuous periodicity. Cooper et al. (1965) showed records where electrically evoked cortical potentials of 1-2 mV were not registered by intracortical electrodes that were only 8 mm away. Due to several influences the type of potential seen initially by a surface electrode may subsequently change as a result of current flow. This could account for the many reports of recorded membrane potentials falling out of phase with simultaneously recorded surface waves. Arising from many cortical generators EEG waves are usually complex and irregular. While it is established that scalp-derived EEG originates mainly from the cortex, the activity of the subcortical areas, though considered to be attenuated has profound influence on cortical function because of the extensive anatomical and physiologic interconnections.

Electroencephalographic montages

Standardised montages are not available for the various species in veterinary EEGy. A commonly used scheme (Fig. 3a) of electrode location and identification proposed by Klemm (1968) involves the placement of electrodes in ten skull positions. Eight of these are placed, in four pairs, in the left and right occipital, parietal, temporal and frontal cortical regions. Usually a 9th electrode is placed in the centre between the left and right parietal

electrodes. The 10th is placed in the nasal region and is used as a reference (relatively inactive) electrode. All EEG recordings are bipolar and recorded using two or more electrodes. Reference bipolar recordings involve placement of two electrodes, one at an active brain region and the other at a relatively inactive, usually, nasal site. In non-reference bipolar recordings, both electrodes are placed in different but active brain regions.

Electroencephalographic electrodes can be placed on the scalp, or in the subcutis of conscious or sedated animals. However, electrode placement in the dura, on the cortical surface, and in the subcortex necessitates the use of sedation, local and/or general anaesthesia. For safety and efficiency cortical and subcortical placement of electrodes are best achieved with the animal under general anaesthesia. Details of surgical implantation of electrodes in different mammalian and avian species with respect to the surgical techniques, stereotaxic apparatuses for proper head orientation as well as the essentials of electrode making and use are documented (Delgado, 1961; Spooner, 1965; Bures et al., 1967; Klemm, 1969; Osuide and Sokomba, 1978). Stereotaxic atlases for many species have been published. These include atlases for the dog (Adrianov and Merink, 1959), the cat (Bleir, 1961), the sheep and goat (Andersson, 1951) and several laboratory animals (Bures et al., 1967).

Electroencephalographic equipment

Modern electroencephalographs are highly sophisticated and designed to receive input for processing through a combination of filtration, attenuation and amplification. This is followed by the display of various wave forms measured in microvolts(mcV) as a function of time. To meet these objectives the machines are therefore designed to maximally remove "noise" thereby allowing only physiological signals to pass through. A delicate balance subsequently exists between the animal, machine, electrodes and the sorrounding environment. The electrodes used in veterinary EEG recording are of various types and designed to suit their different applications. They include the silver-silver chloride type affixed to the scalp and the platinum (or other metals) needle electrodes that are inserted subcutaneously. Surgically implanted electrodes in specific brain regions are of different metal types and characteristics.

Animal preparation for EEG recording

Effective grounding of the animal involves its placement in a shielded environment, usually a cage with poorly conducting materials . In the shielded environment the animal should be free of any interference. Similarly, it is essential to properly ground the electroencephalograph, and through appropriate use of its various adjustment controls, an artefact-free EEG can be obtained, all other conditions being satisfactory. Electrode contact with the

subject at one end and with the machine at the other must be secure and adequate.

Established EEG wave patterns

In scalp recordings of animals, the amplitudes and frequency duration of the EEG wave forms, though highly variable are, respectively, in the range of 5-75 mcV and from less than 1/sec to more than 100/sec depending on the recording equipment, species, age, and other factors. Though usually compounded, EEG waves may, under certain conditions, possess definite characteristics.

Alpha rhythms have frequencies of 8-13 cycles per second (cps) in adults and are most prominent in the posterior areas. They are present most markedly when the eyes are closed in the awake-resting state, but are attenuated during attention, especially visual. Their voltages are about 50 mcV (Kool et al., 1978).

Beta rhythms have frequencies higher than 13 and rarely up to, but not exceeding, 50 cps (Guyton, 1981). They are most frequently recorded from the parietal and frontal regions of the scalp and can be divided into two major types, namely the Beta I and Beta II. Beta I waves disappear during intense electrical activity. On the contrary, Beta II waves appear during intense activation of the CNS.

During the state of disappointment, frustration and some brain disorders EEG waves of

frequencies between 4 and 7cps appear, they are the theta waves (Guyton, 1981).

Delta waves have frequencies less than 4 cps and chiefly occur in deep sleep and in very serious organic brain disease.

Pathological processes inevitably change the electrical activity of diseased neurones and of the healthy neurones with which they are intimately connected. These changes may manifest in the EEG as:

(1) High voltage slow activity (HVSA)
(2) Low voltage fast activity (LVFA)
(3) High voltage fast activity (HVFA)
(4) Spikes and Spindles
(5) Beta and theta rhythm outbursts
(6) Electrical silence
(7) Seizure activity

Aids for recording the EEG

Electroencephalographs have several facilities for fine adjustment and control of wave form amplitude, configuration and duration. Such facilities include:

(1) Calibration selector switch-the commonest calibration voltage used is 50 mcV to 10 mm. The selector switch however, could have a wide range of calibration voltages quite below and far above the, common, 50 mcV.
(2) Half, low and high amplitude frequency switches permit proper

selection of frequency range.

(3) Sixty hertz filters for "noise" filtration

(4) Sensitivity and equaliser adjustment controls.

(5) Chart drive paper speed control.

Some machines also have accessories that permit simultaneous recording, playback, oscilloscope coupling and computerised analog to digital conversion.

Restraint during EEG recording

Restraint is also essential in recording aretefact-free EEG from animals. It is however often not always easily achieved because even under general anaesthesia the EEG may still contain temporal muscle potential artefacts (Klemm, 1976). Table 3b summarises the advantages and disadvantages of the use of physical restraint, muscle relaxants and general anaesthetics in recording and interpretation of the EEG (klemm, 1976). Despite its hazards, general anaesthesia may be considered the most reliable method of restraint for obtaining the EEG from animals. It enables artefact-free recording since it abolishes motion and muscle artefacts and the claims that it may mask normal or abnormal activities have not

been proved (Klemm, 1976).

This aspect of the research was conducted to establish baseline scalp EEG data for Nigerian indigenous cats, dogs, goats and sheep, and to correlate depth EEG patterns of the normal chicken prior to and following administration of some neuroactive drugs.

Materials and methods

Cats, dogs, goats and sheep were used for scalp EEG recordings. All animals used were clinically normal, and for each species both sexes were used varying in age from one to two years. For each species, seven animals whose EEG was sufficiently stable were selected for voltage and frequency range computation.

Studies involving recordings from surgically implanted cortical and subcortical electrodes were conducted with clinically normal chickens in their first week of life (Fig. 3b).

Restraint for EEG recording

For the scalp recording all animals used were first premedicated with appropriate doses (0.045 mg/kg) of atropine (Scanpharm, Copenhagen, Denmark) administered subcutaneously. Surgical anaesthesia was then induced in dogs with sodium pentobarbitone

(30 mg/kg, IV), and in cats with an equal volume-mixture of two anaesthetics (Beaver and Klemm, 1973). The mixture was made of 6% pentobarbitone sodium (May & Baker Ltd, Degenham, England) and 2.5% thiopentone sodium (May & Baker, Ltd, Degenham, England) solutions and then diluted by a half with sterile water. Dogs and cats in surgical anaesthesia were then appropriately intubated with endotracheal cannulae. In premedicated sheep and goats however, xylazine (Bayer, Germany) at a dose of 0.25 mg/kg, IM, was then administered.

Implantation of sterile electrodes in the chick brain involved general anaesthesia and surgical procedures. Twenty- five clinically normal White Ranger cockerels were initially weighed and then safely brought into surgical anaesthesia with halothane. The head was fixed and then shaved in a stereotaxic apparatus. Under aseptic conditions, a midline incision was made in the scalp from behind the comb to the neck region. The exposed skull and neck muscles were subsequently kept moistened with normal saline throughout the implantation procedure. After levelling the head, the co-ordinates for the accessory hyperstriatum (AH), optic tectum (OT) and reticular formation (RF) were determined using the bregma as a reference position. The actual co-ordinates used for the

implantation were then obtained using the correction values shown in Table 3c.

The corrected co-ordinates obtained were then used to implant the electrodes in the respective regions of the chicken brain in accordance with the methods of Spooner(1965) and Osuide and Sokomba (1978). A pair of electrodes for recording the electromyogram (EMG) was then placed in the proximity of the head and insertion of the semispinalis capitis muscle of one side of the dorsolateral aspect of the neck. A reference comb electrode was then positioned. Dental acrylic powder and solution were used for adequate fixing of the electrodes in their implanted areas. Following completion of the surgical implantation, chickens were kept warm under electric bulb heat until recovery. At the end of each experiment, electrode positions in the brain tissues were verified histologically as described by Osuide and Sokomba (1978).

Electroencephalographic electrodes

Needle electrodes aseptically inserted in the scalp were used for recording the EEG in dogs, cats, sheep and goats. Monopolar and bipolar electrodes were made from the pointed ends of stainless steel insect pins No. 00. One variety of monopolar electrodes was used for cortical implantation and another for the neck muscles and comb. The bipolar electrodes were used for RF implantation.

Electroencephalographic recorder

The electroencephalograph (Grass Inst., Model 79D) used had four principal units:

(1) The main frame housing, among other things, the EEG amplifiers (Model 7P511) with their various sensitivity and filter controls.

(2) The input cable-receptacle system for electrode communication from the animal to the appropriate EEG amplifiers.

(3) The electrode selector panel for efficient simultaneous electrode selection and calibration of all amplifiers in use.

(4) The chart paper-drive with a wide range of speed, 2.5 to 100 mm/sec.

Calibration signals were amplified with a gain of 50 mcV/cm of pen deflection, half-amplitude low frequency setting was 0.3 to 1 Hertz(Hz) and the half-amplitude high frequency setting of 100 to 1000 Hz for the output jack and 60 to 90 Hz for the recording pens.

Tracings were made on standard EEG paper (Grass Inst) at a speed of 2.5 to 25 mm/sec.

Recording procedure for the EEG

Scalp EEG

In sheep and goats the scalp EEG was obtained from sedated but conscious animals; general anaesthesia was necessary in recording the EEG in dogs and cats. Occasinally, it was necessary in a few animals to shave the specific areas of the head to achieve adequate electrode contact thus avoiding hair-generated artefacts.

Following preparation the animal was then transferred into a special wooden cage with formica floors, insulated sides and roof. The spaces in between the woods on the sides were covered with special nettings. The shielded environment was provided in order to ensure effective shielding of the animals during recording. The electrodes and their specific areas of attachment were scrubbed with 70% alcohol solution. After the sequence of restraint, placement and skin preparation, electrodes were inserted in accordance with the montage proposed by Klemm (1968). A grounding electrode was on each recording occasion clipped to the ear or nose of the animal.

Intracortical and subcortical EEG

Only the chick [illegible]ed for depth EEG. After allowing a minimum of twenty-four hours

post-implantation, the chicken was then placed in the shielded cage. Appropriate male-female "connector" mating was then made to ensure adequate electrode communication between the animal and the electroencephalograph. Because the recording procedure always began with the chicken in its normal conscious state it was routinely necessary to allow the caged chicken to calm down.

The normal EEG of the chicken and alterations following administration of pentobarbitone (May & Baker Ltd., England), xylazine (Bayer, Germany), halothane (ICI, London, UK), apomorphine (Macfarland, Edinburgh, UK) and leptazol (Sigma chemicals, USA) were recorded.

The EEG derived from all animals was obtained under conditions of satisfactory quiescence free of distractions from within and outside the recording room. The calibration voltage, determined at the beginning of each recording, varied with the recording pr[illegible]. However, it was most frequently 50 mcV to 1cm deflection of the pens. Both reference and non-reference tracings from the brain r[illegible] were recorded. In addition, simultaneously recorded EMG from the chicken served as a good indicator of the concurrent muscle tone.

Results

A. Behaviour and EEG of Normal chickens

Adaptation of recommended implantation methods enabled successful placement of electrodes in cortical and subcortical areas of White Ranger cockerels used in this study. Most chickens so implanted were fully recovered and essentially normal in behaviour within 3 to 24 hours post-implantation.

The wake, alert EEG (Figs 3c & 3d) in the normal chicken during the first week of life was characterised by background activity (20-75 mcV, 1-3 wps) and dominant wave forms varying in voltages and frequencies as 5-65 mcV and 5-10 wps in the AH, 5-140 mcV and 6-10 wps in the OT, and 3-100 mcV and 8-14 wps in the RF. Sharp-wave transients usually in association with sudden motion were occasionally observed.

During normal sleep state relative synchrony occurred in the EEG pattern characterised by a shift to slower wave forms of higher voltages. The voltage and frequency ranges of the dominant wave forms were 5-80 mcV and 4-8 wps in the AH, 5-170 mcV and 4-8 wps in the OT, and 5-175 mcV and 3-8 wps in the RF. Sleep spindles were infrequently observed.

Behaviour and EEG of chickens under effects of some drugs

(I) Halothane

Prior to halothane inhalation the chickens were alert and calm, and usually squatted on the sternum. Their EEG was characterised by dominant wave forms of respective voltages and frequencies of 5-65 mcV and 5-10 wps in the AH, 25-140 mcV and 6-9 wps in the OT, and 3-30 mcV and 8-11 Hz in the RF (Fig. 3c) as well as infrequent sharp-transients.

Following halothane inhalation the chickens assumed incomplete lateral recumbency with periodic outstretching of the limbs and wings, their eyes remained semi-closed. Corresponding to the sedated behaviour the EEG was relatively synchronised with significant increase in the voltage and decrease in the frequency of the wave forms. During the drowsy state, the wave forms were generally of the range 25-80 mcV and 5-8 wps in the AH, 25-170 mcV and 4-8 wps in OT, and 5-35 mcV and 4-8 wps in the RF (Fig. 3c). Sharp waves, though still present were quite infrequent.

Under halothane general anaesthesia the chickens were laterally recumbent, completely non-responsive to auditory, photic and tactile stimuli, and had

both eyes completely shut. However, low-grade chirps to complete arousal could be induced in response to noxious stimuli such as pin-prick. The brain activity during anaesthesia was characterised by greater synchrony of dominant wave form ranges of 25-150 mcV and 4-6 wps in the AH, 25-190 mcV and 4-6 Hz in the OT, and 5-15 mcV and 4-8 wps in the RF. Sharp waves and spindles were observed in the cortical, but not subcortical tracings (Fig. 3c).

Progressive decrease in muscle tone from wakefulness to unconsciousness was indicated in the EMG (fig. 3c). During recovery muscle tremors due to light shivers were observed but disappeared with complete wakefulness.

(II) Pentobarbitone sodium

Following intraperitoneal administration of pentobarbitone sodium (20 mg/kg), anaesthesia was easily induced in the chickens within 4-8 minutes. The duration of general anaesthesia was 3.5-4.5 hours characterised by uncounsciousness, regular respiratory rhythm, closed eyes, complete lack of response to arousal stimuli in the first one-third to one-half of the period of anaesthesia. Later varying degrees of [illegible] stimuli to the [illegible]ens elicited corresponding responses from low-[illegible] chirps to catalepsy followed by [illegible] reversion to

deep sleep. Arousal to complete wakefulness as was possible under halothane could not be induced under pentobarbitone anaesthesia.

In the anaesthetized state majority of the chickens adopted lateral recumbency with the head in contact with the cage-floor. The posture was maintained until emergence from anaesthesia which was most usually signalled by tremors, shrieks, catalepsy, bizarre sternal posture, and finally alert wakefulness. In correlation with the behaviour the EEG became synchronised during general anaesthesia.

The onset of a stable EEG pattern occurred 10-18 minutes following administration of pentobarbitone sodium. HVSA in the AH (100-175 mcV, 6-8 wps), OT (20-50 mcV, 4-6 wps) and RF (30-100 mcV, 6-8 wps), replaced the relatively LVFA of wakefulness (Fig. 3d). Spindles, sharp waves and spikes mainly cortical as well as a pseudo-stereotypy pattern in the AH were recorded.

The EMG (Fig. 3d) showed appreciable decrease in muscle activity during anaesthesia. During recovery tremors and catalepsy were accompanied by exaggerated spikes and sharp waves on a desynchronised background EEG (Fig. 3d).

Anaesthetic overdose characterised by severe depression of the CNS and manifested by relative

electric silence on the EEG (Fig. 3e) was usually followed by death.

(III) Xylazine

The EEG during wakefulness (Fig. 3f(A)) prior to and following (Fig. 3f(B)) intramuscular administration of xylazine (80 mg/kg) adequately summarise the CNS depressant and muscle relaxant effects due to the drug in chickens. Most chicks dramatically went limp within a minute of administering the drug. The EEG showed marked synchrony characterised by dominant HVSA in the AH (20-75 mcV, 2-4 wps), OT (10-100 mcV, 3-4 wps) and the RF (35-180 mcV, 1-3 wps) overlying a background low voltage moderately slow activity. Frequently, spikes and sharp waves were observed.

Compared to the EEG pattern under xylazine sedation the overall dominant activity in behavioural wake or sleep state was characterised by less wave synchrony in the AH (5-65 mcV, 4-7 wps), OT (5-75 mcV, 5-10 wps)and the RF (5-175 mcV, 3-14 wps) with occasional spindles .

Marked reduction in muscle tone was shown in the EMG (Fig. 3f).

Following xylazine administration the characteristic changes in behaviour included marked muscle relaxation, sedation, practical immobility

(except regular respiratory rhythms), sternal recumbency and, in some, abnormal postures. The duration of effect lasted 2-5 hours.

(IV) Apomorphine

A sequence of changes in behaviour and EEG patterns was obtained following administration of apomorphine (10 mg/kg, IP) in chickens.

Within the first 5 minutes of drug administration original posture (sternal or standing) was maintained with head-drooping and half-closed eyes. There were occasional head-shakes that gradually became more frequent and severe in the succeeding 5 minutes. Progressive restlessness became more evident by 20 minutes and was characterised by marked alertness, vigorous head-shakes, tremors and aimless pecking of the cage-wall, floor and sternal region. By 40-50 minutes of drug administration restlesness was at its peak with sudden and frequent attempts to fly. Subsequently, within the ensuing 1.5 to 2 hours, there was gradual increase in calmness with changes in posture, from standing to sternal recumbency. Chickens generally crouched at the cage-corner with closed eyes, drooped head and low-grade chirps. The head-shakes, though reduced in frequency and severity, were usually continued even after 3 hours of onset.

Desynchrony and irregularly frequent spikes and sharp waves as well as exaggerated episodes of paroxysms (Fig. 3g) characterised the dominant EEG pattern following apomorphine administration in chickens. The characteristics varied in correlation with the three behavioural states (initial mild excitement , peak restlessness and relative sedation) described above.

The EMG showed corresponding changes (Fig. 3g) in relation to variation in the behavioural and EEG patterns. In particular, irregular fine tremors hardly noticeable in the feather-covered chickens were evident and lasted several hours.

(V) Leptazol

Following subcutaneous administration of leptazol (80 mg/kg) behavioural drowsiness was suddenly replaced within 2 minutes by alertness. The EEG pattern became desynchronised with high voltage spikes and paroxysms in both cortical and subcortical tracings (Fig. 3h). These were accompanied by corresponding high voltage sharp waves in the EMG. There was also moderate restlessness, some convulsive seizure activity alternating with non-seizure periods characterised by alert, jumpy state while standing.

Generally, in the succeeding 10-15 minutes convulsive seizures were evident, that became

frequent and mixed with high voltage fast activity (HVFA) and desynchrony. By 35 minutes behavioural changes were striking and included continuous seizures (Fig 3h) with occasional HVFA of short duration succeeded by convulsive episodes. These episodes were characterised by chirping, abnormal leg stretches, exhibition of all kinds of abnormal postures in sternal and lateral recumbency and in standing positions. At 40-50 minutes the convulsive seizures became more severe (Fig. 3h), cessation of breathing followed, and death ensued abruptly; the eyes and mouth remained open.

Scalp EEG in the dog, cat, sheep and goat

Behaviour and EEG of the dog in anaesthesia

Dogs were brought into surgical anaesthesia following sequential administration of atropine (0.045 mg/kg, SC) and sodium pentobarbitone (30 mg/kg, IV). Within 10-15 minutes regular cardiac and respiratory rhythms have become established. Subsequently, a variable period of 5-35 minutes followed during which the EEG was most stable. The characteristics of the stable EEG, summarised in Table 3d consisted of dominant waves of theta to alpha rhythms with spindles (4-7 bursts/minute) and occasional sharp waves.

Progressive amplitude decrease in an anterior-posterior transhemispheric bipolar recording arrangements was frequently observed (Fig. 3i). However, common nasal reference intrahemispheric recording arrangements showed progressive amplitude increase in an anterior-posterior direction (Fig. 3i). Responses to arousal stimuli absent during the initial stages returned only in the last one-third or one-quarter of the anaesthesia period. Recovery was marked by relative desynchrony with occasional spikes and sharp transients some of which were artefactual (Fig. 3i). During the period of anaesthesia, 2.5-4 hours, the dogs generally remained calm. Responses to arousal stimuli gradually returned in the last

one-quarter of the anaesthesia period.

Behaviour and EEG of the anaesthetised cat

Following premedication with atropine (0.04 mg/kg, SC) smooth surgical anaesthesia was induced using IV sodium pentobarbital and thiopental sodium in equal-volume mixtures. During the period of anaesthesia which lasted 3-5 hours, most cats remained calm for 2-3 hours after which infrequent body jerks of 1-3 seconds were observed in the absence of any arousal stimulus. These movements involved the thoraco-cervical region and usually succeeded expiratory efforts. Respiration was quite regular while the eyes remained moderately dilated initially with prolapsed third eyelid but became semi-closed [illegible]s recovery.

Responses to auditory, photic and tactile stimuli were completely absent during the first one-third to one-half of the period of anaesthesia. Subsequently, mild to marked responses to arousal stimuli were manifested in form of movements of the eyelid, ear, limbs and other parts of the body. Towards recovery, respiration became faster, and there were frequent tremors beginning with the forelimbs and eyeball movements.

Following smooth induction of anaesthesia stability of the EEG was usually obtained within 20-35 minutes, and remained so for the next 1-3

hours in the absence of any arousal stimulus. A summary of the dominant EEG activity during the stable state is presented in Table 3e. However, there were associated spindles (4-12 bursts/min) and infrequent sharp-wave transients. Great similarity and a lot of in-phase activity over the cortex were shown in bipolar transhemispheric anterior-posterior recording arrangement (Fig. 3j). Common reference tracings revealed great similarity as well as the not infrequent dissimilarity between homologous regions of the cortical hemispheres (Fig. 3j).

Behaviour and EEG pattern in sedated sheep

Under xylazine sedation, sheep remained calm and in right lateral recumbency for 40 minutes to two and a half hours. Respiration was relatively regular and there was occasional tailwag or short-duration bleating. The dominant EEG activity consisted of stereotyped waves of theta to alpha rhythms of voltage ranging from 5-60 mcV. Occa[illegible] background activity (of relatively higher frequencies and lower voltages) with sharp waves were evident. Table 3f is a summary of the dominant activity of common reference and bipolar anterior-posterior intrahemispheric and transhemispheric recordings. There was apparent increase in amplitude in a frontal, temporal to occipital axis of reference recordings (Fig. 3k).

The sedated state was also characterised by great in-phase activity over the whole cortex as shown in both reference and bipolar transhemispheric tracings (Fig. 3k). Spindle bursts usually of low voltage and spread were frequently recorded appearing as a superimposition on the background activity (Fig. 3k (i)).

Xylazine-sedated sheep remained calm in right lateral recumbency for a variable period, 40 minutes to 2.5 hours. Usually, the most reliable patterns were best obtained in the first 20-50 minutes after entry into the shielded cage and electrodes affixation. Due to motion artefacts in the later stages of the sedation the EEG became irregular and characterised by desynchrony, spikes and abnormal baseline deflections.

Behaviour and EEG of the sedated goat

Behaviour of goats, duration of sedation and recovery EEG under xylazine were quite similar to those of xylazine-sedated sheep. Goats were however relatively calmer except for occasional "grunts". Stable EEG pattern was available only after 25 minutes of placement in the cage.

The principal features of the EEG of xylazine-sedated goat as summarised in Table 3g consisted of waves moderate in voltage and of slow activity in theta to alpha regions; HVSA (Fig. 3l)

tended to predominate with occasional spindles (7-15 bursts/min). Background activity consisted of waves of low voltage (3 - 5 mcV) and moderately fast activity (6-11 wps).

Discussion

Depth EEG in the chicken

Halothane is the inhalant anaesthetic of choice in birds (Mapletoft and Futter, 1969) and has been used successfully in several avian species. The drug is known to depress all functions of the CNS with marked effect on the cardiopulmonary system. The literature is however silent on the EEG pattern of the chicken under halothane anaesthesia. The EEG patterns in humans under halothane anaesthesia as was similarly observed in chickens used in this study, show progressive replacement of LVFA by HVSA with increasing depth of halothane anaesthesia (Gilman and Goodman, 1985). Halothane caused marked skeletal relaxation (Fig. 3c). This property has been used in conjunction with its marked anaesthetic potency to produce adequate surgical anaesthesia for orthopaedic surgical procedures in the eagle (Wingfield and De Young, 1972), hawk, heron, and crane (Bush, 1977).

This study also confirms the absence, in the chicken EEG, of the characteristic "barbiturate burst" activity that features in the human EEG under pentobarbitone anaesthesia (Oakawa and Gotoh, 1965). However, occasional spindles, sharp waves and spikes were observed quite frequently (Fig.

3d). Electric silence characteristic of the EEG of deep anaesthesia reported by Drohocki and Drohocka (1939) in several animal species probably resembles the "isoelectric" EEG obtained in anaesthetic overdose in some chickens in this study (Fig. 3e).

Although halothane and pentobarbitone produced stable EEG within 10 minutes of administration, mild to moderate tremors were observed in chickens recovering from both drugs. The tremors were probably due to hypothermia resulting from depression of the hypothalamic thermoregulatory center as with halothane, or decreased basal metabolism and peripheral vasodilation as occurs with pentobarbital (Booth and McDonald, 1982).

Xylazine dramatically produced rapid and adequate sedation and muscle relaxation in chickens (Fig. 3f). Response to arousal stimuli persisted throughout the sedation period,3 - 5 hours. But xylazine produces potent analgesic effects (Schmitt et al., 1974) probably due to activation of central alpha- adrenoceptors (Loc. et al., 1974) especially alpha-2 adrenoceptors that control release/uptake of central neuronal dopamine and norepinephrine (Hedler et al., 1981). The stereotyped HVSA and spikes produced by xylazine in this study may be considered characteristic of this drug (Fig 3k).

Relative to morphine, apomorphine has diminished analgesic properties, but retains potent stimulatory effects on the dopaminergic receptors of the chemosensitive trigger zone (CTZ) inducing emesis in several species other than the swine and chickens (Booth and McDonald, 1982). It also produces a combination of excitation and sedation in mammals (Jaffe and Martin, 1985). Chickens showed progressive increase in excitatory behaviour and desynchrony in the EEG with spikes and exaggerated paroxysms (Fig. 3g). Peak restlessness occurred 40 to 50 minutes following apomorphine (10 mg/kg, IP) administration. Subsequently, sedation gradually set in with marked sleep-like behaviour and a shift to synchrony in the EEG with spindles.

Differences in behaviour of animal species observed following morphine administration is probably due to variation in the distribution of opiate receptors. The amygdala and frontal cortex are reported to contain at least two times the level of the opiate receptor in those species that show CNS depression (e.g. dog) than in species known to show CNS excitation (e.g. cat) to opiates (Simon, 1977).

Leptazol is known to act primarily on the CNS, the medulla and the midbrain being the most responsive regions; but with larger doses the

cerebral cortex and spinal cord are also stimulated (Booth, 1982b). In the chicken both cortical and subcortical levels were markedly stimulated (Fig. 3h) and respiratory paralysis preceded death at peak convulsion. The rapid onset and potent effects of the drug justify its use as an analeptic.

The general trends of mammalian scalp EEG

(1) Reference recording

(i) Intrahemispheric montages generally showed marked similarity in wave form and phase as well as progressive anterior-posterior increase in amplitude. That was probably due to increasing distance between nasal reference electrode with the recording electrodes arranged in an anterior-posterior direction (Klemm, 1976). Often the temporal voltages were intermediate between those obtained from frontal and parietal regions (Fig. 3i(A)).

(ii) Common reference recordings were generally characterised by voltages higher in montages involving electrodes placed in the other hemisphere than from those positioned in the same hemisphere in which the reference electrode is also located. Since EEG voltage differences reflect dissimilarity of activity between brain regions of electrodes positions (Klemm, 1976),

this finding is suggestive of greater similarity within, than between, individual hemispheres. Furthermore out of- phase and dissimilar wave forms (Fig. 3j(b)) could be evidence that the two hemispheres act independently. However similar activity shown in some of the tracings probably indicates at least some common activity beteween, as well as within, the hemispheres.

Bipolar recording

(i) Intrahemispheric montages were associated with maximal and minimal potentials with parietal-frontal and occipital-parietal bipolar recordings respectively. That was indicative of greater similarity in and/or synchrony of activity generated between the parietal and occipital cortices than between either region and frontal cortex.

(ii) Transhemispheric tracings showed marked similarity in wave form with lots of in-phase activity (Fig. 3j(A)). Such montages also showed progressive anterior-posterior voltage decrease with little difference in frequency.

The uniform characteristics observed in the scalp EEG of species used in this study could be used as baseline data in clinical evaluation or for research purposes. Dominant moderate voltage and low frequency

wave forms obtained under chemical restraint in normal animals could, in diseased states, be replaced by LVFA, HVSA or HVFA. This has been shown to occur with such disorders as canine epilepsy (Croft, 1965) and encephalitis (Klemm, 1976). It must be stressed that uniform EEG activity is most reliably obtained in chemically restrained animals since the wake state is characterised by continuously changing patterns due to responses to a variety of arousal stimuli. This greatly supports the suggestion (Klemm, 1976) for, whenever feasible, the use of chemical restraint in clinical recording of the EEG in animals. Furthermore, reference to data obtained in this study is reliable, meaningful and justifiable only to EEG determined under similar conditions for the species in question.

Common reference bipolar recordings between two active cortical regions showed greater similarity in voltage and phase in intrahemispheric than transhemispheric montages (Fig. 3k). This suggests lesser differences in the activities of cortical generators of one hemisphere than in the other. This may be explained by the fact that certain nuclei and fibre tracts are not homogeneous to both hemispheres (Thomas, 1971). However, the EEG in certain fits of animals (Croft, 1965) and humans (Kool et al., 1978) is characterised by repetitive activity of variable duration over both hemispheres due to recruiting

process and/or the presence of an epileptic focus(i) in the subcortical region(s) capable of influencing the entire cortex.

Low variation in EEG frequency (Tables 3d & 3e) established in this study for dogs and cats were similarly reported by Klemm (1976). Since voltage showed greater variation than frequency it could be assumed, as suggested by Klemm (1976), that frequency change is a more sensitive indicator of brain disorder. Clinically therefore, appearance of short or prolonged abnormal changes in cortical EEG wave frequency, form, voltage and/or phase [illegible] used together in detecting encephalopathies. However, certain brain disorders can be reflected in the EEG as abnormalities of wave voltage and form, e.g. spikes, without significant alteration in frequency (Fig. 3g).

Spindles were seen in the EEG of species used in this study. In xylazine sedated sheep and goats they appeared distinctly only occasionally but most often were masked by, or formed parts of, dominant slow frequency waves. The cat had higher number of bursts/min (4 - 12) than the dog (4 - 7). Though derived from electrodes in the major cortical regions, they were most prominent in frontal and parietal recordings (Fig. 3j(A)). Spikes, sharp and triphasic waves many of which were associated with sudden motion

appeared in several tracings. Those that appeared in absence of any motion or stimulus (Fig. 3i(C)) were however considered genuine. But their appearance in the EEG of clinically normal, chemically restrained animals has been reported (Klemm, 1976).

The stereotyped synchrony in xylazine- sedated chickens, sheep and goats probably reflected general depression of the CNS thereby allowing such marked synchrony. Such waves have been ascribed to drugs such as ether and disorders like myxoedema (Kool et al., 1978).

GENERAL DISCUSSION AND CONCLUSION

Prior to this study, there had been no reports on the electrocardiogram (ECG) and electroencephalogram (EEG) of clinically normal Nigerian domestic animals. Baseline electrocardiographic and electroencephalographic data are therefore established for clinical and research uses in Nigerian indigenous domestic cat, chicken, cow dog, donkey, goat, horse and ram.

Alongside the normal patterns, the characteristic effects of myocardial infarction as well as of atropine, carbachol, chlorpromazine, digoxin, and pentobarbitone on the ECG, and due to apomorphine, halothane, leptazol, pentobarbitone and xylazine on the EEG were reproduced.

The preference for baseline ECG and EEG data on Nigerian indigenous animal species is necessitated since significant differences are known to exist even between breeds of the same species managed under the same conditions (Ettinger and Suter, 1970; Hilwig, 1976).

Veterinary clinical pharmacology aims at the rational use of drugs in veterinary clinical diagnosis and/or therapy. Veterinary electrocardiography and electroencephalography are important in the diagnosis, evaluation of therapy

and prognosis of clinical disorders of the heart (Detweiler et al., 1965; Ettinger and Suter, 1970; Hamlin, 1976) and brain (Croft, 1963; Redding et al., 1966; Klemm, 1976) respectively.

Both electrocardiographic and electroencephalographic data obtained from this study are similar to those of previous works conducted on exotic breeds (McCrady et al., 1966; Ettinger and Suter, 1970; Prynn and Redding, 1968; Klemm, 1976). Therefore the ECG and EEG criteria established for the diagnosis of heart and brain disorders in exotic breeds could still be cautiously applied in our poorly characterised indigenous breeds.



Fig. 3b: Alert chicken twenty-four hours post-implantation of brain electrodes.

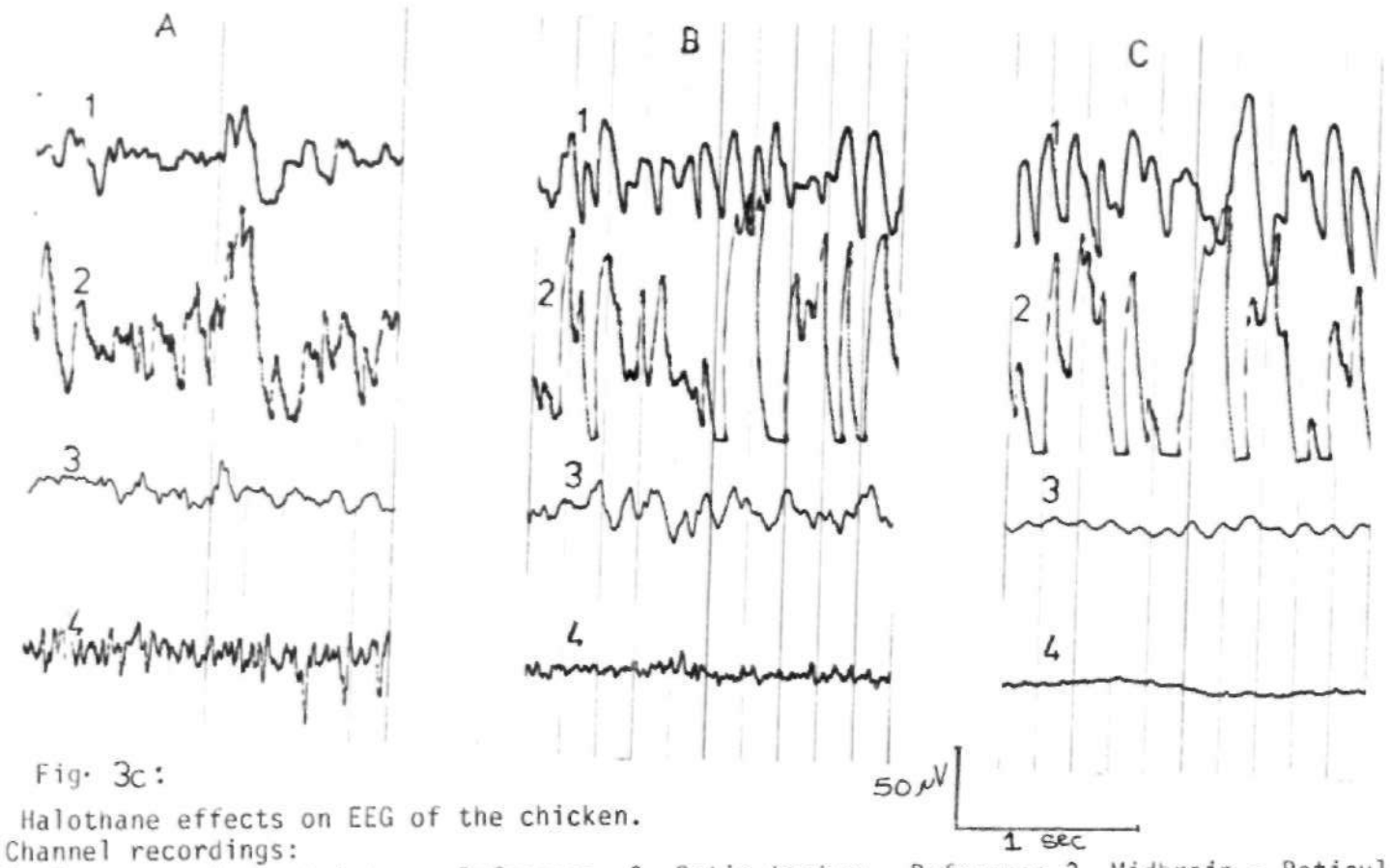
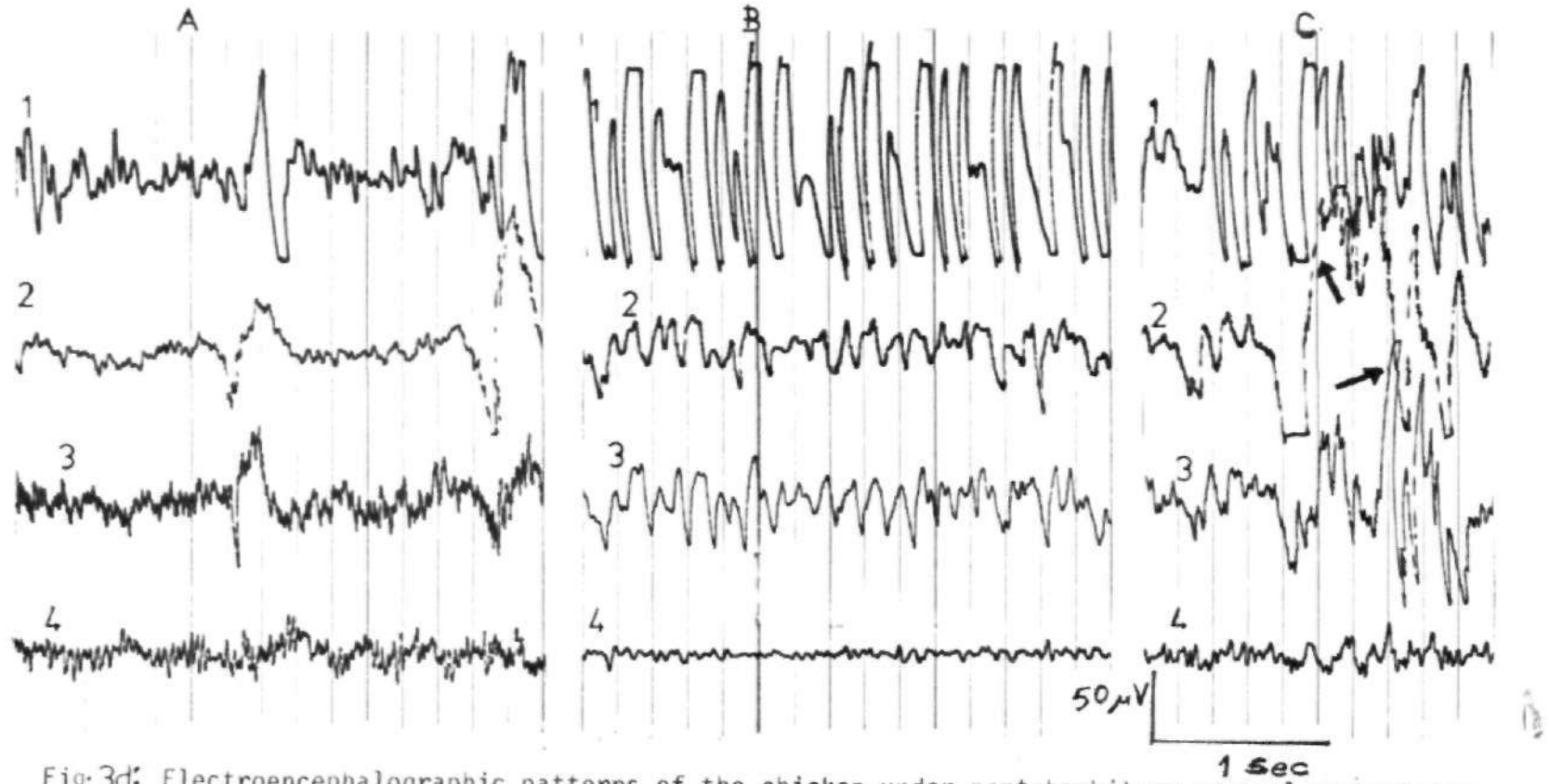


Fig. 3c:

Halothane effects on EEG of the chicken.
Channel recordings:
1: Accessory Hyperstriatum - Reference 2. Optic tectum - Reference 3. Midbrain - Reticular formation
4. Electromyogram
Patterns: A: awake, sternal recumbency B: halothane-sedated, abnormal posture C: halothane general anaesthesia, lateral recumbency NOTE (A through C): progressive synchronisation of the EEG in the cortical tracings (1), decrease in subcortical activity (2 & 3) and reduction in muscle tone (4).



Fig·3d: Electroencephalographic patterns of the chicken under pentobarbitone general anaesthesia.
Electrode designation in fig. 3c.

A: Desynchronised EEG and occasional sharp wave transients characterise the wake, alert normal state.

B: The anaesthetized state. Note the marked synchrony in the cortical and subcortical activities (1, 2 & 3), as well as the extensively reduced muscle tone (4).

C: Features of recovery include relative desynchrony and sharp high voltage transients (arrows).

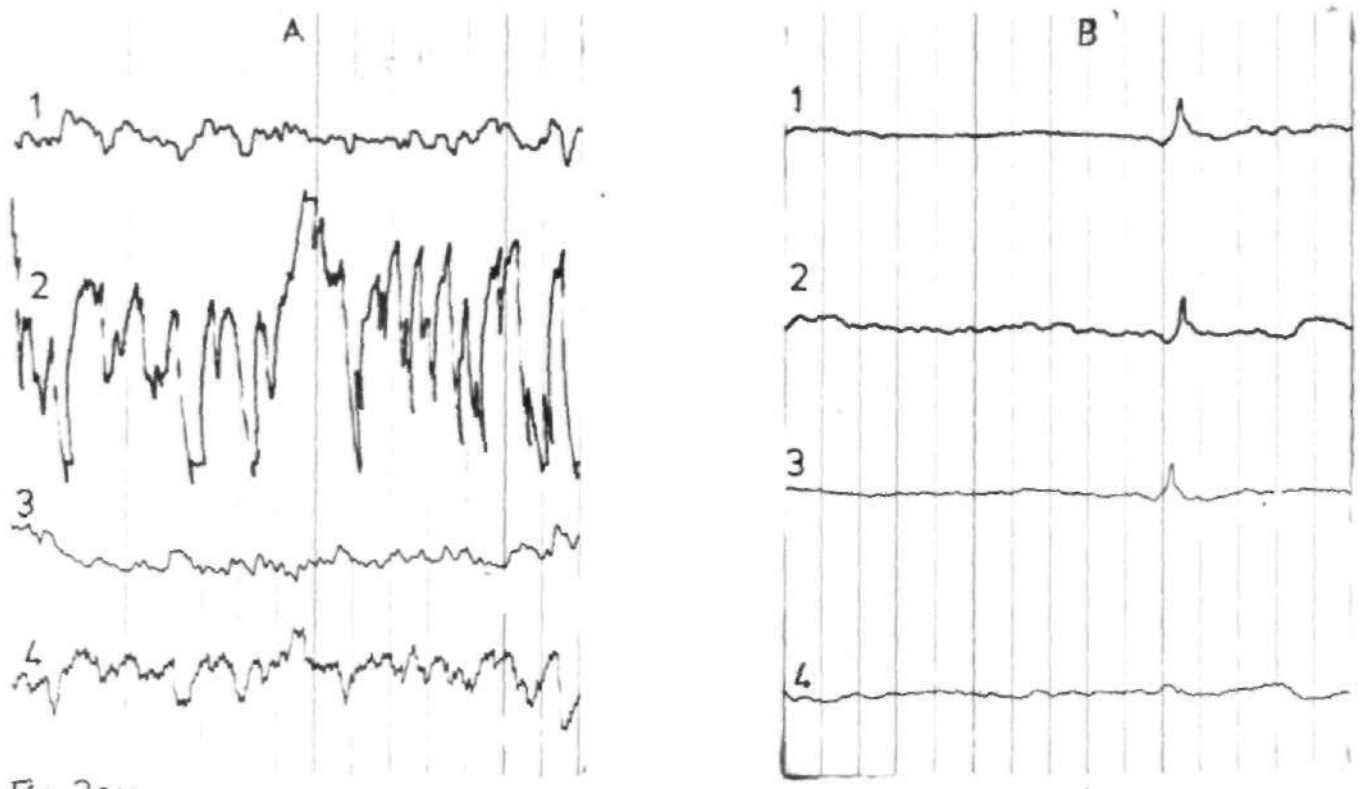
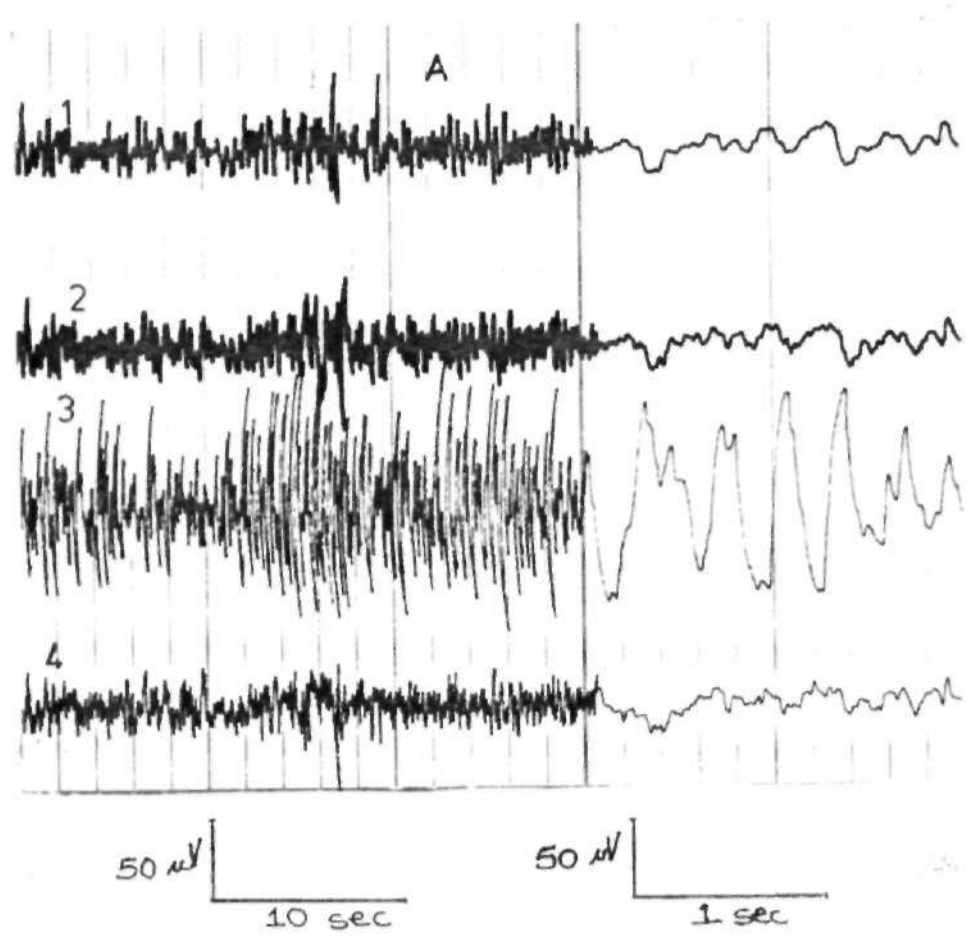
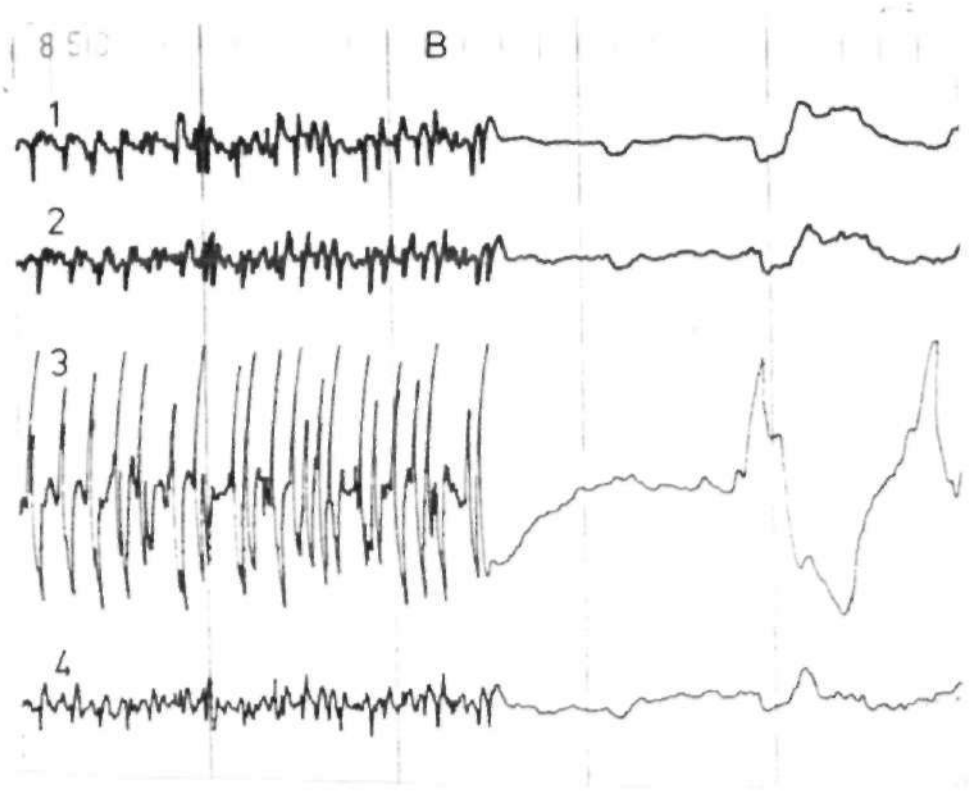
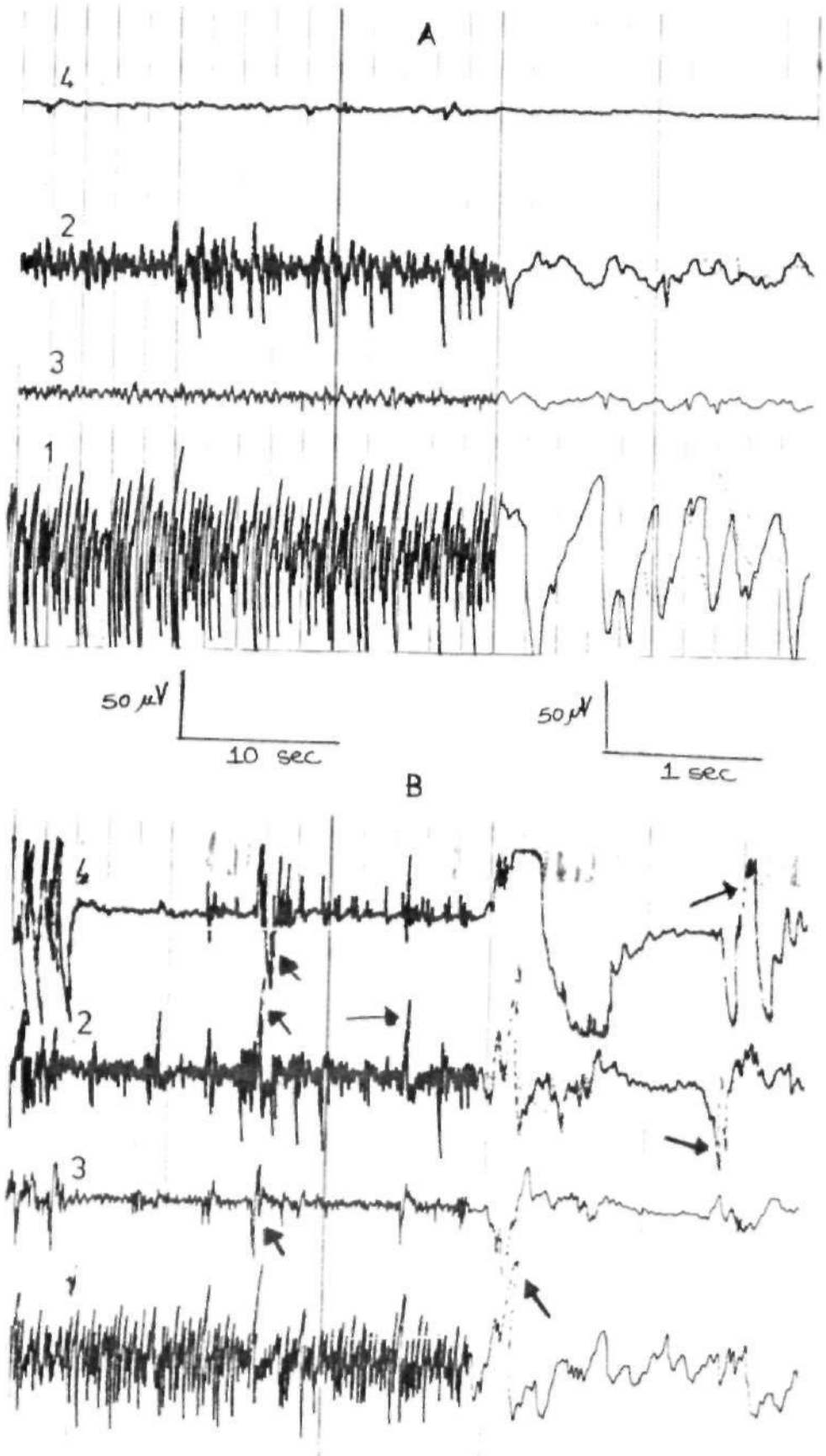


Fig. 3e:
Features of anaesthetic overdose on the chicken EEG.
Electrode positions as in fig. 3c
A: Activity of the standing alert normal chicken.
B: Persistent electric silence in the EEG and EMG after 15 minutes of pentobarbital overdose (50 mg/ kg, IP).


A
1
2
3
4
50 µV
10 sec
50 µV
1 sec


B
1
2
3
4


A
4
2
3
1
50 µV
10 sec
50 µV
1 sec
B
4
2
3
1

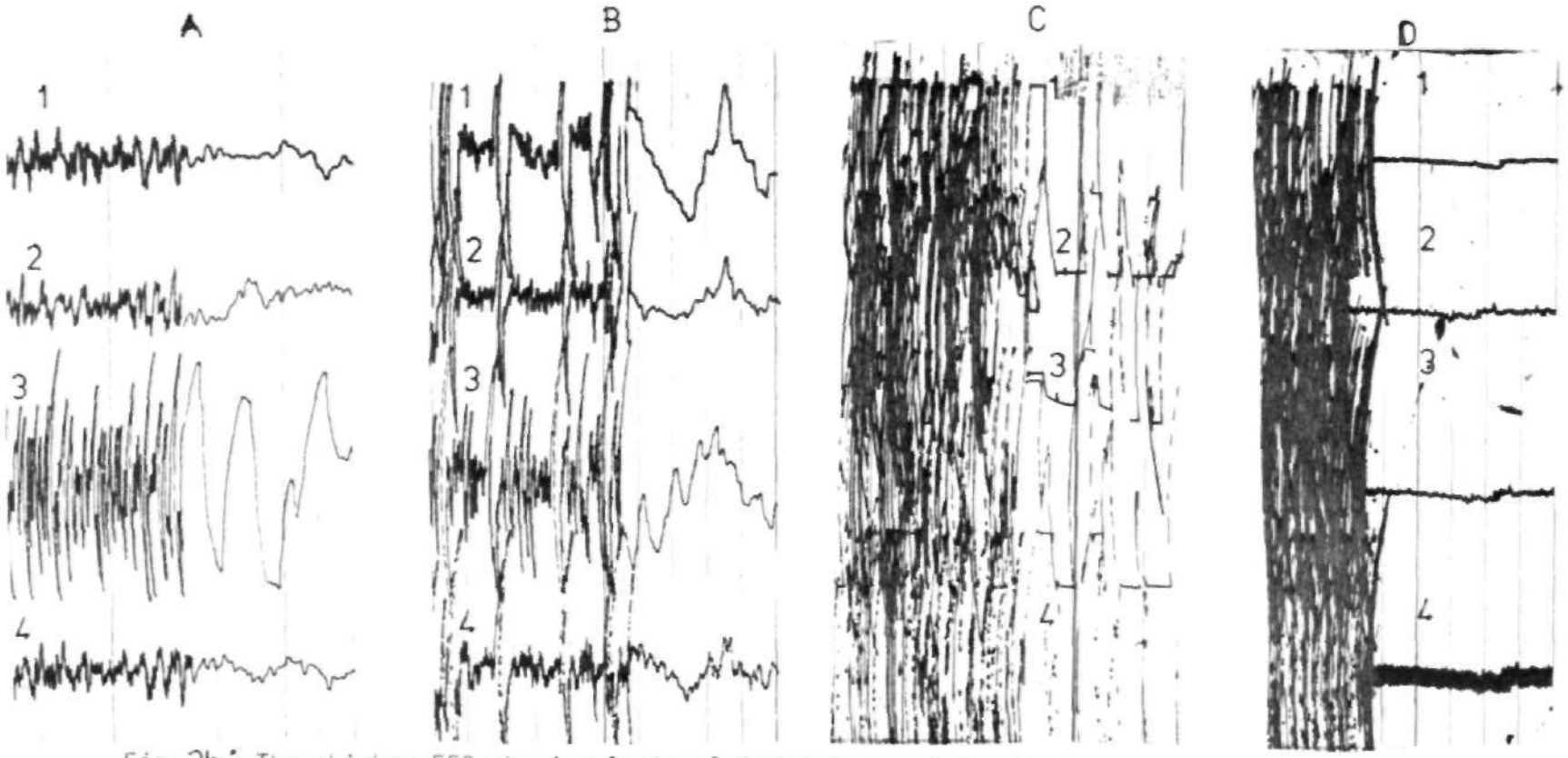
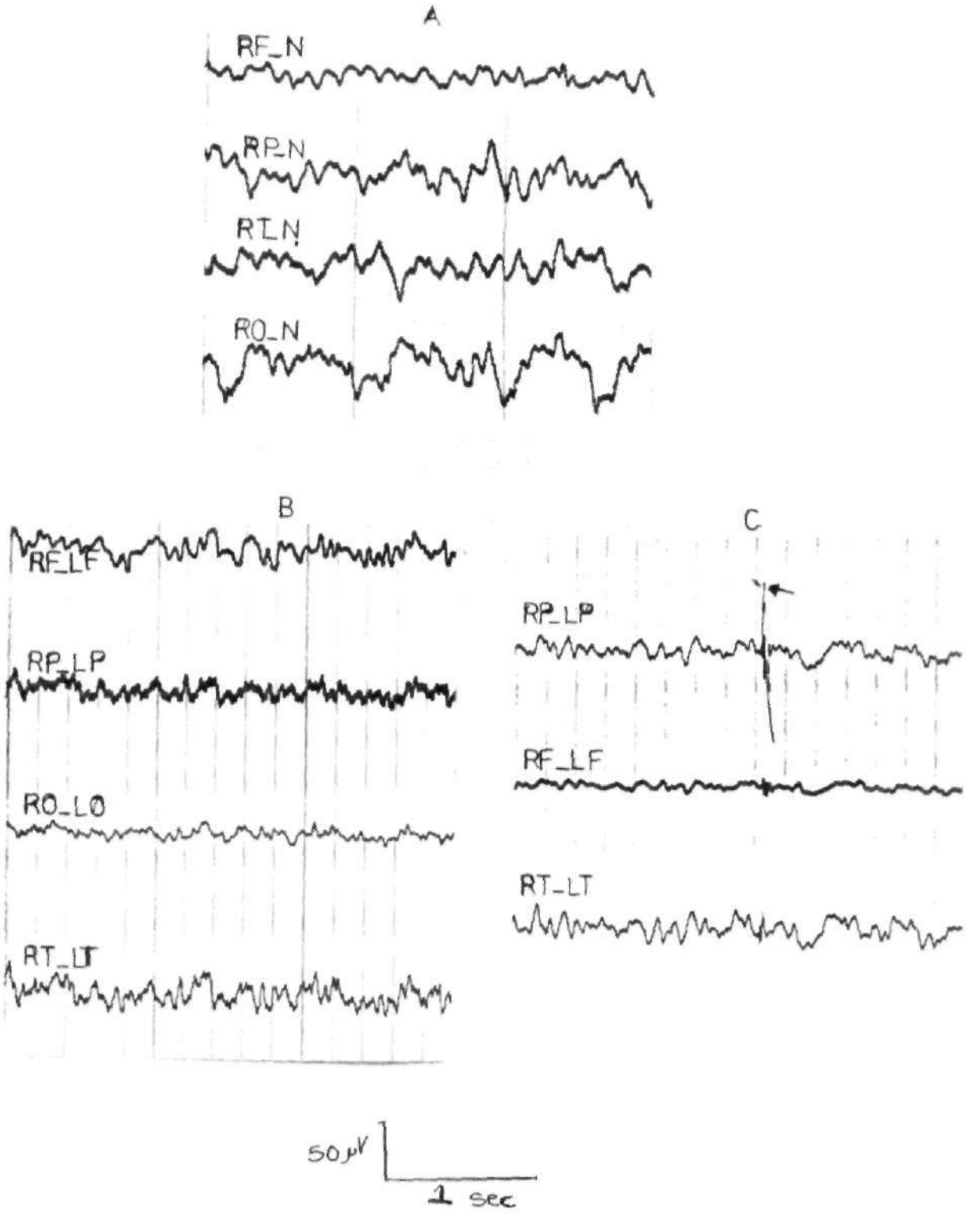


Fig. 3h: The chicken EEG showing leptazol-induced convulsive features.
Electrode designations as in fig. 3c.
A: Normal drowsy state B: Mild convulsive state; note the frequent discontinuous spikes and paroxysms C: Marked convulsive state; continuous fits of high voltage waves D: Peak convulsive state- more severe than in C; sudden cessation of electrical activities accompanied by respiratory paralysis occur terminally. NB Paper speeds for each tracing are:

in A, B and C: initial (slow) - 2.5 mm/sec
succeeding (fast) - 25 mm/sec

D: uniform (fast) - 25 mm/sec.


A
RF_N
RP_N
RT_N
RO_N
B
RF_LF
RP_LP
RO_LO
RT_LT
C
RP_LP
RF_LF
RT_LT
50 μV
1 sec

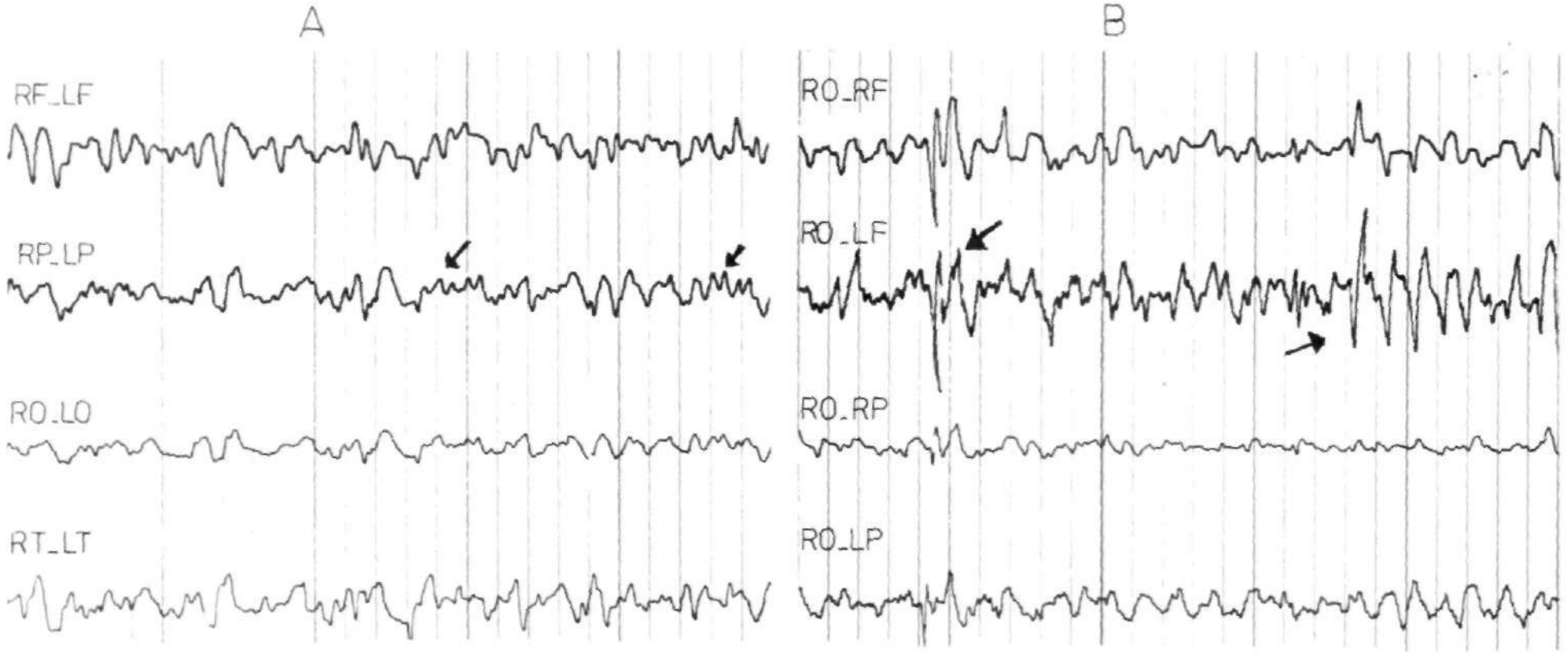


Fig. 3j: The electroencephalogram of the anaesthetized cat (thiopental-pentobarbitone mixture). Electrode designation as in fig 3i .

A: Transhemispheric bipolar recording indicating similarity in wave forms, in-phase activity and anterior-posterior decrease in amplitude. Arrows indicate spindles.

B: Common reference bipolar recording; pattern indicates incomplete similarity between the two hemispheres. Arrows indicate spikes occurring in association with sudden body jerks.

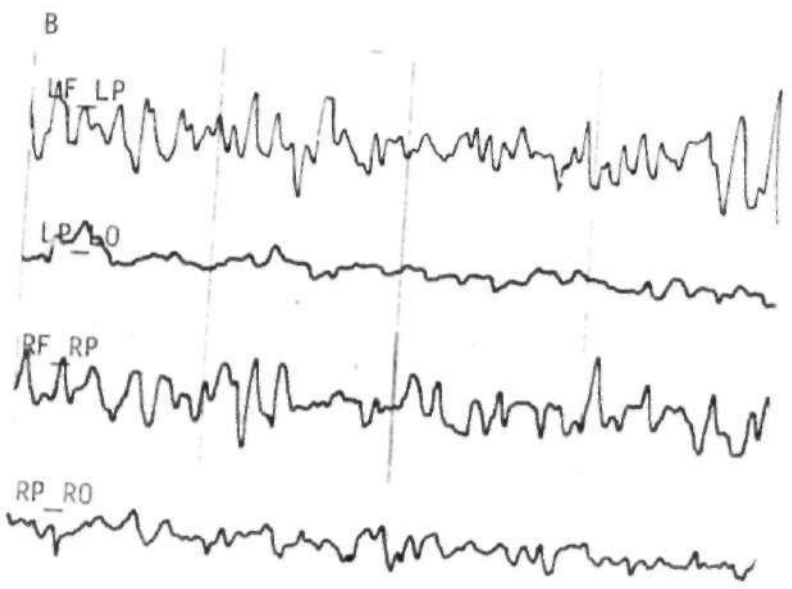
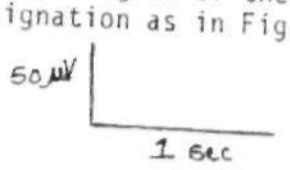


Fig. 3j (i) The electroencephalogram of the anaesthetised cat. Electrode designation as in Fig. 3i.



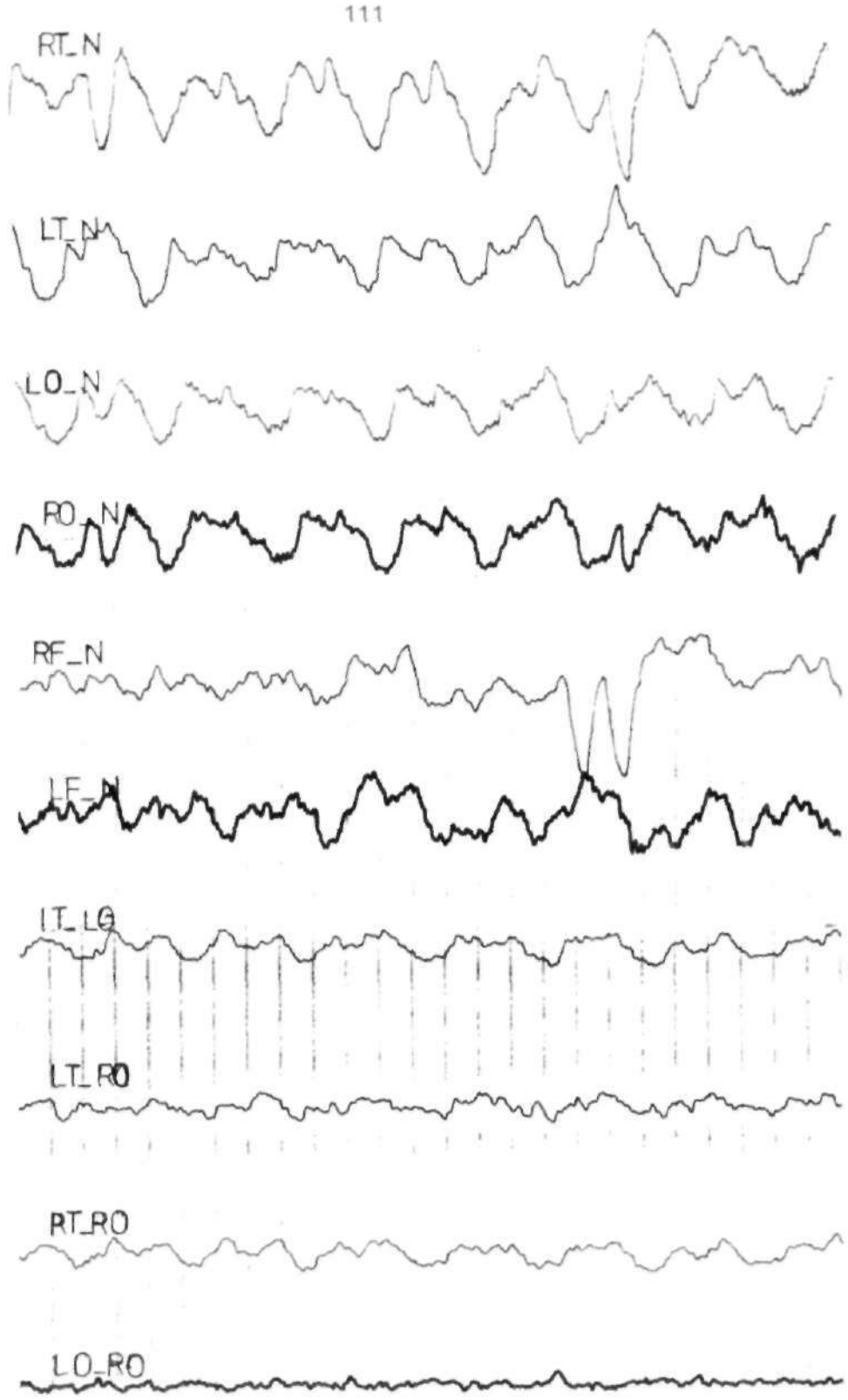


Fig. 3k : . The electroencephalogram of the sheep under xylazine sedation. Electrode designation as in fig. 3i. . In the reference tracings (upper six) - note marked in-phase activity and similarity in wave forms especially between corresponding cortical regions of the two hemispheres. The bipolar (lower four) tracings show that there is greater difference in activity between two electrodes placed in dissimilar cortical regions and hemispheres (LT_RO), than those placed in corresponding areas of the two hemispheres (LO_ RO).

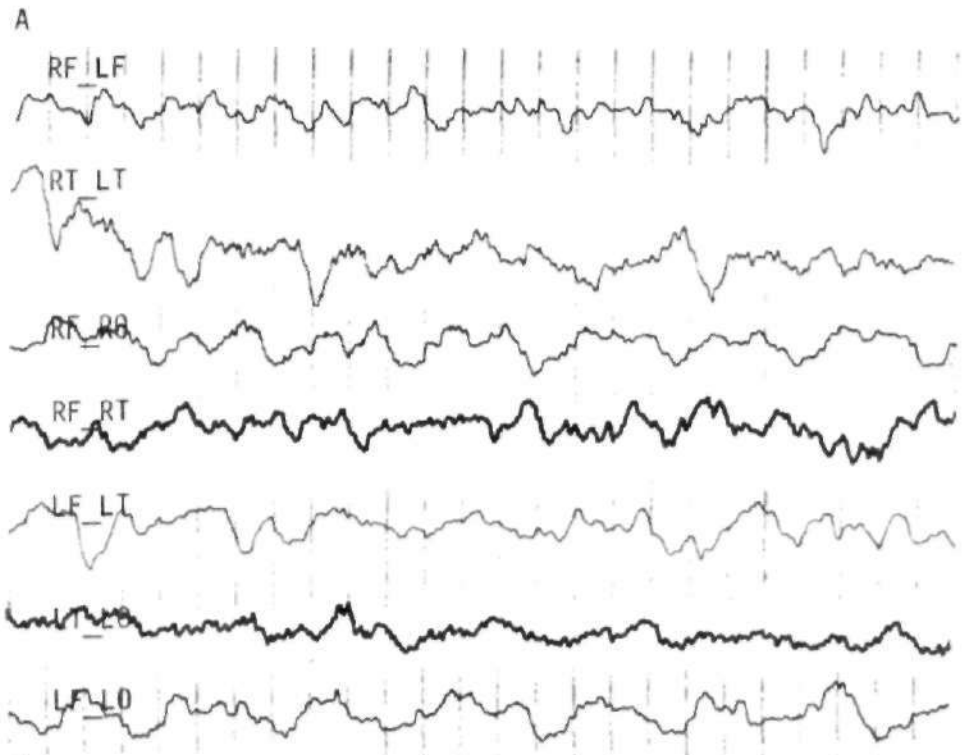


Fig. 3k(a) The electroencephalogram of the sheep under xylazine sedation. Electrode designations as in Fig. 3i.

Table 3a : Voltage and Frequency characteristics of the scalp electroencephalogram of adult normal dog, cat, horse and pig.

Animal		Restraint	Voltage (μV)	Frequency (wps)
dog[a]	21	Pentobarbital sodium (IV)	2 to 40	4 to 20
cat[b]	16	Thiopental-Pentobarbibital equal-volumes mixture (IV)	3 to 80	2½ to 20
horse[c]	25	Xylazine (IV)	5 to 90	0.5 to 40
pig[d]	51	Halothane	5 to 70	2 to 26

Source: [a] Klemm, 1968; [b] Beaver and Klemm, 1973; [c] Mysinger et al., 1985; [d] Rose et al., 1972

Table 3b: Advantages and disadvantages of three means of restraining animals in recording the electroencephalogram*

Disadvantages	Physical Restraint (no drugs)	Muscle Relaxants	General Anaesthetics
Muscle artefact	+	-	-
Motion artefact	+	-	-
Time consuming	+	+	-
Low voltages, difficult analysis	+	+	-
Mixed arousal, sleep; interpretation problems	+	+	-
Poor contrast between awake electroencephalogram and abnormal low voltage fast activity	+	+	-
No activation by disinhibition	+	-	-
Hospitalization required	-	-	+
Hazardous	-	+	+
Masking of encephalographic abnormalities	-	-	+?

*Source: Klemm, W.R. (1976). Applied Electronics For Veterinary Medicine And Animal Physiology·Charles C Thomas, Springfield, Illinois.

Table 3c: Correction factors for implanting electrodes in brains of chickens

	Anterior-Posterior	Lateral	Vertical
Accessory Hyperstriatum	+3.8 mm	-3.4 mm	-
Optic Tectum	-2.4 mm	-5.9 mm	-
Reticular Formation	-2.0 mm	+1.5 mm	-8.1 mm

Table 3d: Dominant electroencephalographic activity of indigenous dogs in surgical anaesthesia: $\bar{O} = 7$

MONTAGE	WAVE CHARACTERISTICS	
	Voltage (uV)	Frequency (wps)
Reference		
L. Frontal	5 - 15	6 - 8
R. Frontal	5 - 25	6 - 7
L. Temporal	15 - 50	6 - 7
R. Temporal	10 - 50	6 - 7
L. Parietal	15 - 50	6 - 8
R. Parietal	15 - 50	6 - 7
L. Occipital	15 - 75	5 - 8
R. Occipital	10 - 70	5 - 7
Bipolar		
L. Frontal - R. Frontal	5 - 45	4 - 11
L. Temporal - R. Temporal	5 - 25	6 - 8
L. Parietal - R. Parietal	10 - 25	4 - 7
L. Occipital - R. Occipital	5 - 20	4 - 9
L. Frontal - L. Parietal	5 - 35	6 - 10
L. Parietal - L. Occipital	10 - 35	6 - 9
R. Frontal - R. Parietal	10 - 30	6 - 8
R. Parietal - R. Occipital	10 - 25	5 - 7

Left (L); Right (R)

Table 3e: Dominant electroencephalographic activity of indigenous cats in surgical anaesthesia; n = 7

MONTAGE	WAVE CHARACTERISTICS	
	Voltage (uV)	Frequency (wps)
Common Reference		
R. Parietal - R. Frontal	5 - 60	5 - 7
L. Parietal - R. Frontal	10 - 80	6 - 8
R. Occipital - R. Frontal	5 - 55	5 - 7
L. Occipital - R. Frontal	5 - 50	6 - 8
R. Temporal - R. Frontal	5 - 50	5 - 7
L. Temporal - R. Frontal	10 - 75	4 - 6
Bipolar		
R. Frontal - L. Frontal	10 - 50	4 - 7
R. Parietal - L. Parietal	10 - 35	4 - 8
R. Occipital - L. Occipital	5 - 25	3 - 5
R. Temporal - L. Temporal	10 - 35	4 - 6
L. Frontal - L. Parietal	10 - 70	5 - 7
L. Parietal - L. Occipital	5 - 30	4 - 6
R. Frontal - R. Parietal	5 - 70	5 - 7
R. Parietal - R. Occipital	5 - 25	5 - 6

Left (L); Right (R)

Table 3f: Dominant electroencephalographic activity in sedated indigenous sheep; n=7

MONTAGE	WAVE CHARACTERISTICS	
	Voltage (uV)	Frequency (wps)
Reference		
R. Frontal	10 - 30	3 - 4
L. Frontal	5 - 30	4 - 5
R. Temporal	10 - 35	3 - 5
L. Temporal	10 - 60	2 - 5
R. Occipital	10 - 60	3 - 5
L. Occipital	10 - 65	3 - 5
Bipolar		
R. Frontal - L. Frontal	5 - 75	4 - 8
R. Temporal - L. Temporal	15 - 50	3 - 5
R. Occipital - L. Occipital	3 - 6	4 - 7
L. Frontal - L. Temporal	5 - 60	4 - 5
L. Temporal - L. Occipital	5 - 20	3 - 5
L. Frontal - L. Occipital	5 - 35	2 - 4
R. Frontal - R. Temporal	5 - 30	4 - 6
R. Temporal - R. Occipital	10 - 50	2 - 4
R. Frontal - R. Occipital	10 - 35	2 - 4

Left (L); Right (R)

Table 3g: Dominant electroencephalographic pattern of sedated indigenous goats; n = 7

MONTAGE	WAVE CHARACTERISTICS	
	Voltage (uV)	Frequency (wps)
Reference		
R. Frontal	15 - 85	3 - 4
L. Frontal	10 - 35	3 - 7
R. Temporal	10 - 40	3 - 5
L. Temporal	10 - 60	3 - 5
Occipital	10 - 30	3 - 5
Bipolar		
R. Frontal - L. Frontal	5 - 20	4 - 7
L. Temporal - R. Temporal	5 - 20	4 - 6
R. Frontal - Occipital	10 - 120	4 - 5
L. Temporal - Occipital	10 - 40	4 - 5
L. Frontal - R. Temporal	10 - 40	3 - 5

Left (L); Right (R)

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