ELECTRONIC ASPECTS OF MEASURING ELECTROCARDIOGRAMS DURING THREE PHASES OF CORONARY CARE

by

MICHAEL FREDERICK DIPROSE

A Thesis submitted for the degree of Doctor of Philosophy
in the Faculty of Natural Sciences at the
University of Kent at Canterbury

CANTERBURY 1976

This thesis is dedicated to my Parents

and my wife, Wendy

ACKNOWLEDGEMENTS

Throughout the years that I have been studying the material for the thesis, I have had much assistance and support from many people both at the University and the Hospital. My thanks go especially to my Supervisor, Dr. R.J. Collier, from whose active enthusiasm, encouragement and guidance I have benefited enormously throughout the course of the Ph.D. I also owe many thanks to Dr. D.J.E. Taylor and Dr. B.E. Crawley of the Kent and Canterbury Hospital whose assistance, suggestions and patience have been invaluable.

I should also like to thank the Director and Staff of The Electronics Laboratories at the University and the Principal Physicist and Staff of the Medical Physics Department at the Hospital for all their help and the use of facilities, and also the Technical Staff of both places for their advice and assistance. Thanks are also due to the E.C.G. Department at the Hospital for their friendly and helpful cooperation over the years.

I should like to thank Miss D. Paine for all her hard work in typing the manuscript and all her assistance in producing the thesis and also Mrs. M. Fowler for labelling the diagrams.

M. F. DIPROSE

CANTERBURY

1976

INTRODUCTION

The growth in the use of electronics in medicine has been considerable during the past few years. Under the name of Bio Engineering, Medical Physics or Medical Electronics, the number of units connected with hospitals and Universities has proliferated. The work undertaken by the various groups is extensive, ranging from the development of small pieces of equipment for use in laboratories[1],[2],[3], to large projects such as routine electrocardiogram analysis, involving large computers and research teams[4],[5].

A measure of the extent of the growth emerged from the controversy sessions at the 15th International Bio Engineering Conference held in Edinburgh this year[6]. Some senior members of the Biological Engineering Society such as Mr. Heinz Wolff and Mr. Keith Copeland and others, stated their belief that now is the time for the creation of a formal profession of Bio Engineering where proper training of candidates at Universities and Hospitals replace the more random modes of entry into the field that exist at the moment. The effects of the advent of professionalism will remain to be seen, especially since at the moment the mixture of people involved i.e. clinicians, electronic and mechanical engineers and surgeons, provide an enthusiastic and fertile soil for a rich growth of ideas of projects.

Perhaps there is too much enthusiasm, however, since in spite of their optimism about the achievments of the discipline and its future, the speakers did introduce a

note of caution. They suggested that all projects should be looked at closely and critically before they are started to try to ascertain their real value and contribution to patient welfare. Small, uncomplicated systems such as implanted cardiac pacemakers, steel pins for bone support, incontinence devices, hearing aids etc., all bring immediate and great relief to large numbers of people, and this was suggested as an important criterion, rather than the pursuit of very expensive projects - perhaps for their own sake. Mr. Heinz Wolff summed it up by stating "at all costs we must avoid a Concorde in Bio Engineering"[7].

This sense of caution was continued in a later paper by Professor Taylor of the University of Edinburgh. Sufficient experience has now been gained in Bio Engineering to allow some retrospective reflections on its impact and usefulness and to examine some of its effects more closely. Studies in the field of patient monitoring have been extensive, and now there is a multitude of electrocardiogram amplifiers, blood pressure monitors, temperature recorders, etc., for a physician to choose from. Until recently their usefulness has been unquestioned, but now Professor Taylor has thrown some doubt onto the subject, which should make all bio engineers think about their work. This arose from his paper[8] when he talked about his experiences when comparing measurements taken by nursing staff with those of electronic measuring apparatus. (This is developed more fully later in the thesis), and could well undermine some of the work undertaken by large groups on extensive, automatic, routine patient monitoring.

These comments were only meant to be cautionary, and to make an engineer think as much as possible before acting. Certainly the future of Bio Engineering looks bright, and electronics engineers and technicians seem to have an assured rôle in the Health Service in the future.

The situation at Canterbury has developed in the last five years due to the keenness and enthusiasm of young consultant staff both at the Hospital and the University. Ever since the opening of the latter in 1965, close cooperation between it and the Hospital has been planned, and this has materialised in the form of work done in this thesis and also by other University departments[9].

It was requested that some electronics be developed to assist the clinicians in their care of patients, right from the earliest possible moments of cardiac distress, through their time in hospital, to the final assessment of their health when they leave. The ambulance telemetry, ward and exercise telemetry and the electrode work was that chosen ~~and which was proposed, and which~~ to form the material for this thesis.

Since initial training was purely as an electronics engineer, it was decided as necessary to include in the work presented a summary of basic medical knowledge that an engineer working with cardiac and intensive therapy units, ought to have, to enable him to understand his work more fully. Accordingly, Chapter 1 consists of the anatomy and physiology of the heart and explains its important place in the circulation of the blood, and hence the life of the system. Chapter 2 is an explanation of the origin and characteristics of the electrocardiogram, and develops its important place in the diagnosis

of cardiac complaints. This is necessary to explain why the transmission of the electrocardiograms from one place to another forms most of the work studied and presented in this thesis. To anyone who is medically qualified or experienced in cardiac anatomy, there is no need to read Chapter 1, or the beginning of Chapter 2.

Mobile Coronary Care Units and the early treatment of cardiac arrythmias form the subject matter of Chapter 3 and 4. The first of the two deals with the concepts and types of mobile units, and proposes a system for the Kent and Canterbury Hospital, whilst the second concerns the electronics developed to enable the proposed system to operate.

An aspect of patient care whilst actually in the Hospital is dealt with in Chapter 5, and is the development of an electrocardiogram transmitter to enable the patient monitoring to be more reliable. Its use as a tool in assessing patient condition and prognosis is also demonstrated, so that information on convalescent period and working capabilities may be obtained.

Chapters 6 and 7 both deal with another idea of providing more reliable patient monitoring. Movement artefact in electrocardiogram recording has long been a nuisance, and has occasioned many, many false alarms from monitoring apparatus relying upon this signal as an input. Chapter 6 investigates the theoretical and Chapter 7 the practical aspects of using specially compensated, low input impedance, electrocardiogram amplifiers instead of the normal high input impedance ones, to reduce this artefact.

In conclusion, Chapter 8 will consist of a summary of the work done, and there will be discussion on both the future

developments of the projects, and on some of the aspects of the rôle of a bio engineer in the provision of patient care systems.

REFERENCES

(1) M.F. DIPROSE "Distilled water tank-level detector to automatically operate a pump" 1975. Project for the Pathology Department, Kent and Canterbury Hospital.

(2) D.T. DELPY "A catheter tip pressure transducer" 1975. Bio Medical Engineering, Vol. 10, No. 1.

(3) O.Z. ROY & R.W. WEHNERT "A simple pulse to pulse cardiac tachometer" 1974. Medical and Biological Engineering, Vol. 12, No. 3.

(4) J.M. NIELSON "A special purpose hybrid computer for analysis of E.C.G. arrythmias" 1971. Proceedings - Conference on Computers for analysis and control in medical and biological research. Sheffield 7-9th Sept., p. 151.

(5) P.W. McFARLANE & T.D.V. LAWRIE "An introduction to automated electrocardiogram interpretation" 1974. Published by Butterworths.

(6) BIOLOGICAL ENGINEERING SOCIETY - 15th Anniversary International Conference 1975. Edinburgh, Scotland.

(7) H.S. WOLFF "Bio engineering in society" Session 1, reference (6).

(8) PROF. D. TAYLOR "Decision making in intensive care" Session 16, reference (6).

(9) The Computing Laboratories and the Health Services Research Unit, The University of Kent.

CONTENTS

Page No.

CHAPTER 1

1.1 HISTORICAL IMPRESSION OF THE FUNCTION OF THE HEART

The existence of the heart as an organ has been known for many centuries[1] although a correct interpretation of its function has only been made comparatively recently[2]. The ancient Egyptian civilisations believed that the only purpose of the heart was as the site of the soul and some of the first cardiac anatomical drawings are to be found carefully painted on the walls of the tombs of the Kings. These show the Gods, Horus, Anubis and Thoth, balancing the dead Pharohs' heart on scales against good and evil, to discover whether or not he should continue with his voyage into the Underworld[3],[4].

Greek medical practitioners such as Hippocrates and Eristratus held a theory that a person's spiritual life force or "pneuma" was pumped into the body by the heart, but they did not postulate the circulation, for the pneuma was a spiritual force, not the blood[5],[6],[7].

It was not until 1628 when Harvey published his work on the circulation of the blood "De Motu Cordis and Sanguinis in Animalibus" that the true nature of the heart as the active centre of the circulatory system was realised; that the blood in an animal body is impelled in a circle and is in a state of ceaseless motion, that this is the act or function which the heart performs by means of its pulse and that it is the sole and only end of the motion and contrac-

tion of the heart"[2],[5]. He also dismissed the belief that was held for centuries concerning the heart's spiritual function - "there is, in fact, no occasion for searching after spirits foreign to or distinct from the blood, to evoke heat from any other source, to bring the Gods upon the scene and to encumber philosophy with any fanciful deceits. What we want to derive from the stars, is in truth, produced at home - the blood is the only caladium innatum - or first engendered animal heat"[2],[5].

The last three centuries have seen major advances in the knowledge of anatomy and physiology and the purpose of the circulatory system as the pathway for the continuous flow of substances necessary for metabolism, is now realised. The heart, with its vital function as the main pump lies at the centre of this system.

1.2 CELLS AND METABOLISM

The life of a system, i.e. a human being, can be considered to be the sum total of activities of that system[8] and for life to continue there must be an unceasing expenditure of energy during these activities.

This energy is derived from the molecular reactions taking place within the cells of that system. The cells are the smallest units in the human body that have a structured function, and when provided with a continuous supply of the necessary nutrients and fuels, are able to organise their internal activities so as not only to maintain their existence, but also to reproduce themselves[9],[10],[11].

The cells group together to form specialised tissues such as nerve and muscle and these tissues comprise the organs which perform the necessary functions of the human body for example, the heart, liver and kidneys. The blood is continually supplied with oxygen and metabolic fuels and its circulation ensures that these are brought into close contact with the cells. The blood also takes the waste products ~~through~~ of cellular metabolism away to the organs which are equipped to render them non-toxic and to discharge them from the body[12].

The actual transfer of metabolic fuels and products, between the cells and the blood is due to the steep concentration gradients of the substances existing across the cell membrane[12],[13],[14] *(Figure (1))*. The need for a fast and efficient delivery of oxygen can be illustrated by the fact that interruption of the blood supply to the brain causes unconsciousness in seven seconds[15].

1.3 THE CIRCULATION

The human circulatory system is divided into two main sections : the pulmonary and the systemic circulations *(Fig. (2))*. The purpose of the former is to pass the blood through the lungs and back to the heart and then the systemic circulation passes the blood around the body through the arterial system to nourish the cellular mechanisms. It returns to the heart via the venous system to pass to the pulmonary circulation once again to complete the cycle[16],[17].

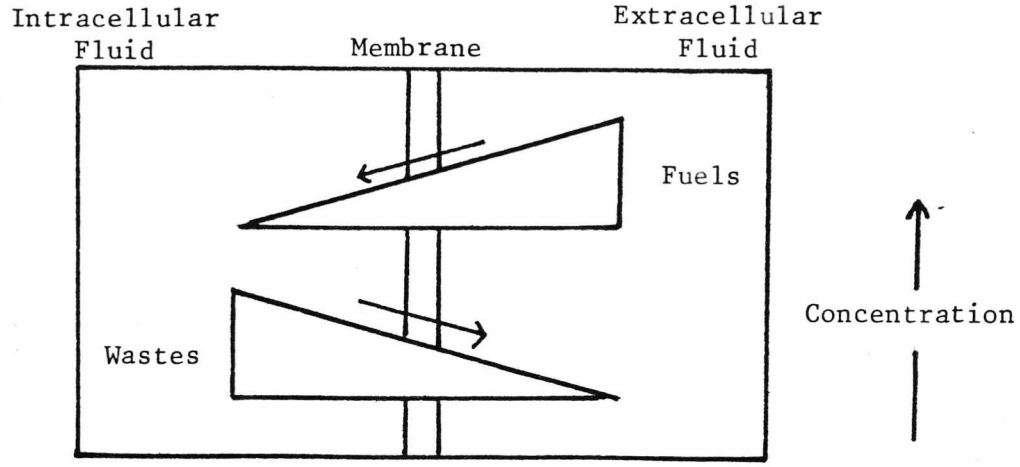


FIG. (1)

The Transfer Of Fuels and Wastes Due To Concentration Gradients

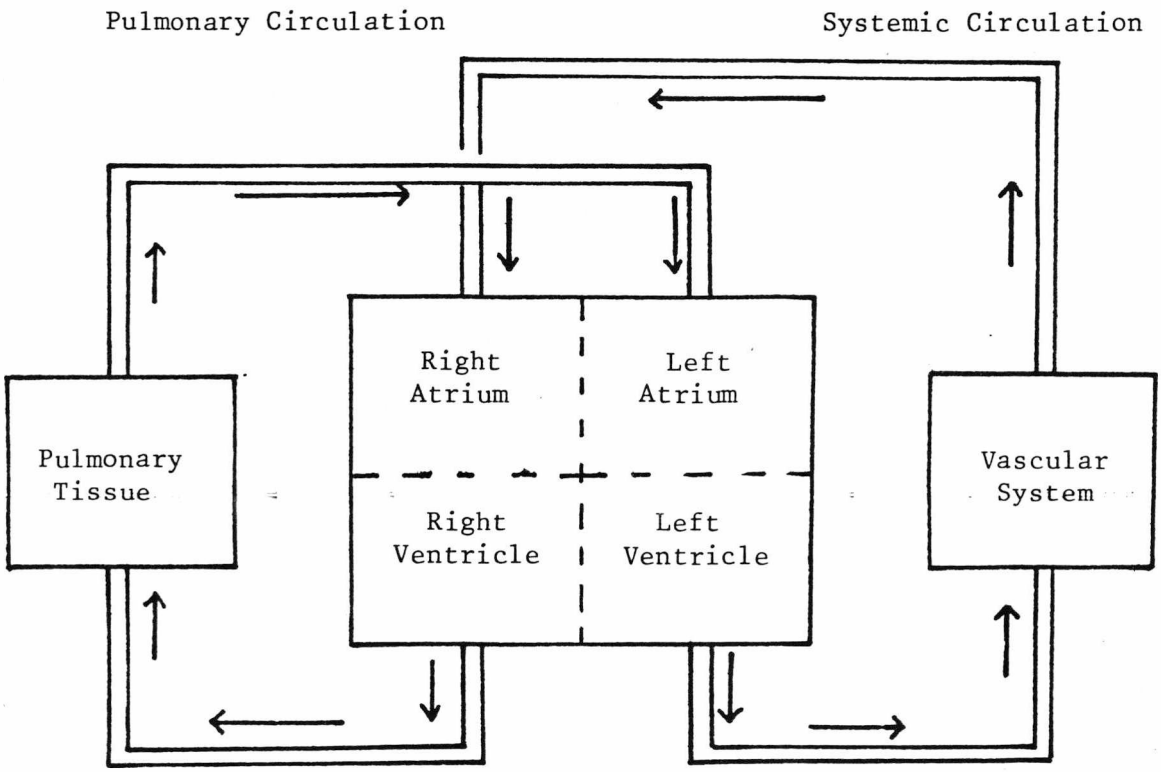


FIG. (2)

The Systemic and Pulmonary Circulations

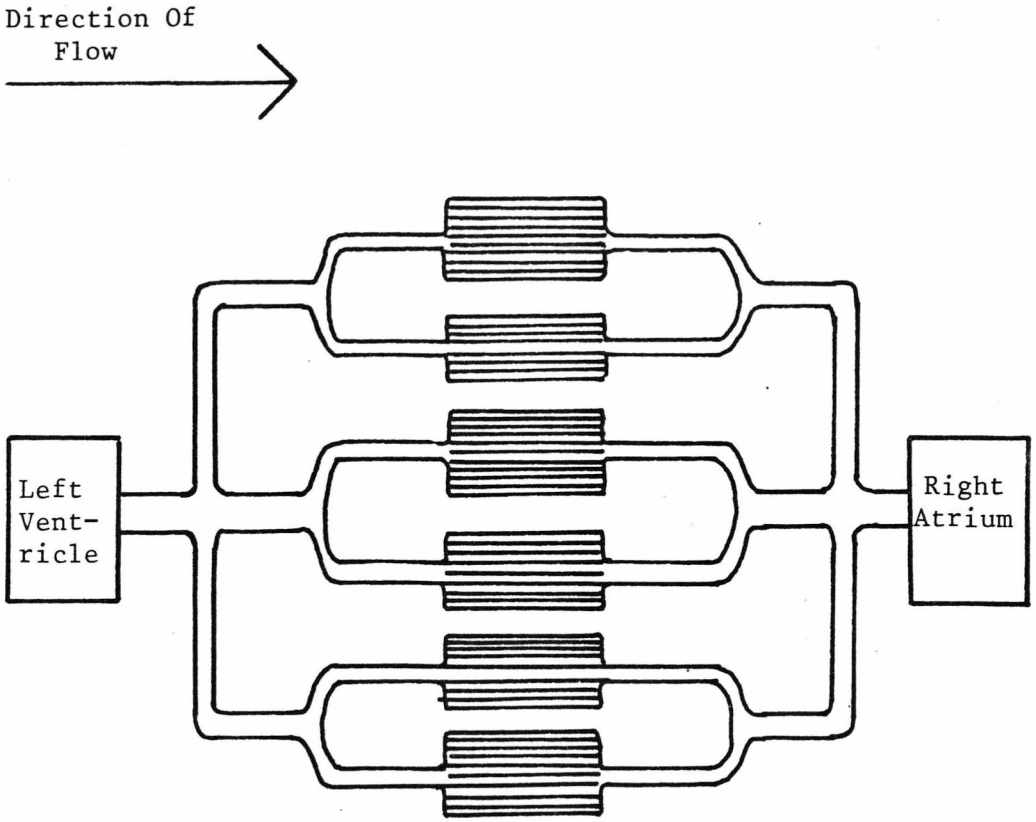


FIG. (3a)

The Various Sections of the Systemic Circulation

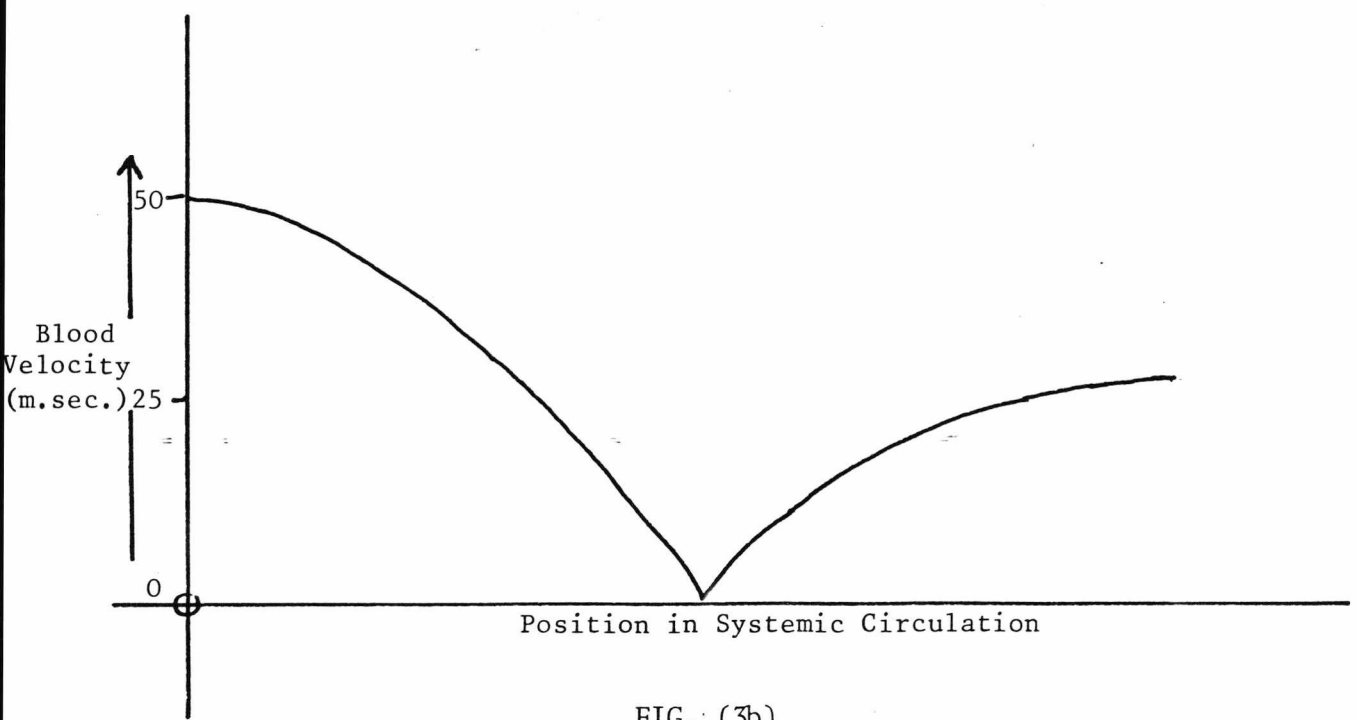


FIG. (3b)

Blood Velocity Throughout the Systemic Circulation (this diagram has the one above as its position reference)

A typical systemic pathway consists of an artery which branches into arterioles which, in turn, lead to the capillaries. These drain into the venules which connect to the veins and then return to the right atrium of the heart. The arterioles are the vessels which control the flow to the organs. They constrict or dilate both by central nervous system control or by local control and by so doing they alter the hydraulic resistance of their part of the circuit and so control the quantity of blood flowing through it[16]. The capillaries are the exchange vessels and nutrients and waste products diffuse across their walls to and from the cells. It has been estimated that there are approximately 10 billion capillaries in the body with a total surface area of 100 square metres, and that each cell is within 20 μm to 30 μm of a capillary. Since the total cross sectional area of the capillary system is 500 to 600 times that of the supplying artery *(Fig. (3a))* the flow rate through the capillaries is very slow and enables the necessary diffusions to take place[18] *(Fig. (3b))*.

The pulmonary circulation does not have the need for flow regulation and so pulmonary arterioles do not exist[19]. The arteries branch directly into the capillaries which perfuse the pulmonary tissue and enable the gas exchange mechanisms to take place.

The surface area of the pulmonary capillaries has been estimated at 70 to 80 square metres with capillaries between 0.36 μm to 3.5 μm from alveolar air[19].

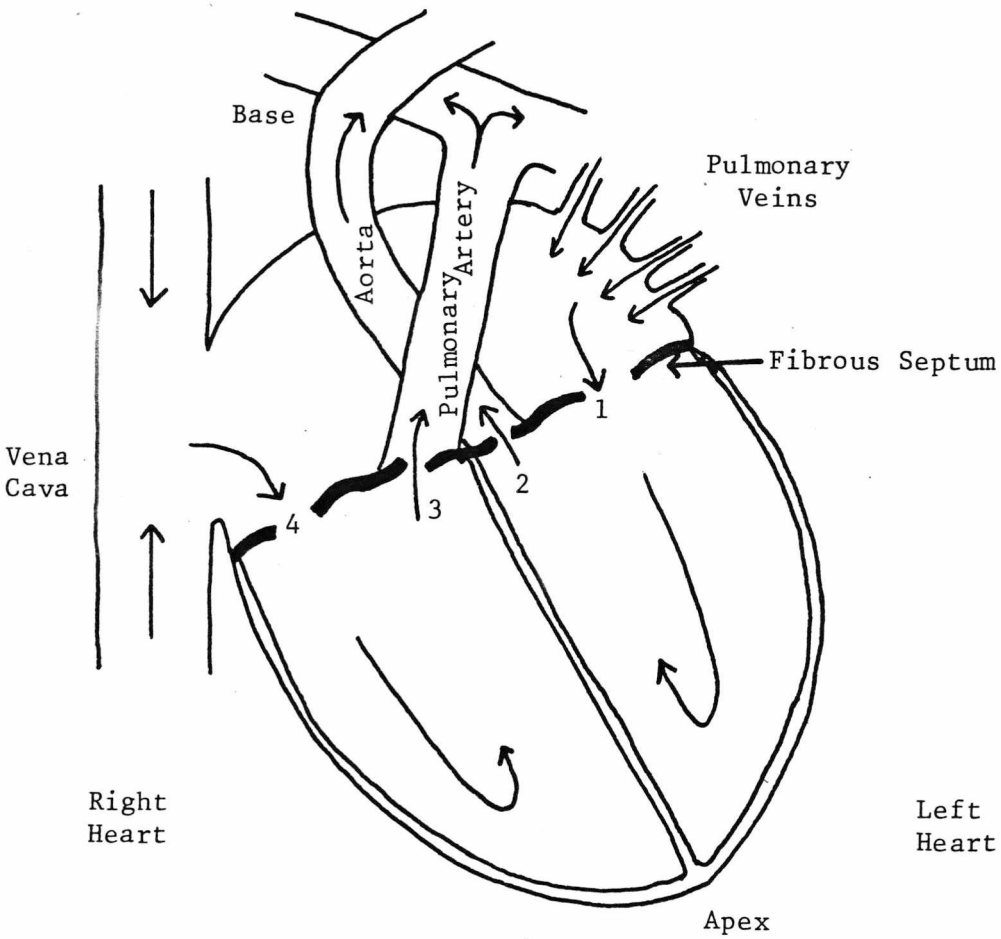
1.4 THE ANATOMY OF THE HEART

The overall shape of the heart is of an inverted cone with the main blood vessels connected[20] at the top, and the apex pointing towards the diaphragm.

The musculature of the heart is in two sections separated by a tough fibrous ring - the septum, within the plane of which lie the four main heart valves[21] *(Fig. (4))*. Above the septum are the two atria, and below it lie the two ventricles. The walls of the atria and the interatrial septum separating the two are thin, since only low pressures (e.g. 7 mm.Hg average) are developed upon contraction[21]. The walls of the ventricles are much thicker with the right ventricle crescent shaped which is suitable for pumping large volumes of blood at low pressures (e.g. 13 mm.Hg average) whilst the cylindrically shaped left ventricle is able to develop the high pressure (e.g. 100 mm.Hg average) needed to impel the blood through the systemic circulation[22] *(Fig. (5))*.

The left and right atria are connected to their corresponding ventricles by the mitral and tricuspid valves respectively, whilst the left and right ventricles are connected to the systemic and pulmonary circulations by the aortic and pulmonary valves. All the valves are unidirectional flap type and are passive in operation - that is they open and close according to the resultant force determined by the pressures either side of the valve[23],[24].

Cardiac muscle cells are striated in appearance - like those of skeletal muscle, but the contractile properties are similar to those of smooth muscle i.e. when the heart



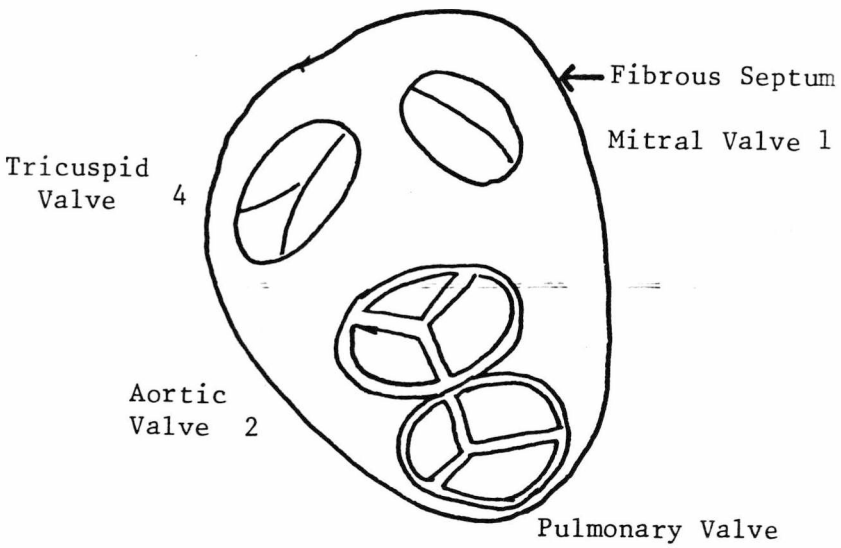


FIG. (4)

The Flow System Through the Heart, and the Heart Valves in the Fibrous Septum

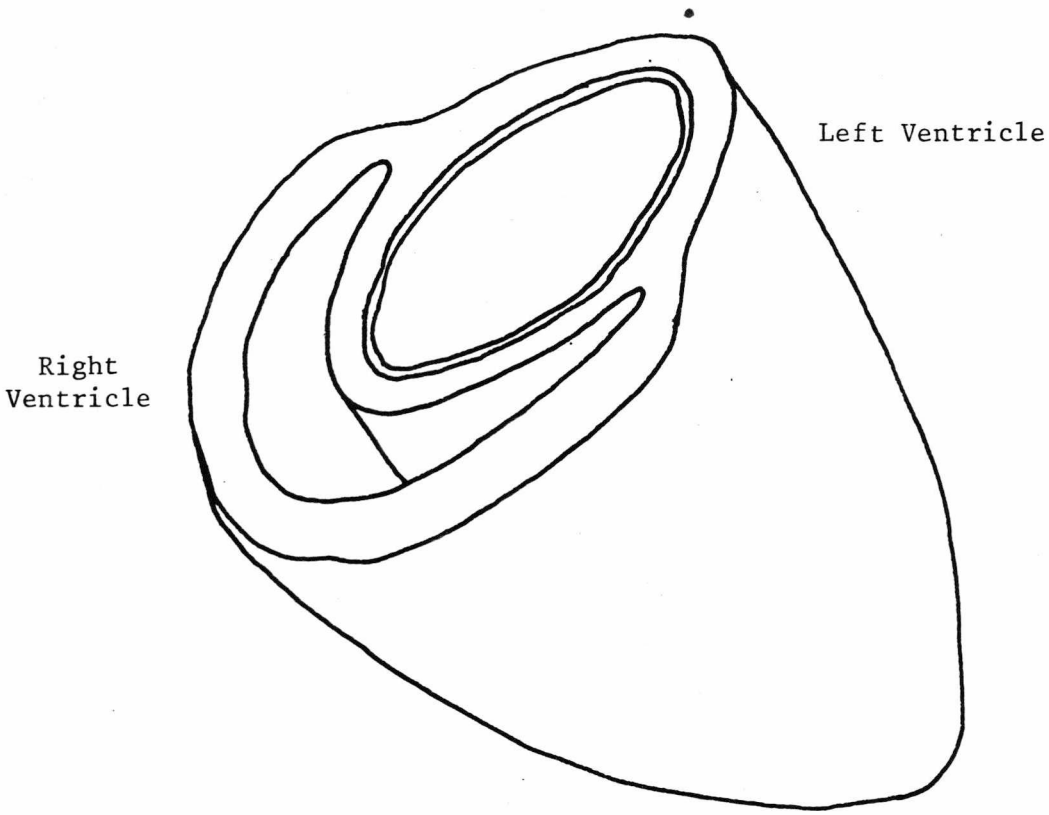


FIG. (5)

The Shapes of the Right and Left Ventricles

muscle contracts it does so as a syncytium. This is because the spread of electrical activity throughout the muscle (which is always associated with muscular contraction) does not depend upon the activation of each cell by a motor nerve - but is due mainly to cell-to-cell conduction. Although each one is surrounded by a membrane of high resistivity (2000 Ω.cm), good electrical contact between cells is made at the intercalated discs[25], where the resistivity is only 5 Ω.cm. The intercalated discs are formed where the membranes of adjacent cells make very close contact by having complicated interlocking surfaces[25] *(Fig. (6))*.

Since the fibrous septum acts as an insulator between the atria and the ventricles, special conductive tissues are needed to convey the electrical signals concurrent with muscular contraction between the two. This tissue is known as the atrio ventricular (A-V) node.

Following the A-V node is a distribution system, to enable the ventricular myocardium to contract efficiently. The Bundle of His connects to the A-V node and then this splits into three branches - one to the right ventricle, and two to the left. These are known as the right bundle branch and the left bundle branches respectively. At the end of these is a network of fibres which spread throughout the myocardium called Purkinje Fibres. The excitation wave for the atria relies upon cell-to-cell conduction only[26] *(Fig. (7))*.

1.5 THE MEMBRANE POTENTIALS OF CARDIAC MUSCLE

1.5a The Membrane Potentials

The membranes of cells constituting excitable tissues

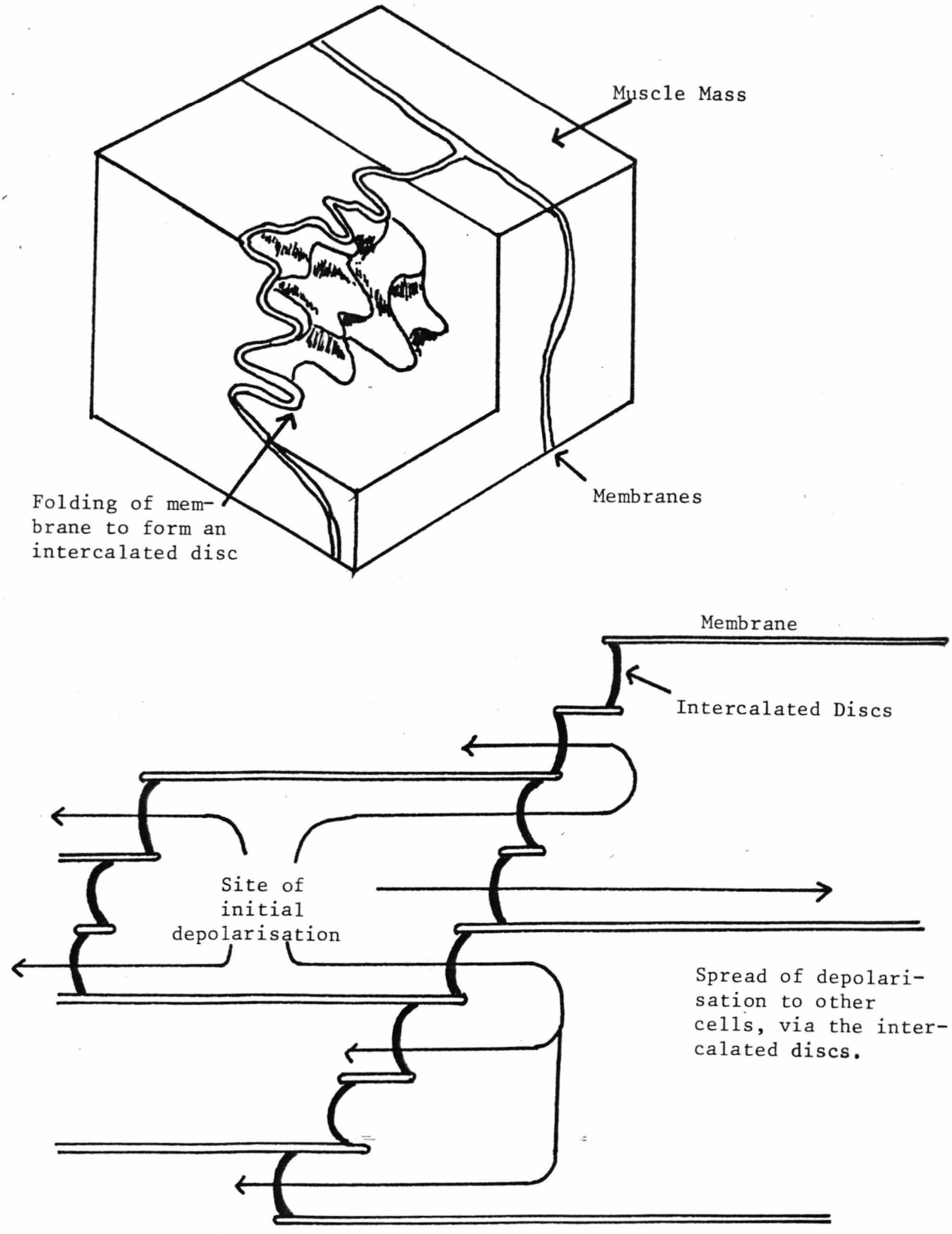


FIG. (6)

The Intercalated Discs between muscle cells and their function in the spread of depolarisation

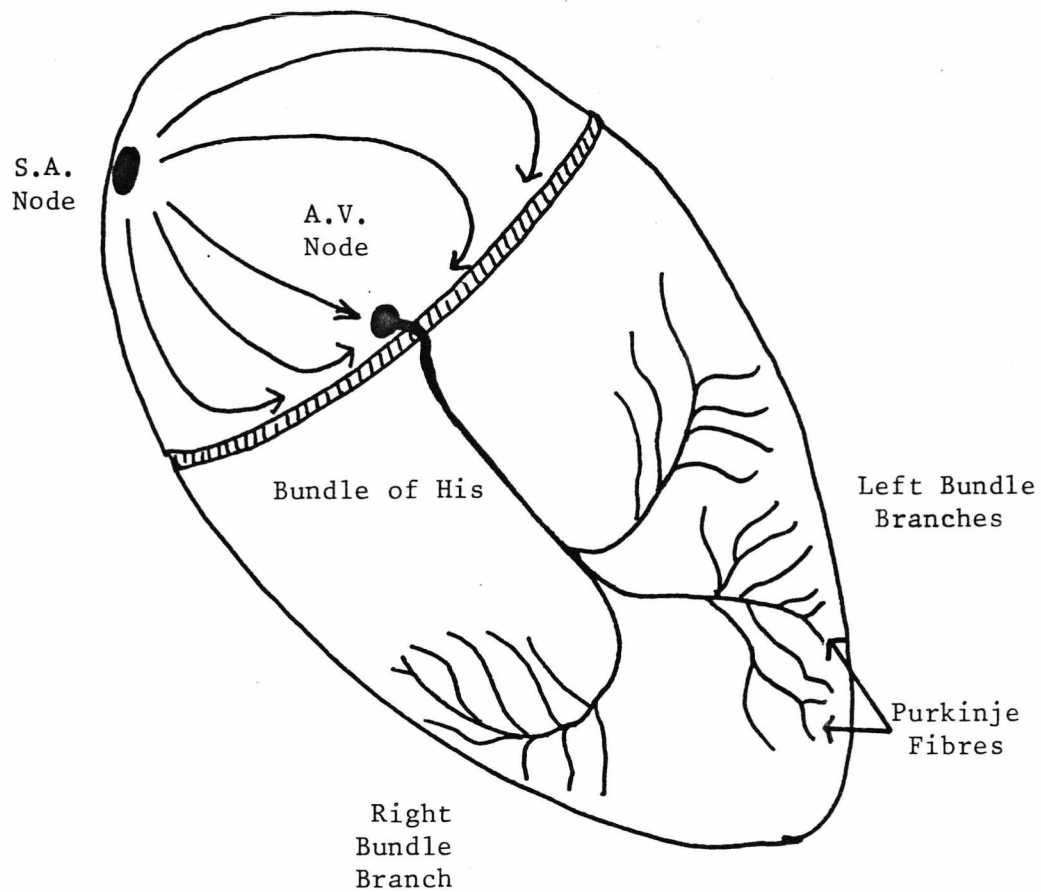


FIG. (7)

The Pathway of the Cardiac Excitation Process

such as nerve and muscle, are semi-permeable to certain ion species. This property and the metabolically controlled distribution of ions between the intracellular fluid and extracellular fluids, generate a potential across the membrane. The size varies from tissue to tissue and in cardiac muscle the resting potential is in the order of 90 mV with the inside of the cell negative with respect to the outside[27].

When a muscle cell is excited and contracts, the first event is a change in potential across the membrane. This is called depolarisation and the inside of the cell rapidly becomes 20 or 30 mV positive to the outside. This is followed by a plateau phase, and it is during this time that the cell actually begins to contract in length. After a period of time (about 200 m.secs.) the potential automatically reverts to its original value and this process is called repolarisation *(Fig. (8))*, and the muscle tension slowly begins to decrease. The time of the whole excitation process, or action potential, is 250 m.secs. to 300 m. secs., and varies according to the heart rate i.e. 250 m.secs. at 75 beats per minute and 150 m.secs. at 200 beats per minute. This is much longer than the action potentials of skeletal muscle cells - whose durations are only of a few m.secs. This does enable cardiac muscle to exert a long, steady force upon the blood to eject it efficiently into the circulations. This process can be compared with postural control where much finer movements of the muscle are needed. The short action potential enables a wide range of firing rates to occur, and so the degree of shortening of the muscle can be finely controlled[28],[29].

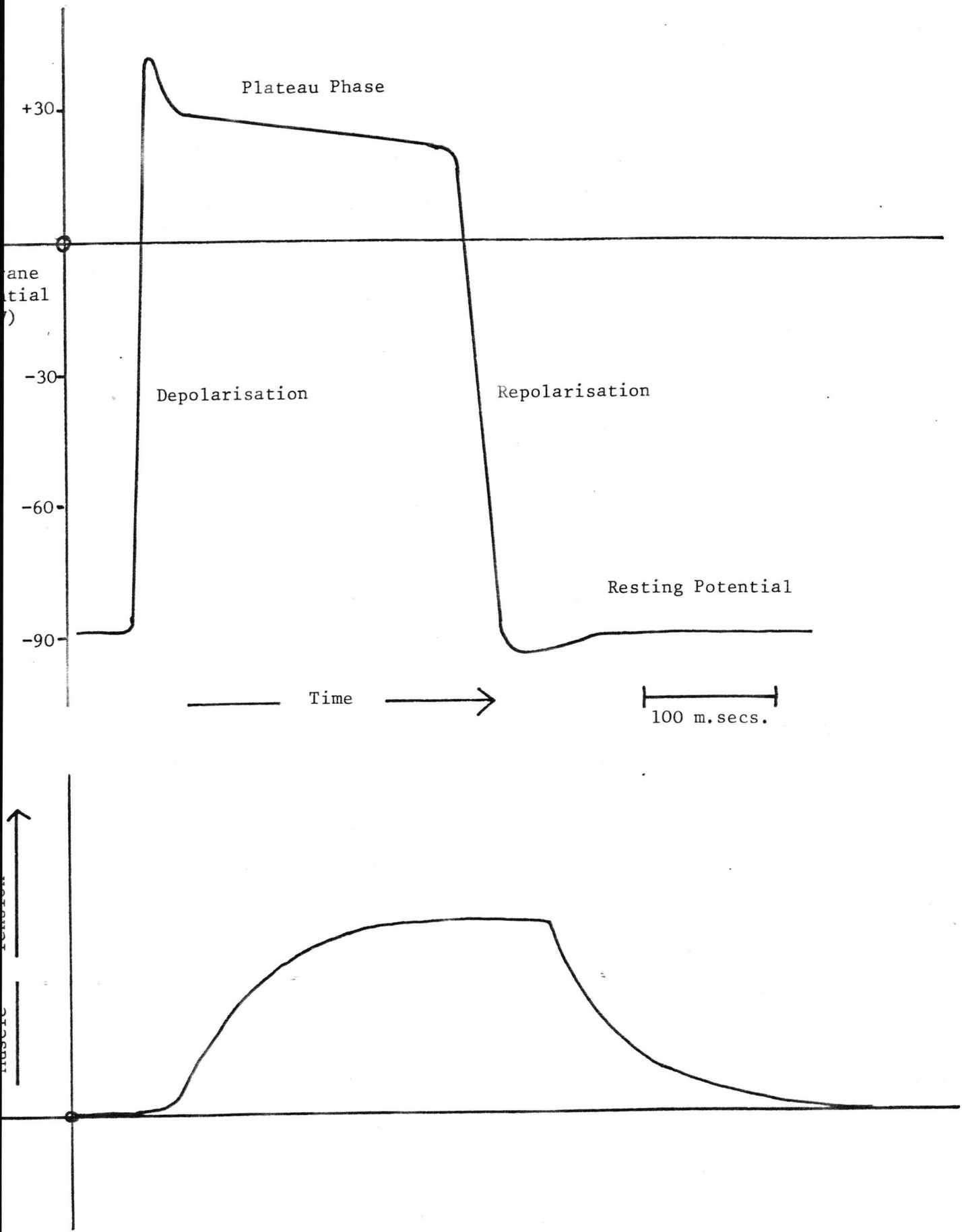


FIG. (8)

The Relation between Excitation of a Muscle Cell, and the resulting changes in tension

There is a degree of difference in the duration of the action potentials within different parts of the myocardium itself, *(Fig. (9))*, with atrial muscle having shorter periods than ventricular muscle. Certain cells do not have a constant resting potential, instead they have a steadily decreasing potential called the prepotential *(Fig. (10))*. These are the pacemaker cells and are self-exciting and, as will be seen later in section 1.6, control the rate of beating of the heart[29].

Before an action potential can occur the membrane has to depolarise a certain amount to its threshold level. Above this, an action potential occurs, but if the stimulus is sub-threshold - nothing happens at all[28],[30] *(Fig. (11))*.

1.5b The Ionic Theory Of Membrane Potentials

The normal situation of the cardiac cell is that the concentration of intracellular potassium ions is 30 to 40 times greater than that of sodium ions. Conversely, the extracellular sodium ion concentration is greater than that of the potassium ions *(Fig. (12))*, and also there is a difference in the ionic conductances through the membranes, with that of potassium being higher[31],[32]. The chlorine ions adjust themselves to prevailing conditions since their permeability is extremely high, and they migrate to and fro to maintain an electrical balance in the intracellular and extracellular fluids.

The potential across the membrane is almost equal to that given by the Nernst equation for potassium ions alone[31] *(Fig. (12))*, and although there are several ion

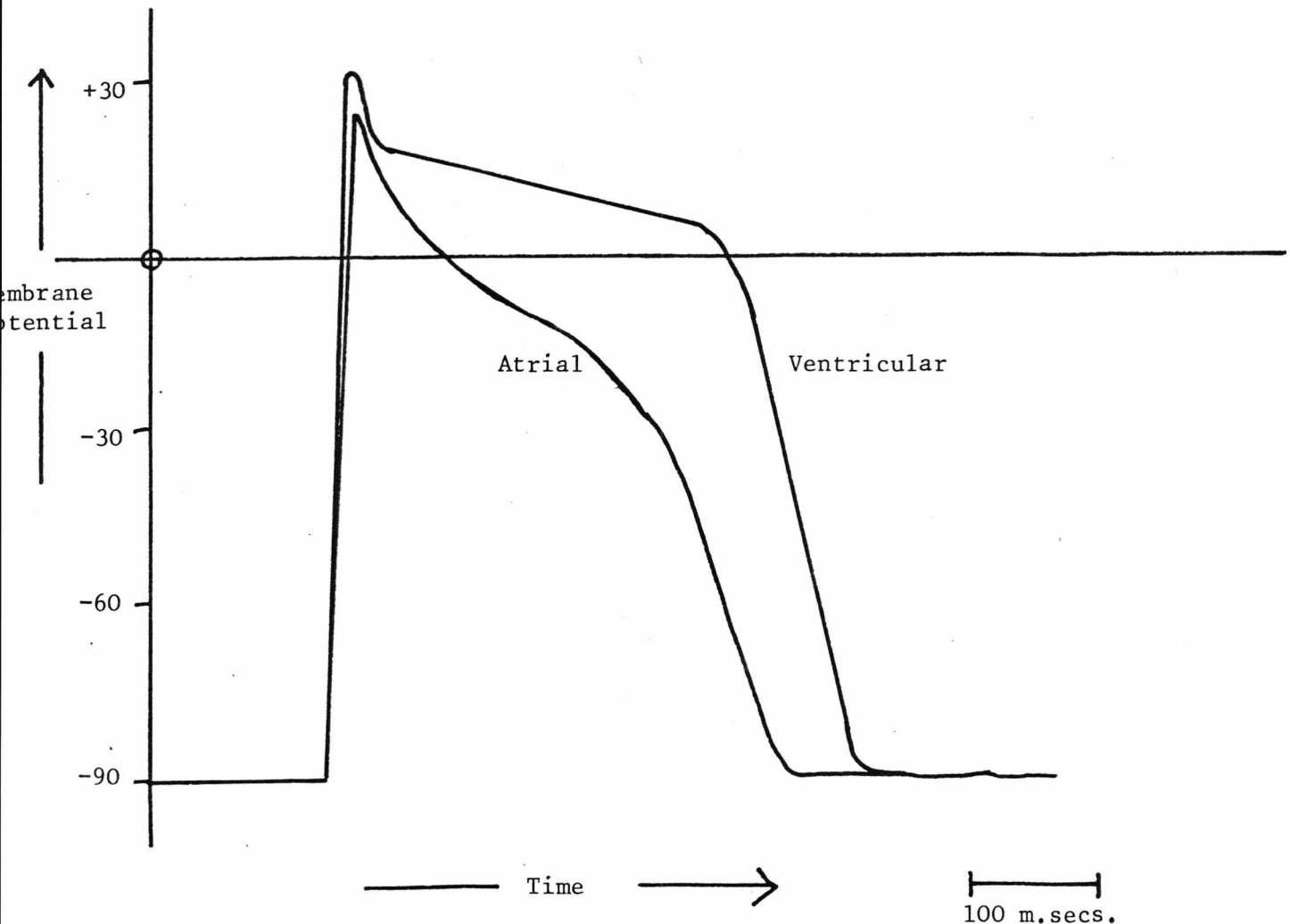


FIG. (9) Excitation Characteristics of Different Cardiac Cells

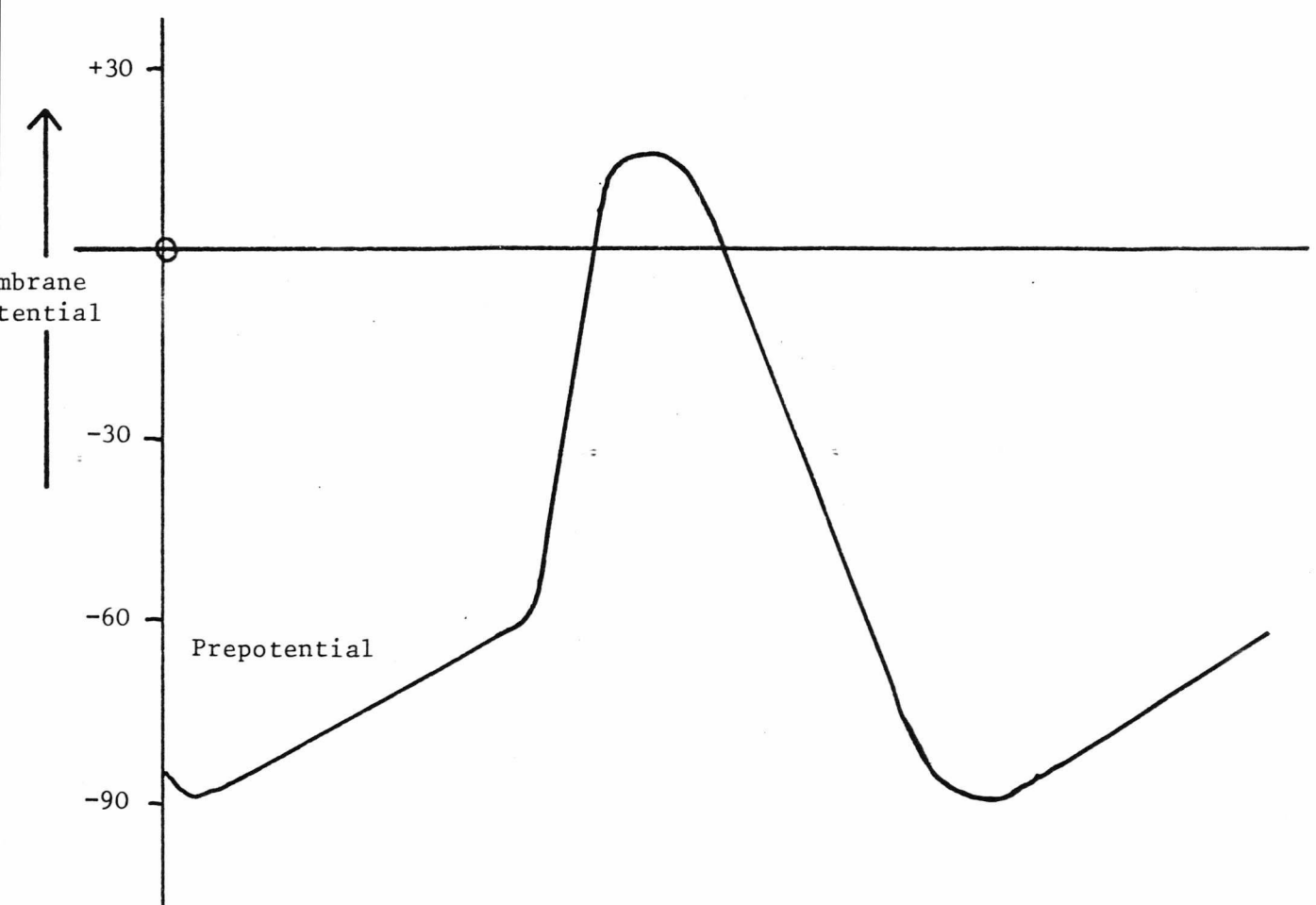


FIG. (10) Excitation Characteristic of Cardiac Pacemaker Cell

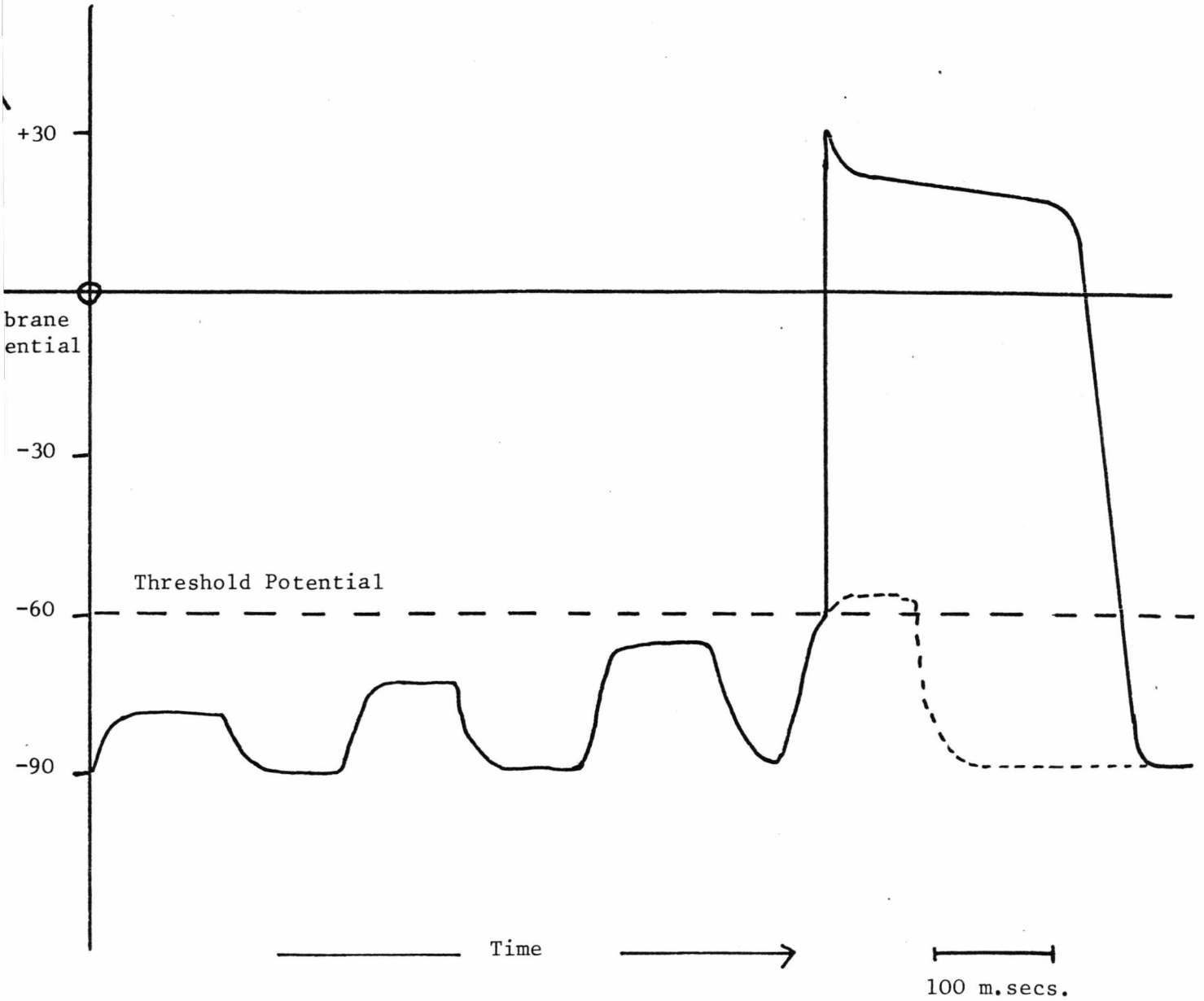


FIG. (11)

This diagram shows the response of a cardiac cell to three successive sub-threshold stimuli, and finally an action potential occurs for a supra threshold stimulus

	Ion Species	Extra ~~Intra~~-cellular Concentration μ Moles/cc	Intra. ~~Extra~~-Cellular Concentration μ Moles/cc	Nernst Potential $\varepsilon(mV) = \frac{61}{Z} \text{LOG}_{10} \frac{[Ion_i]}{[Ion_o]}$ Z = valency $[Ion_o]$ = extra-cellular $[Ion_i]$ = intra-cellular
Cations	Na^+	145	12	66
	K^+	4	155	-97
	H^+	$3{\cdot}8 \times 10^{-5}$	$1{\cdot}3 \times 10^{-5}$	-32
Anions	Cl^-	120	4	-90
	HCo_3^-	27	8	-32
	others	7	155	

FIG. (12)

A table of the concentrations of the ion species in the intra-cellular and interstitial fluids, and the resulting Nernst potential for each species

types present in the cellular environment, the higher conductance of potassium means that its potential will predominate.

When a suprathreshold stimulus occurs, then suddenly, the permeabilities change. That of sodium increases rapidly and so numbers of these ions[33],[34] enter the cell. This causes the membrane potential to change to +30 mV (near the Nernst potential for sodium ions). After a period of time, the potassium permeability increases to begin the repolarisation phase, and that of sodium decreases *(Fig. (13))*. This means that potassium ions leave the cell and no more sodium ones enter. This tends to restore the original charge balance and the membrane potential returns to the resting potential.

There is an active process called the "sodium pump"[34] which removes sodium ions from the cell during each resting phase. This has to be done since the normally low permeability of the membrane to sodium and the direction of the concentration gradient would not allow passive clearance of sodium from the cell. Although it would take many thousand (i.e. 200,000)[35] stimuli, eventually the cell would become poisoned and would not operate due to the intercellular sodium.

1.6 THE SPREAD OF DEPOLARISATION THROUGHOUT THE HEART

There are several sites in the myocardium where pacemaker cells exist[26] but those of the sino-atrial node (S.A. node) have the fastest rate of activity and so they determine the overall heart rate. They are under the

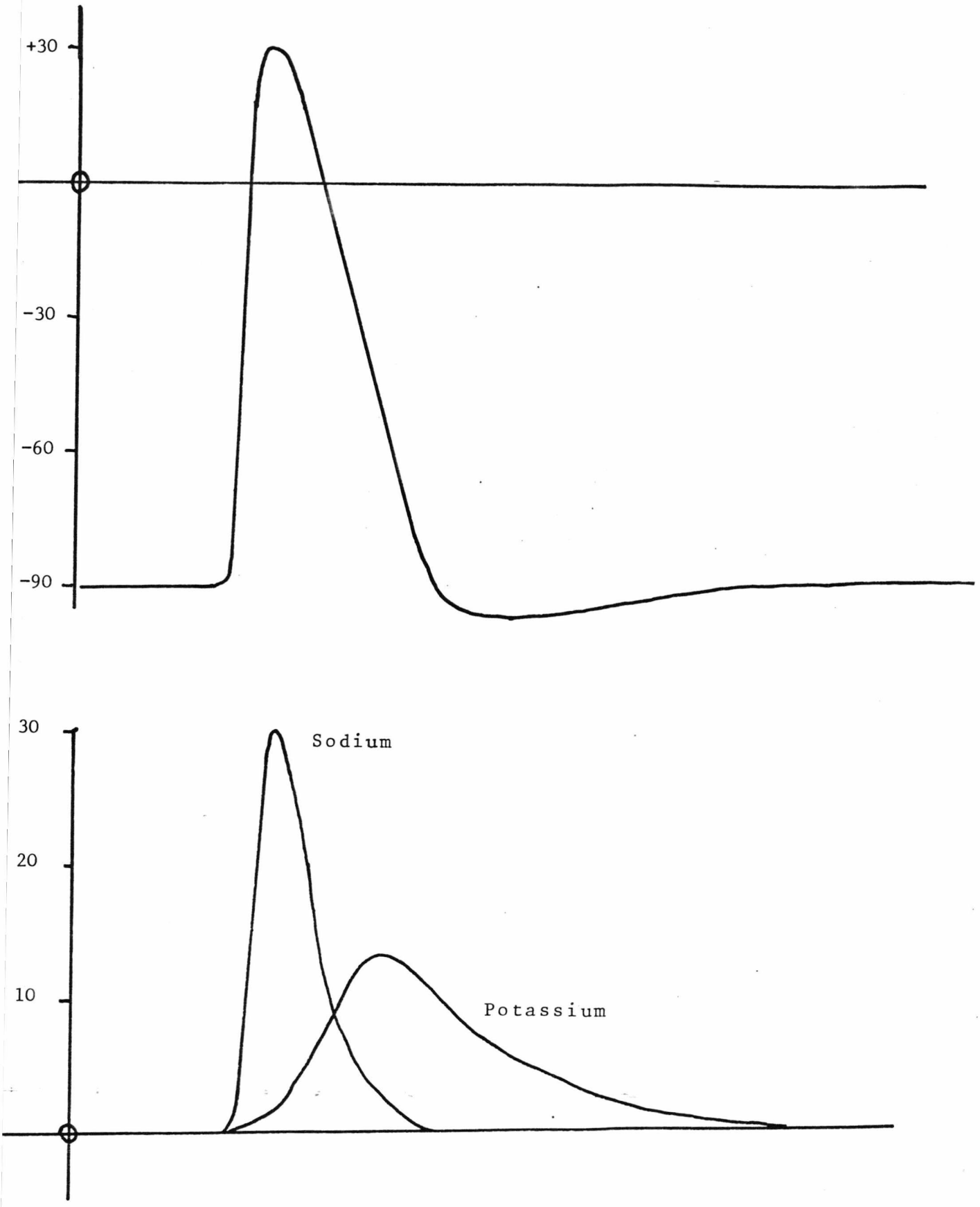


FIG. (13)

The Change in Sodium and Potassium Conductance During Depolarisation (in Squid Giant Axon)

influence of the Central Nervous System, and can have their prepotential rates varied, but they will function if the heart is severed from central nervous control and keep the heart beating at a rate of about 100 beats per minute. The automatic sites in the ventricular tissue have an inherent rate of 30 beats per minute.

When the decreasing prepotential reaches the threshold value of the pacemaker cells in the S.A. node, they depolarise. This activity is spread by cell-to-cell conduction throughout the atrial muscle. At first, the propagation velocity is very slow ≈1 m/sec. The whole of the atria is depolarised in about 60 m.secs. Since the fibrous septum does not allow conduction to spread directly into the ventricular muscle, the latter does not exhibit any activity until the depolarisation has passed through the A.V. node.

The junctional tissues marking the boundary between atrial muscle and A.V. node, have very low conduction velocities (0.05 m/sec.) and in the node itself values of ≈0.1 m/sec. are measured. It takes 90 m.secs. before the electrical signals reach the Bundle of His. When they do so, they quickly increase in velocity to about 1 m/sec. and pass into the left and right bundle branches at the same speed. When they reach the Purkinje Fibres, conduction velocities are in the order of 3 m/sec. The extensive Purkinje network and the cell-to-cell conduction ensure that the whole of the ventricular muscle depolarises very rapidly - in about 50 m.secs. *(Fig. (14))*.

Ventricular depolarisation starts in the endocardium of the intraventricular septum, and spreads to the epi-

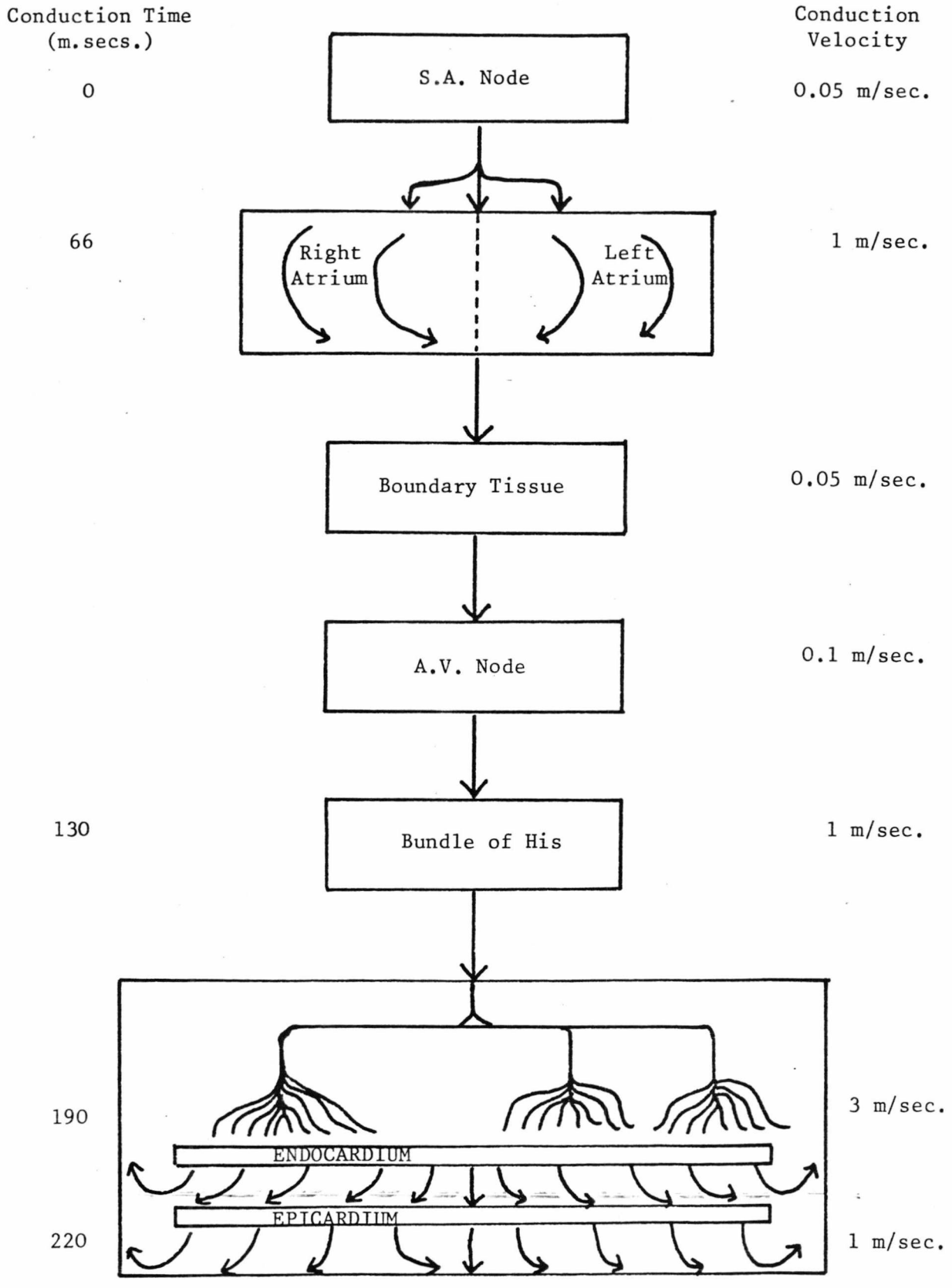


FIG. (14)

The Spread of Depolarisation and Conduction Velocities

cardium at the same time as moving towards the apex, and then round and up towards the atrio-ventricular septum. The last area to be depolarised is the posterobasal area of the septum.

Repolarisation is a slower process, taking about 200 m.secs., and occurs in the reverse direction to depolarisation. Atrial repolarisation occurs during the period of ventricular depolarisation[28],[36].

1.7 MECHANICAL EVENTS OF THE CARDIAC CYCLE

Muscular contraction starts about 50 m.secs. after membrane depolarisation, and so the course of the mechanical action of the heart follows that of the electrical activity. Cardiac muscular events can be divided into two main phases - diastole and systole, corresponding to the relaxation and contraction of the muscle respectively.

When the atrial muscle is depolarised it begins to contract and the period of atrial systole begins, and the pressure in the chambers increases. This forces more blood into the ventricles, and at the same time the contraction of the atrial walls squeezes the openings to the veins so that very little blood is pumped back down these passages. About 30% of ventricular volume is supplied during atrial systole[26] and this contribution is especially important at high heart rates when diastolic filling times are much shorter. Peak systolic pressures are 10 mm.Hg for the left atrium and 5 mm.Hg for the right atrium.

Towards the end of atrial systole, the ventricles are depolarised and ventricular systole begins. The pressure in the chambers begins to rise and when it is greater than atrial systolic pressures, then the mitral and tricuspid valves close. The period of ventricular isometric contraction then follows where the pressure rises but the volume of the ventricles stays constant since all valves are closed. The shape of the heart becomes more spherical as muscular tension increases, and when the ventricular pressures exceed the arterial pressures, then the aortic and pulmonary valves open, and the blood is rapidly ejected from the ventricles. Peak left ventricular systolic pressure is about 120 mm.Hg whilst the right ventricular systolic maximum is only 25 mm.Hg.

When the pressures in the ventricles have dropped below those in the arteries due to both the expulsion of blood, and the repolarisation of the muscle, the aortic and pulmonary valves close, and the diastolic period begins. When the ventricular muscles have fully relaxed, then the pressures are below those in the atria and so the atria-ventricular valves open, and ventricular filling begins. At first, there is a rapid filling phase and then the blood flow falls in the reduced filling phase. The latter continues until the commencement of the next period of atrial systole[37],[38],[39] *(Fig. (15))*.

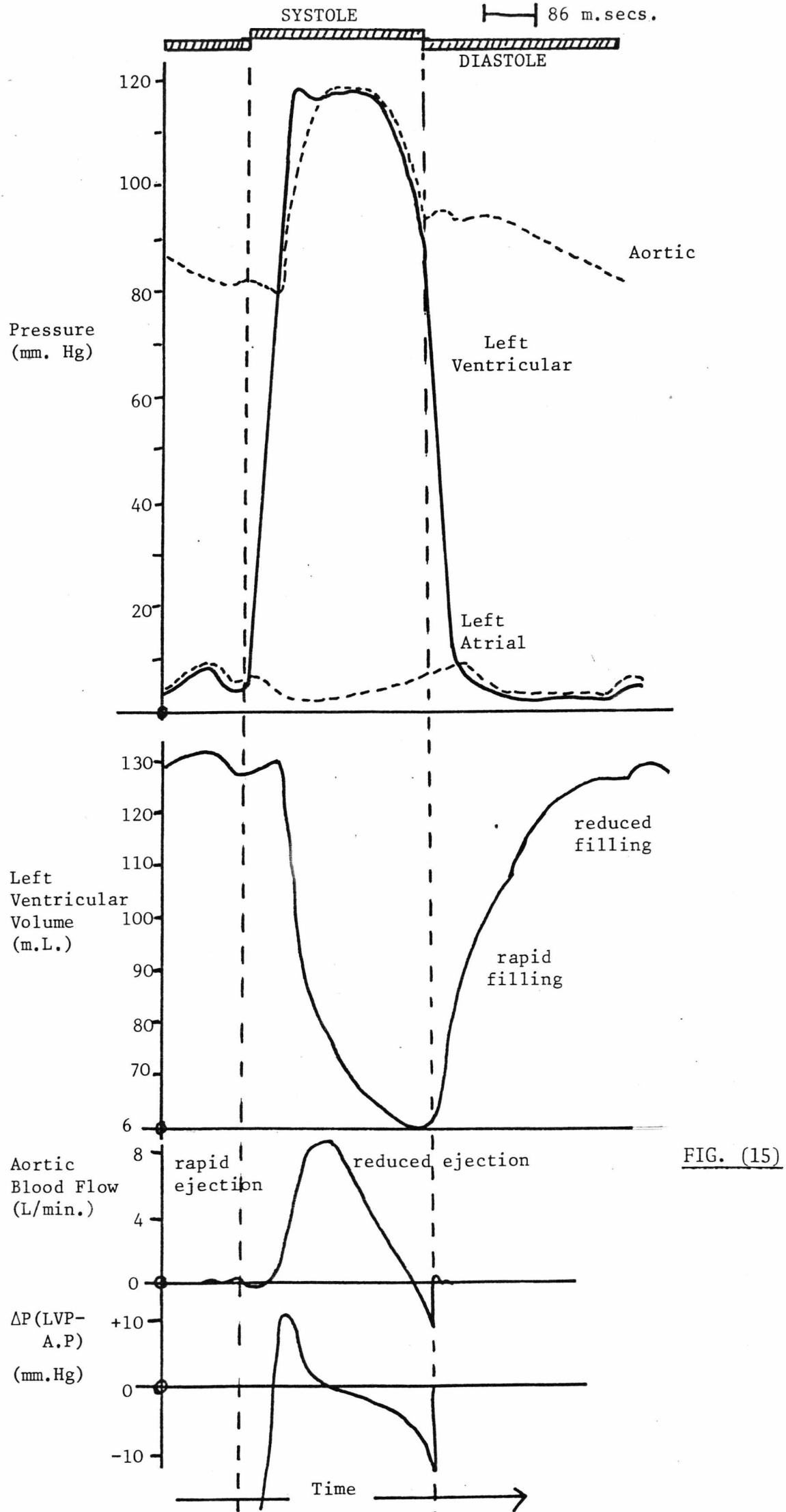

SYSTOLE
86 m.secs.
DIASTOLE
120
100
80
60
40
20
Pressure
(mm. Hg)
Aortic
Left
Ventricular
Left
Atrial
130
120
110
100
90
80
70
6
Left
Ventricular
Volume
(m.L.)
reduced
filling
rapid
filling
8
4
0
Aortic
Blood Flow
(L/min.)
rapid
ejection
reduced ejection
ΔP(LVP-
A.P)
(mm.Hg)
+10
0
-10
Time

FIG. (15)

REFERENCES

Most of the Physiology and Anatomy was studied from the following books:-

T.C. RUCH & H.D. PATTON "Physiology and BioPhysics" 1966. 19th Edn. Saunders.

E.E. SELKURT (Ed.) "Physiology - 10 Authors" 1966. 2nd Edn. Little, Brown & Co.

J.H. GREEN "An Introduction to Human Physiology" 1968. 2nd Edn. Oxford University Press.

D.J. AIDLEY "The Physiology of Excitable Cells" 1971. Cambridge University Press.

G.R. KELMAN "Applied Cardiovascular Physiology" 1971 Butterworths.

Hereafter, they will only be referred to by the authors names.

(1) S.G.B. STUBBS "Sixty centuries of health and physick" Sampson Low, Marstan & Co. Ltd.

(2) W. HARVEY "De motu cordis and sanguinis in animalibis" 1628.

(3) R. PATRICK "All colour book of Egyptian mythology" Plate 88. Octopus Books.

(4) Author's Personal Observations 1973, Egypt.

(5) W. LIBBY "History of medicine" p. 122-135. Constable 1923.

(6) C. SINGER "A short history of medicine" p. 38. Clarendon Press 1928.

(7) C. SINGER "A short history of medicine" p. 56.

(8) S.D. GERKING "Biological systems" 1969. Saunders & Co.

(9) E.E. SELKURT . Chapter 1.

(10) SCIENTIFIC AMERICAN "The living cell" 1965. Introduction Part I. Freeman & Co.

(11) S.D. GERKING "Biological systems" 1969. Saunders & Co.

(12) T.C. RUCH & H.D. PATTON, p. 543

(13) SCIENTIFIC AMERICAN "The living cell" 1965. Introduction Part II. Freeman & Co.

(14) SCIENTIFIC AMERICAN "The living cell" 1965. Introduction Part II. Freeman & Co.

(15) T.C. RUCH & H.D. PATTON, p. 545.

(16) T.C. RUCH & H.D. PATTON, Ch. 28.

(17) E.E. SELKURT

(18) T.C. RUCH & H.D. PATTON, p. 548.

(19) T.C. RUCH & H.D. PATTON, p. 549.

(20) J.H. GREEN, p. 19.

(21) E.E. SELKURT, p. 269.

(22) E.E. SELKURT, p. 270.

(23) G.R. KELMAN, p. 53-54.

(24) E.E. SELKURT, p. 273.

(25) T.C. RUCH & H.D. PATTON, p. 567-568.

(26) J.H. GREEN, p. 20.

(27) D.J. AIDLEY, Ch. 3.

(28) E.E. SELKURT, p. 289-293.

(29) T.C. RUCH & H.D. PATTON, p. 133-138.

(30) D.J. AIDLEY, p. 43.

(31) T.C. RUCH & H.D. PATTON, p. 4.

(32) D.J. AIDLEY, p. 19.

(33) D.J. AIDLEY, p. 71-87.

(34) T.C. RUCH & H.D. PATTON, p. 5.

(35) D.J. AIDLEY, p. 96.

(36) G.R. KELMAN, p. 32-33.

(37) T.C. RUCH & H.D. PATTON, p. 544/545.

(38) E.E. SELKURT, p. 278/281.

(39) J.H. GREEN, Ch. 3.

CHAPTER 2

ELECTROCARDIOGRAPHY

2.1 THE CLINICAL ELECTROCARDIOGRAM

The previous chapter concluded by showing the temporal relationships between the electrical activity of the cardiac muscular mass, and the pumping and relaxation of the ventricles. Under normal circumstances, the heart is sealed within the torso, and so when information on the heart's activities is required, an external method of detection is needed. One method is the recording of the electrocardiogram (E.C.G.). When electrodes are placed at suitable sites on the body surface, then voltages appear between them - the result of the ionic movements at the heart and the conducting properties of body tissue[1],[2],[3],[4]. When these voltages are amplified and displayed the result is the ECG - a typical example of which is seen in *Fig. (1)*.

The conventional electrocardiogram used in clinical practice today consists of twelve leads - each lead being the recording obtained between one pair of electrodes[5]. The series is derived from the work of Einthoven[6] published in 1913, who proposed that the heart be considered as a generator situated at the centre of an equilateral triangle formed by the two arms and the left leg, (Einthoven's triangle - *Fig.(2)*). As the wave of depolarisation of the muscle cells spreads throughout the heart, it can be said, at any one instant, to possess an overall direction of propagation and magnitude, depending upon how many cells are depolarising and their location. This resultant is called

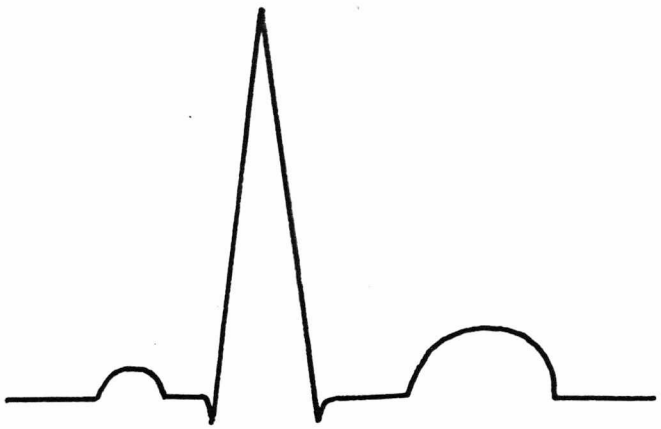


FIG. (1)

One complex from a typical electrocardiogram recorded between the right arm and left leg.

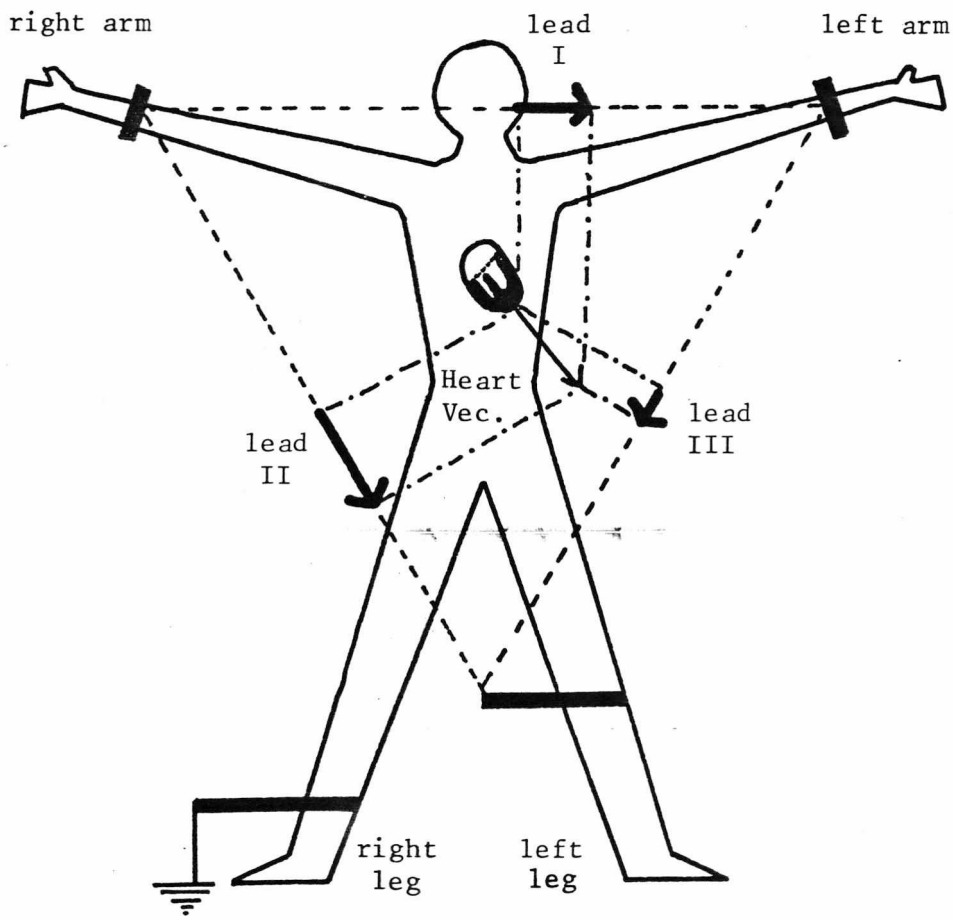


FIG. (2)

"Einthoven's triangle" - the first practical lead system for recording ECG's in the frontal plane.

by Einthoven the heart vector. He also assumes that the heart vector is equidistant from each of the limbs and is surrounded by a homogeneous conducting medium.

Work by Wilson (1934)[7],[8] and Goldberger (1942)[9] extended the basic concept to record unipolar potentials from the chest and limbs. A typical full 12 lead electrocardiogram from a healthy 28 year old male is shown in *Fig. (3)*.

The first three leads called lead I, lead II and lead III are obtained from between the right arm and left arm, the right arm and left leg and the left arm and left leg respectively. From *Fig. (2)*:

$I = V_R - V_L$ V_R = the potential of the right arm
$II = V_R - V_F$ V_L = the potential of the left arm
$III = V_L - V_F$ V_F = the potential of the left leg

The unipolar set of leads uses one exploring electrode placed over different sites on the chest, in conjunction with an indifferent electrode at theoretically zero potential. This indifferent electrode was proposed by Wilson et al[8] and uses the basic concept of the Einthoven triangle, and although not strictly true - it does serve a useful purpose. It is obtained by summing the potentials from the limbs with a network of low value resistors (5kΩ → 20kΩ) as shown in *Fig. (4)* (later workers have proposed that these can be too small with certain types of electrodes and suggest much higher values[10],[11].

In *Fig. (5)*, let V_R, V_L and V_F be the potentials at the respective limbs produced by the rotating heart vector V_H at

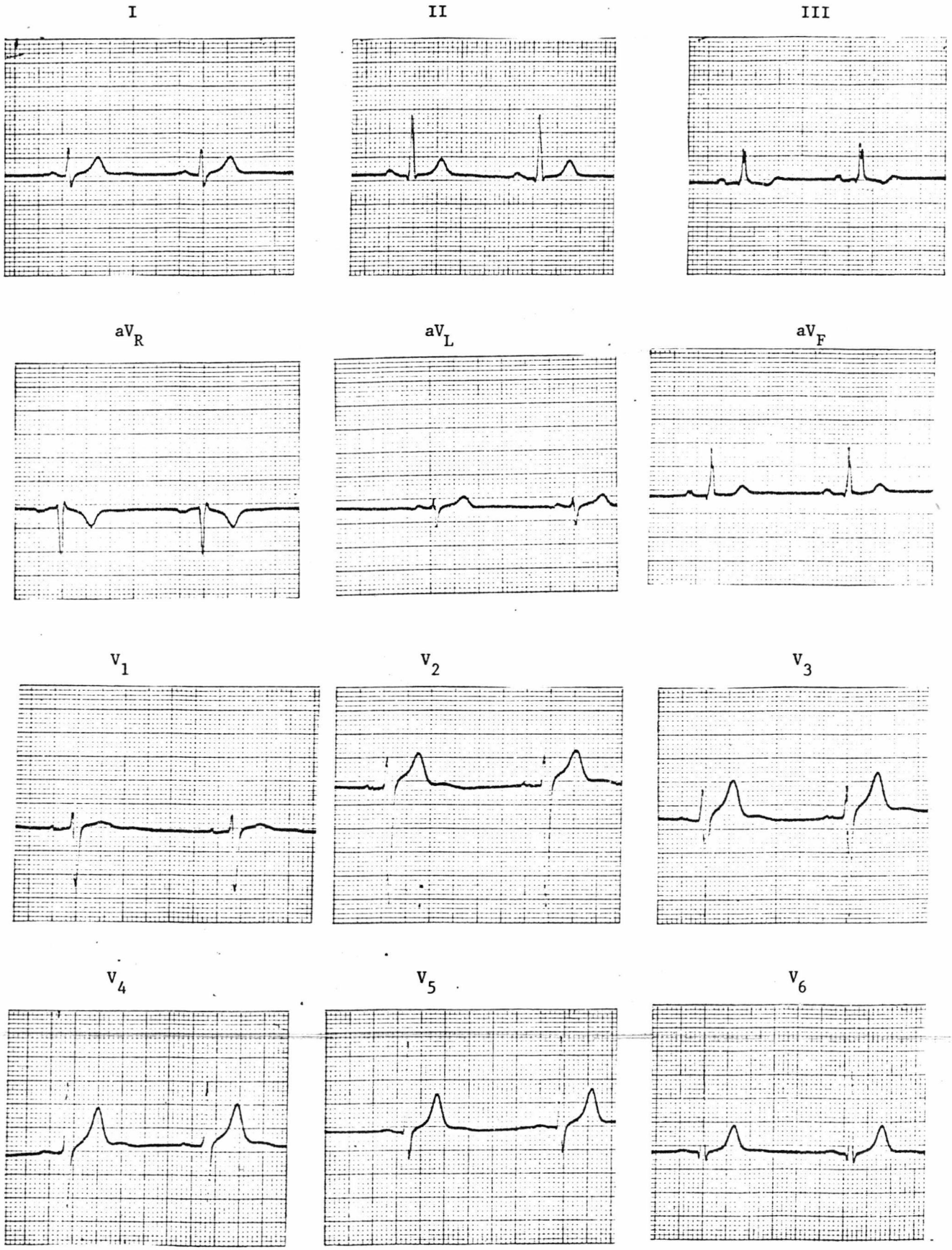


FIG. (3) : A full 12 lead ECG from a normal patient.

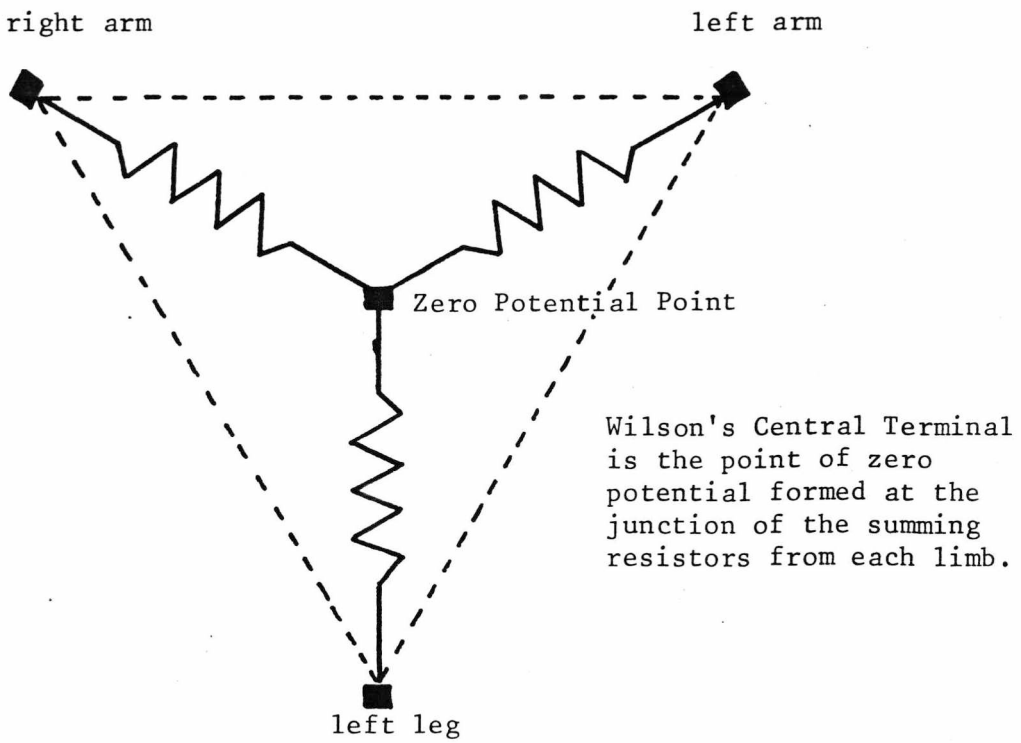

right arm
left arm
Zero Potential Point
Wilson's Central Terminal is the point of zero potential formed at the junction of the summing resistors from each limb.
left leg

FIG. (4)

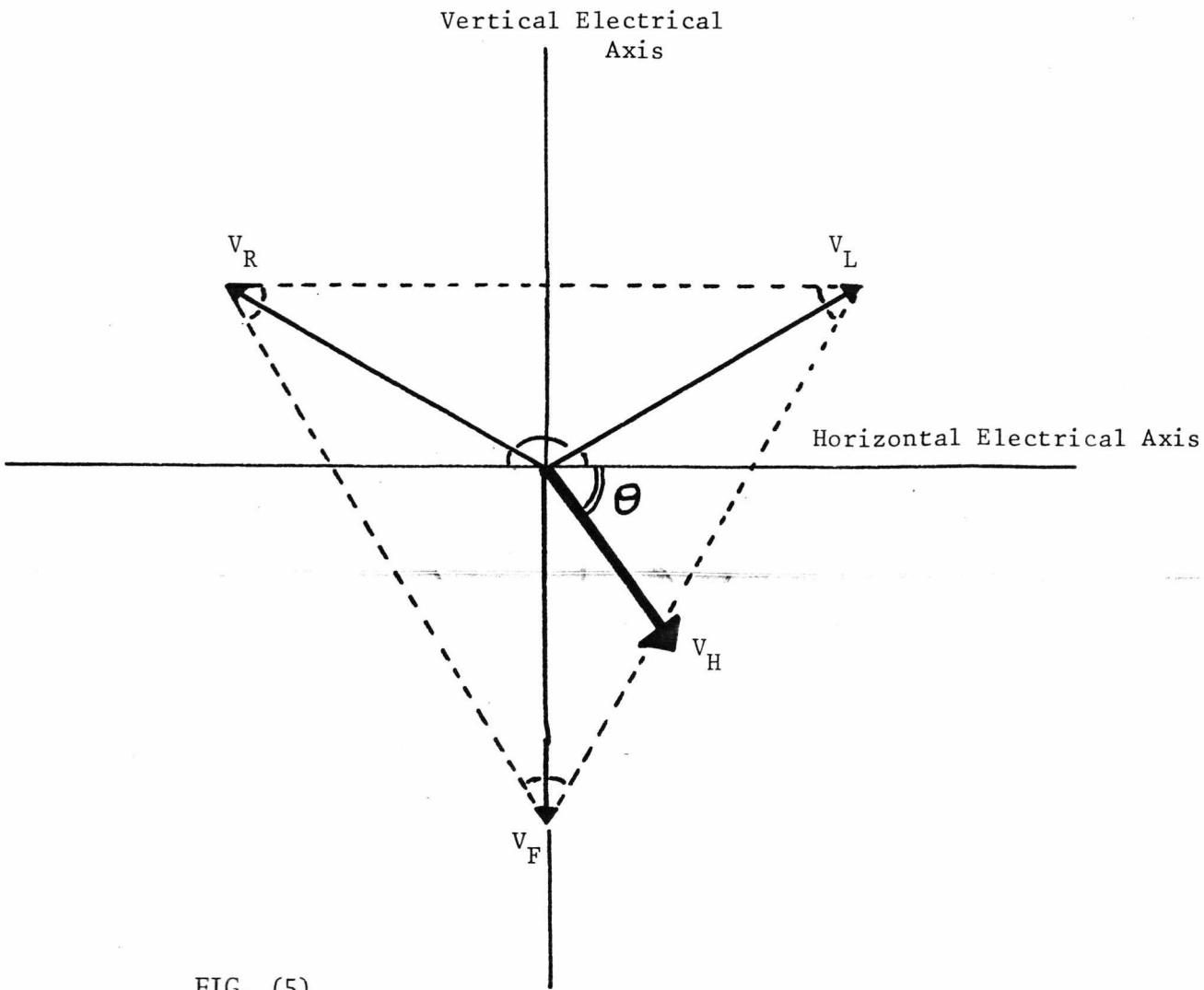

Vertical Electrical Axis
V_R
V_L
Horizontal Electrical Axis
θ
V_H
V_F

FIG. (5)

any instant in the cardiac cycle. Then, if θ is the angle between V_H and the horizontal electrical axis:-

$$V_R = V_H \cos(150^o+\theta)$$
$$V_L = V_H \cos(30^o+\theta)$$
$$V_F = V_H \cos(90^o-\theta)$$

The summation of these three gives

$$V_R + V_L + V_F = V_H(\cos(150^o+\theta)+\cos(30^o+\theta)+\cos(90^o-\theta)$$
$$= V_H(0)$$
$$= 0$$

Thus, assuming the Einthoven triangle to be correct, a point of zero potential can be found as follows:-

From *Fig. (4)*:

$$V_P = V_R \times \frac{R/2}{3R/2} + V_L \times \frac{R/2}{3R/2} + V_F \times \frac{R/2}{3R/2}$$

$$= (V_R + V_L + V_F) \times \frac{1}{3}$$

$$\underline{\underline{= 0}} \qquad \text{since } V_R + V_L + V_F = 0.$$

This is what is known as Wilson's Central Terminal.

The six unipolar leads from the chest are known as V_1,V_2, V_3,V_4,V_5 and V_6 and are shown in *Fig. (6(i))*.

Goldberger[8] proposed another lead system to record unipolar leads from the limbs. This time his indifferent electrode consisted of only a pair of limb leads summed by resistors, instead of all three. *Fig. (7)* shows the arrangement for recording a unipolar lead from the right arm.

A section through the Throrax illustrating the precordial lead positions with respect to the heart.

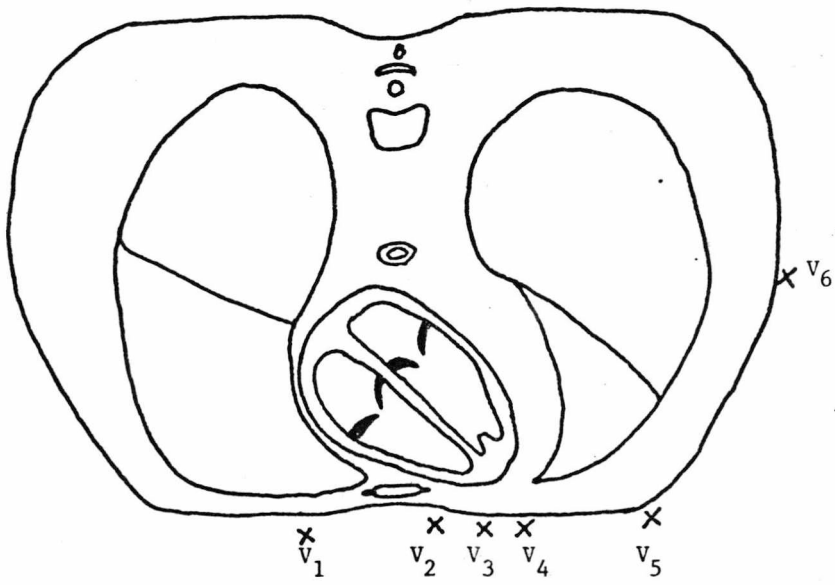


FIG. (6)

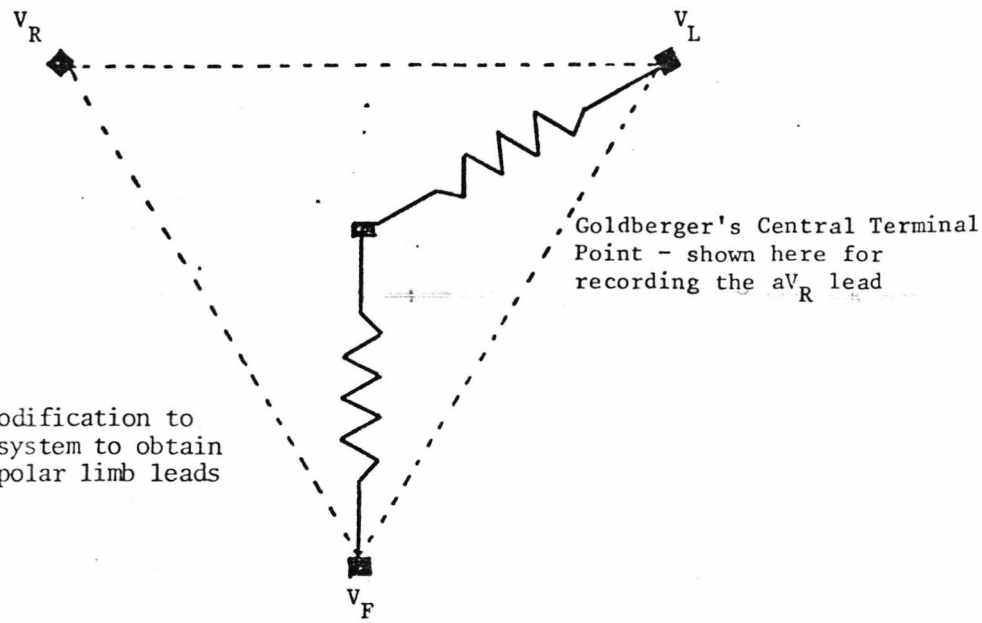


FIG. (7)

Goldberger's modification to Wilson's lead system to obtain augmented, unipolar limb leads aV_R, aV_L, aV_F.

$$\text{Now} \quad V_P' = V_L \times \frac{R}{2R} + V_F \times \frac{R}{2R}$$

$$= \frac{V_L + V_F}{2}$$

and since $V_R + V_L + V_F = 0$

$$= \frac{-V_R}{2} \quad .$$

$\therefore$ the unipolar lead $= V_R - V_P'$

$$= V_R - \left(\frac{-V_R}{2}\right)$$

$$= \frac{3V_R}{2}$$

These are known as augmented limb leads and are designated aV_R, aV_L and aV_F for the right arm, the left arm and the left leg respectively.

2.2 THE RELATION BETWEEN ECG AND THE CARDIAC CYCLE

A typical lead II ECG is shown in *Fig. (8)* with all its component parts labelled[(1),(2),(3),(4)].

The P wave occurs during atrial depolarisation as the latter proceeds from the S-A node throughout the atria, and lasts about 100 m.secs. The P-R interval is an isoelectric period when the wave is passing from the atria through the A-V node to the ventricles and lasts about 100 m.secs. The main Q.R.S. complex is the surface potential observed due to the depolarisation of the ventricles and is normally 60 to 80 m.secs. long. After this, is the S-T segment, again isoelectric, and this period is the last part of systole. The T wave is that of the repolarisation process of the ventricles

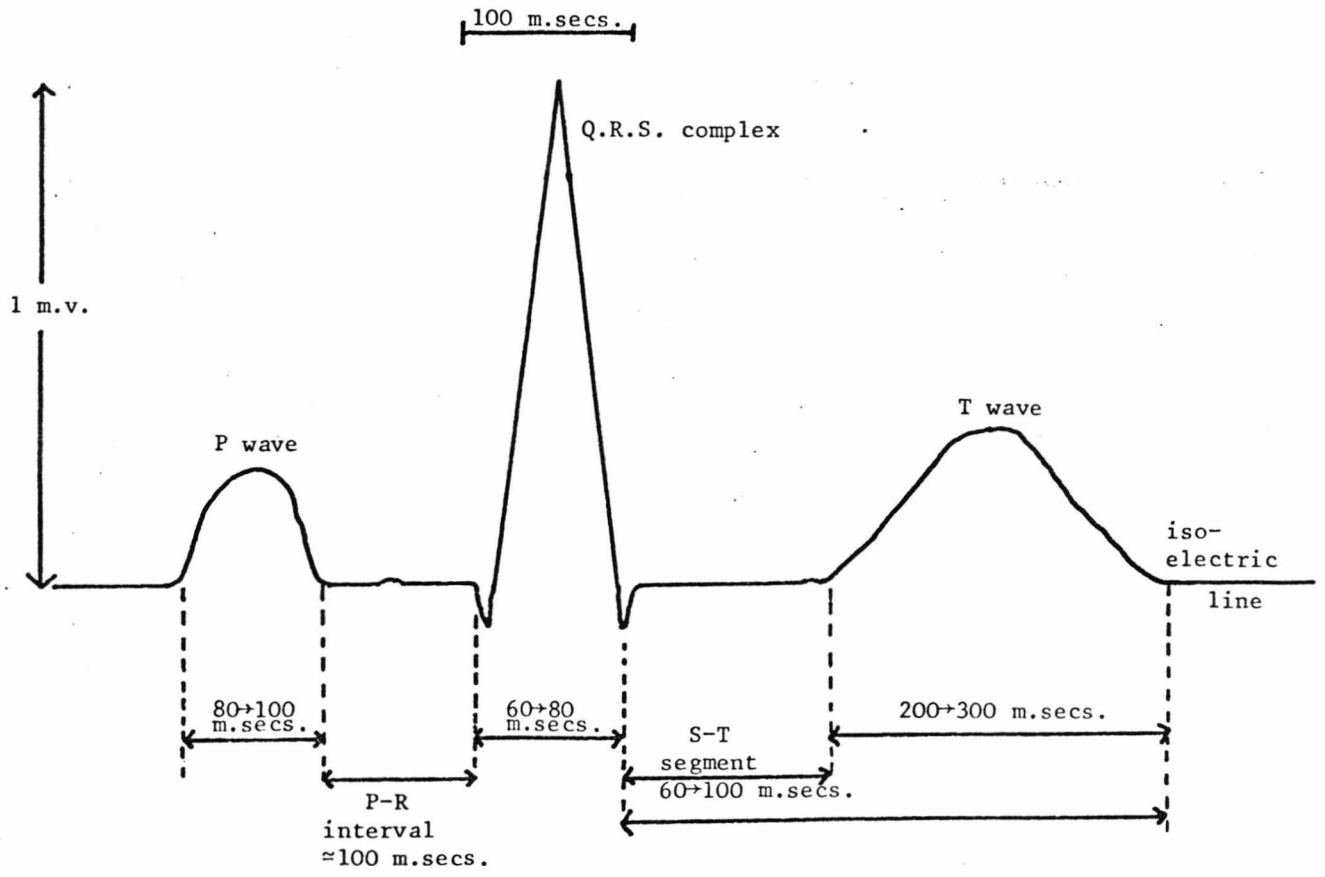


FIG. (8)

A Typical Lead II ECG Complex

and lasts much longer than the Q.R.S. complex, since the process occurs much slower. The beginning of the T wave marks the beginning of diastole. It's polarity is such that although the cell potentials are reversed so is the direction of propagation and these facts combine to give a surface T wave of the observed polarity.

Atrial repolarisation occurs during ventricular depolarisation, and so cannot be observed on the ECG as it is lost in the Q.R.S. complex (work is, however, in progress at the Kent & Canterbury Hospital[12] to see if P wave repolarisation can be observed in patients with second degree heart block).

2.3 THE DIPOLE THEORY IN ELECTROCARDIOGRAPHS

2.3a Introduction

It was shown in section 2.1 how the present system of recording ECG's depends upon the assumption that in generating body surface potentials the heart can be represented by a dipole. Although controversy still exists over the validity of this assumption, and just how 'dipolar' the heart really is, the method has proved to be a good enough approximation to give valid, repeatable, clinical results and to have gained the widespread acceptance of clinicians.

In this third section of Chapter 2 we shall further examine the dipolar properties of the heart - how such an organ with a finite size and shape can be reduced to a dipole source and how the resulting body surface potentials depend upon the properties of the body itself and the position of the recording electrodes.

2.3b The Derivation of the Equivalent Dipole

Consider a single muscle cell surrounded by an infinite, homogeneous, isotropic conducting medium. Due to the distribution of sodium and potassium ions across the resting cell membrane, it can be represented as in *Fig. (9)* with the inside of the cell negative with respect to the outside[13]. At rest, we can invoke Gauss's Law to find the potentials in the medium due to the cell.

Gauss's Law states that the total flux from a closed surface is equal to the charge enclosed by that surface[4]

$$\oint \underline{E}.\underline{ds} = \rho$$

In this case since the cell is at equilibrium it can be considered as a closed surface with no net charge density, and therefore no fields or potentials can be measured in the surrounding medium.

~~Since~~

$\rho = 0$

$\therefore E = 0$

Apart from the charges bound to the membrane, there can be no other free charge in the conducting medium to create potentials. Should any free charge be introduced, the fields that it would tend to create would then act upon the charges themselves, and so no equilibrium could be realised. It can be shown[14] by using the divergence theorem and Gauss's Law that if there is a region of charge density ρ_o within a conductor of conductivity σ and ε (permittivity), then that charge decays exponentially as

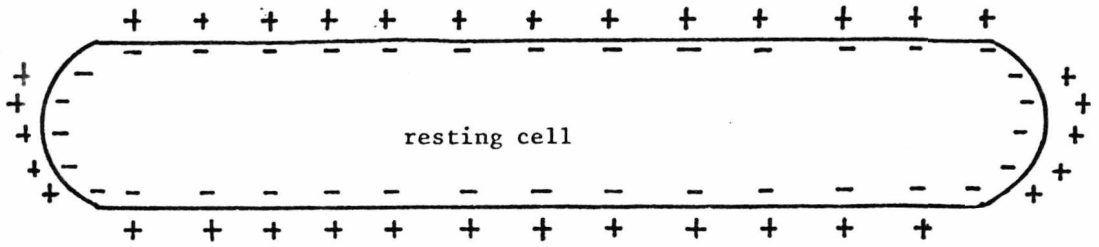


FIG. (9) : Electrical representation of ionic charge distribution across a muscle cell membrane of a resting cell.

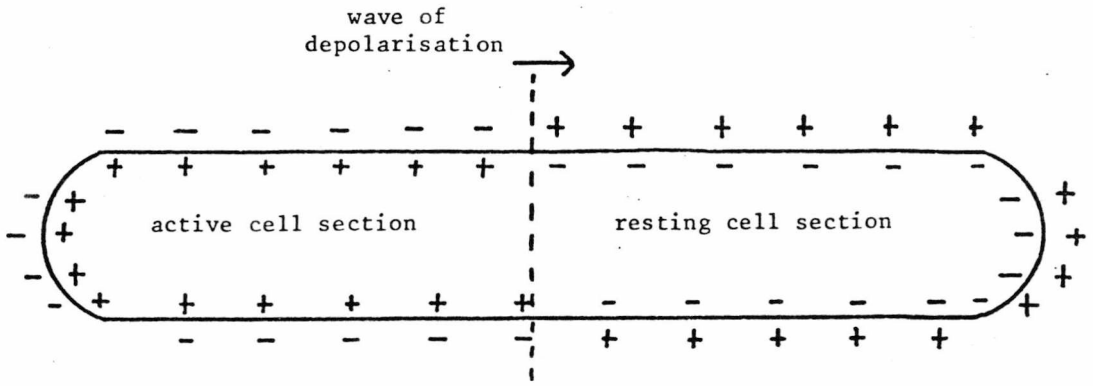


FIG. (10) : The charge movement that occurs, when the cell becomes active, and the depolarisation wave passes down the axon, in the direction shown .

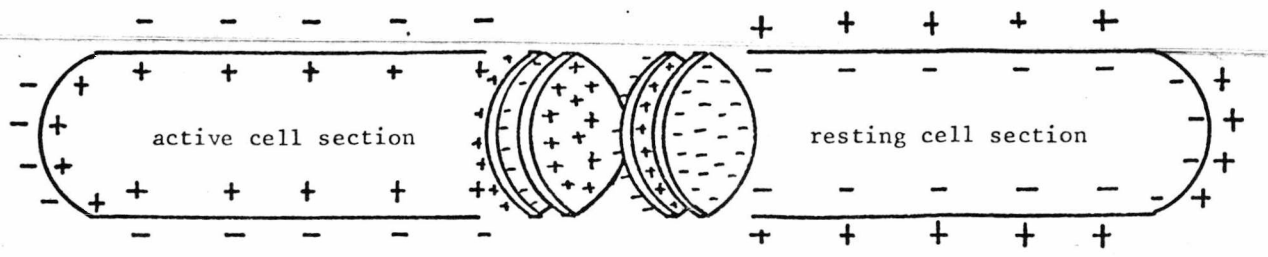


FIG. (11) : The introduction of charged discs, with a neutral net effect for the derivation of an equivalent dipole for the muscle cell.

$$\rho(t) = \rho_o \, e^{\frac{-\sigma t}{\varepsilon}}$$

σ = conductivity
ε = permittivity

Thus for tissue such as lung with a resistivity of 1000Ωcm., and a relative dielectric constant $4.5\times10^{+5}$,(15) this gives a time constant of decay of ~~$\simeq 2.5\times10^{-8}$~~ 4.0×10^{-5} secs., which is a very small time, and thus no free static charge can exist in lung or any other body tissue.

When the nerve is excited, the ions move across the membrane as equilibrium is disturbed, during the time the wave of depolarisation passes down the axon. At any instant it can be represented as in *Fig. (10)*. Thus, it is now no longer a closed uniform surface and fields and potentials are set up in the medium(13) during the passage of the wave.

If we imagine the axon to be split in two at the interface of resting and excited parts of the muscle *(Fig. (11))*, then by introducing charged discs into the interface it is possible to reduce the whole nerve to a dipole. Let the introduced discs be in two pairs (a,b) and (c,d), with each pair consisting of discs, oppositely charged and separated by a small distance such that each set is neutral overall, and let them be placed as in *Fig. (11)*. Disc 'a' can be combined with the excited part of the axon, and disc 'd' with the resting part, and then there is the situation of two closed surfaces again (if the quantities of the charge on 'a' and 'd' are suitably chosen), with discs 'b' and 'c' left. The latter two discs are the only parts of the system which cause potentials in the surrounding medium since the closed surfaces can contribute nothing at all *(Fig. (12))*. These two discs can further be combined into a single charged disc *(Fig. (13))* and thus, electrically, we can reduce an excited

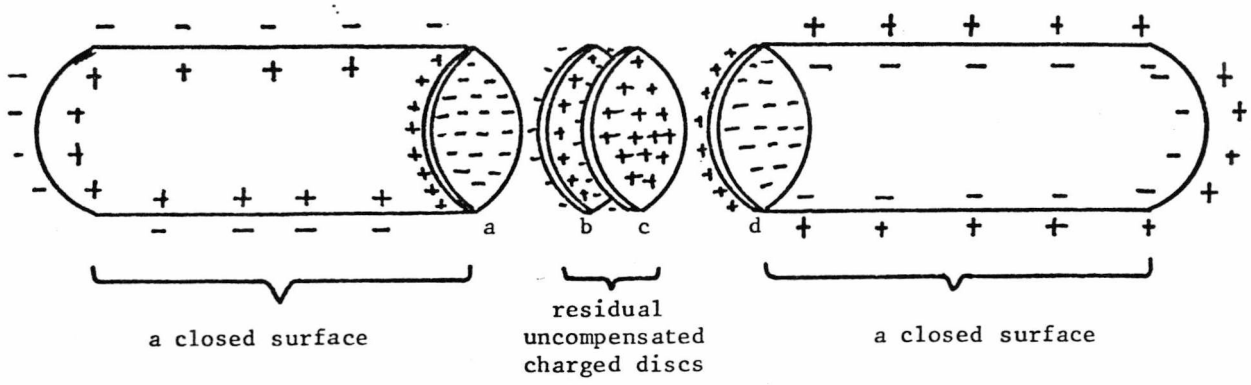


FIG. (12) : When discs a and d are connected to the active and resting cell section respectively, to form closed surfaces, then discs b and c are left to generate electric fields in the medium.

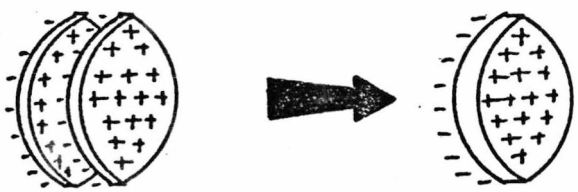


FIGURE (13) : Discs b and c can be combined into one resultant dipole surface moving in the direction of depolarisation.

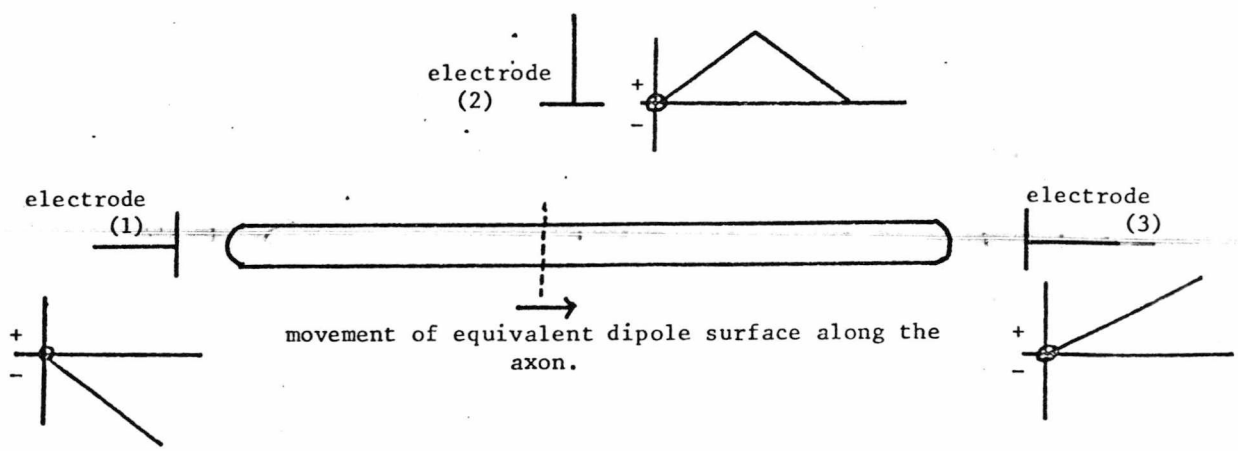


FIGURE (14) : The potentials recorded by unipolar electrodes at different sites, during the depolarisation of a muscle cell.

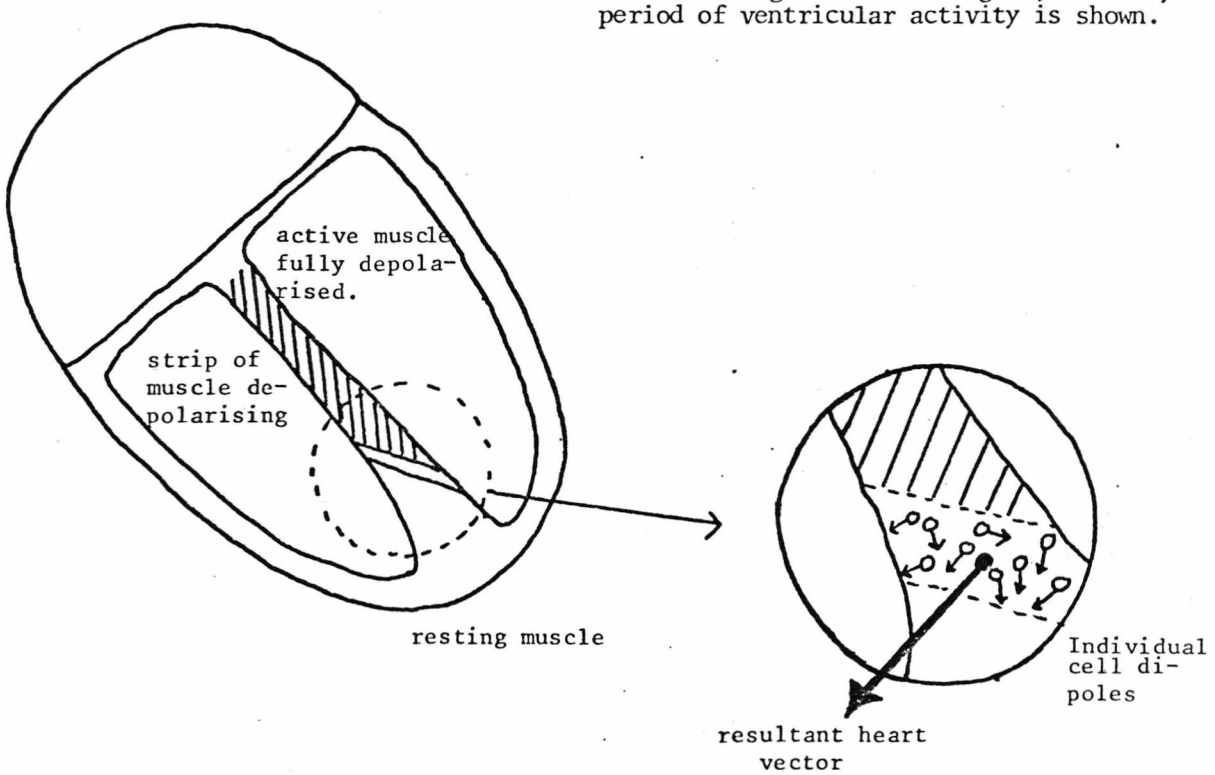
axon to a dipole, moving along the axis with the depolarisation process. Electrodes placed in the medium at the sites 1, 2 and 3 as shown in *Fig. (14)*, would record the potentials shown during the course of the action potential.

This concept can be extended to the whole heart[16]. As the wave of depolarisation passes throughout the musculature then at any one time, the wavefront consists of many muscle cells activating. Applying the previous arguments, the equivalent dipoles of each active cell will sum together - the resultant of which gives an overall dipole strength and duration for the wave at that time *(Fig. (15))*.

2.3c The Lead Vector

Using the concept of a dipole equivalent generator for the heart Burger and Van Milaan developed a generalised lead vector theory[17], and it can be shown that Einthoven's triangle is a special case of this[16]. Since a dipole has a magnitude and a direction it can be considered as a vector. Let this vector be $\overline{H}$, and as such it can be resolved into three orthogonal directions, corresponding to a set of x, y and z axes with their origin that of the dipole location. Thus we have H_x, H_y and H_z components of $\overline{H}$. If the dipole vector $\overline{H}$ is surrounded by a volume of arbitrary shape *(Fig. (16))*, which is linear and conducting, then it is possible to measure potentials P_1 and P_2 on the boundary of the surface. The potential difference between the two (P_1-P_2) can also be resolved into three components of a vector V_x, V_y and V_z with the same axial directions as the heart vector, and since the region is linear then we can say

FIG. (15) : The heart vector can be considered as the resultant of the dipoles of the individual cells that are in the process of activating. In this diagram, an early period of ventricular activity is shown.



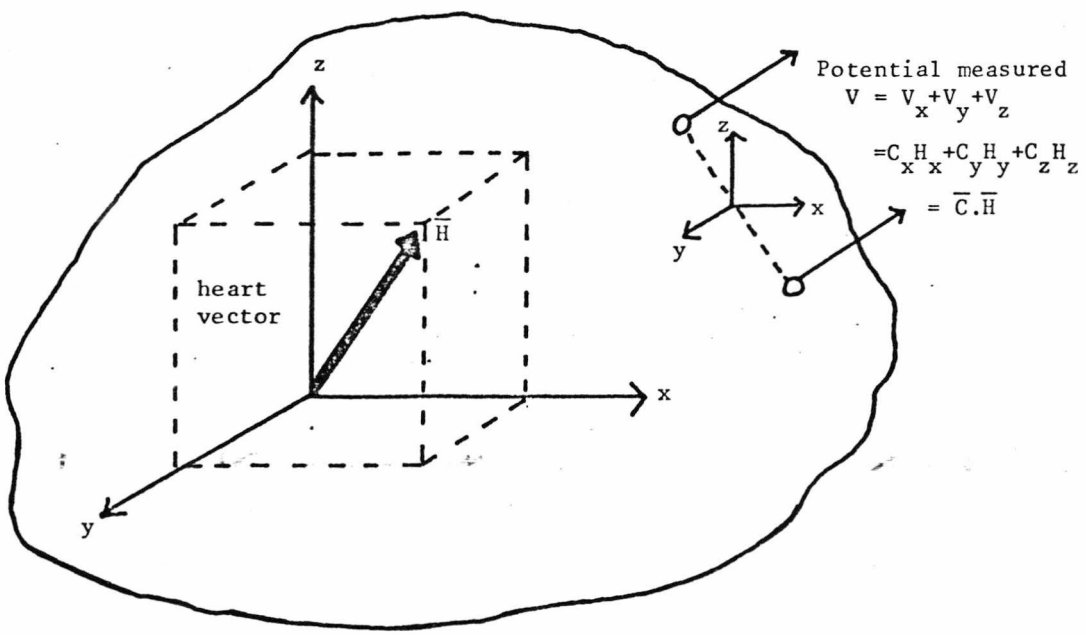


FIG. (16) : The heart vector is surrounded by a homogeneous conducting medium. The potential V measured between two points on the boundary is equal to $\overline{C}.\overline{H}$ where C is the lead vector.

$$V_x = C_x.H_x \qquad \text{where } C_x,$$
$$V_y = C_y.H_y \qquad C_y, \quad \text{are constants}$$
$$V_z = C_z.H_z \qquad C_z,$$

Due to the superposition theorem[18] we can combine all of these into a single sum:

$$V = C_x.H_x + C_y.H_y + C_z.H_z = V_x + V_y + V_z$$

These can be written as a vector equation

$$V = \begin{bmatrix} C_x & C_y & C_z \end{bmatrix} \begin{bmatrix} H_x \\ H_y \\ H_z \end{bmatrix}$$

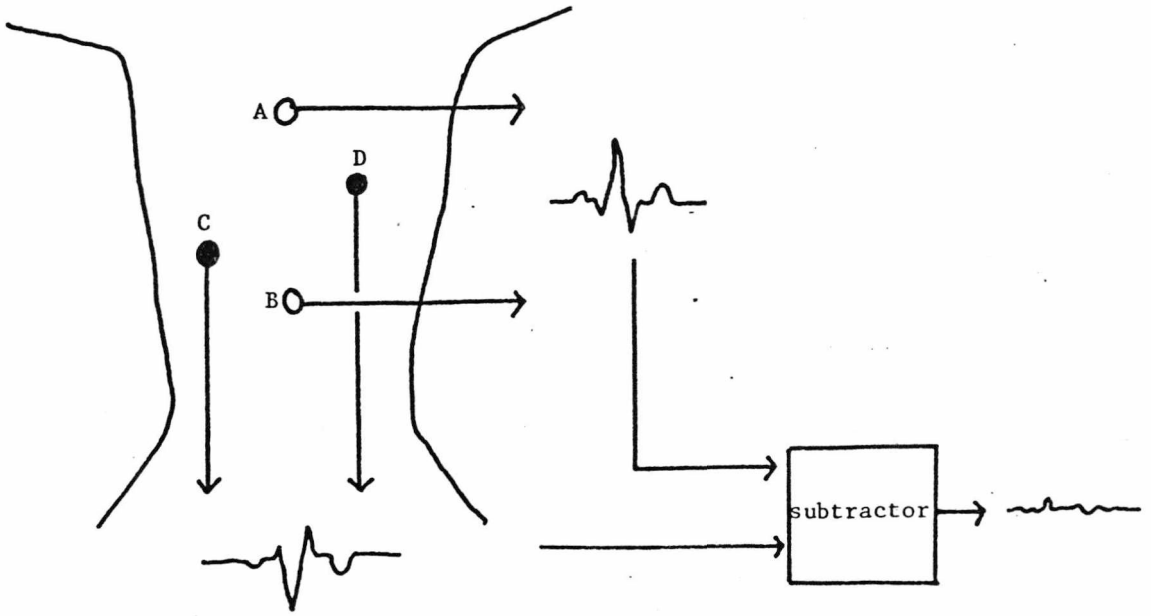
or $V = \overline{C}.\overline{H}$ where $\overline{H}$ is the heart vector

and $\overline{C}$ is a vector called the lead vector or image vector

and is a factor involving the properties of the medium, and the relative directions of the two electrodes to the heart vector. Einthoven's original vector triangle was a special case, where the lead vectors were leads I, II and III, and they were all the same size with the same angular separation - thus forming the equilateral triangle.

To further support the idea of the dipolar heart vector Frank and others[19],[20],[21] performed a series of experiments. The first of these were a set of 'cancellation' tests - as first proposed by Schmitt et al[16],[22]. If the heart were truly dipolar in action, then a potential V_1 as detected by any lead vector $\overline{C}_1$ should have an exact image, $-V_1$ with a different lead vector $\overline{C}_2$ *(Fig. (17))*. Thus it

FIG. (17) : The cancellation test for dipoles. If the heart can be considered as a dipolar generator, then the potential between CD should be a mirror image of the potential between AB. The output from the subtractor indicates the degree of non-dipolarity.



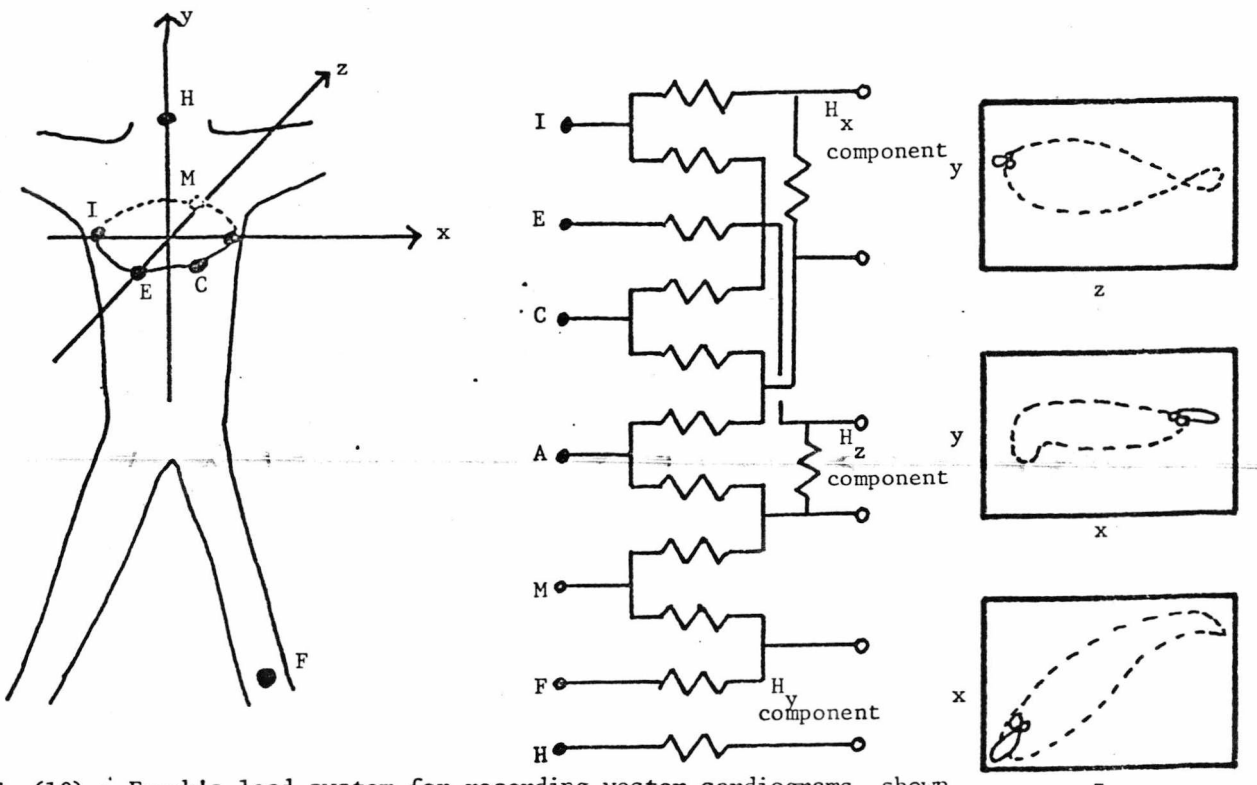


FIG. (18) : Frank's lead system for recording vector cardiograms, shown with the resistance network, and typical examples of VCG's in each plane.

should be possible to find for every ECG detected on the body surface an equal and opposite 'image' ECG somewhere else. If the heart had non-dipolar components then the degree of non-cancellation would indicate the degree of non-dipolarity[19]. The result of extensive tests by Frank was that the single fixed-location dipole hypothesis has an accuracy of 85% to 95% for the Q.R.S. complex - indicating that in the subjects tested the heart was largely dipolar in its electrical activity.

Frank then went further and performed tests by comparing human subjects with a torso model using a dipole as an equivalent cardiac source. Burger[17] had shown the location of the dipole to be really critical, and small variations in its site caused large changes in the ECG patterns on the surface, and so Frank had, first of all, to find the effective dipole position of the human subject's heart, and then place a dipole source in the corresponding location in the torso model, ~~Frank~~[19],[21],[23]. He obtained numerous sets of cancellation leads from his human subject at the mid-ventricular level of the chest, and then adjusted the dipole location in the model, until the same (or almost so) cancellation leads were obtained from the torso model surface. He then compared the ECG's produced by the human and the dipole at positions all over the torsos and especially at distant locations. He claimed successful determinations had been obtained in over 40 patients[19] with quantitative agreement to within ±15% in the comparable size of the Q.R.S. complex produced.

The results of both the cancellation tests and the comparison tests confirmed Frank in his opinion that a suitable equivalent generator was a dipole immersed in a homogeneous medium.

The conclusions that he drew from this were far reaching because if the action is truly dipolar, then only three leads need be taken - one along each of the x, y and z axes of the system. Any further leads give no extra information at all since the only information to be obtained is the H_x, H_y and H_z components of the heart vector $\overline{H}$. Thus the standard 12 lead electrocardiogram becomes redundant, and Frank proposed replacing it by the three lead vector cardiogram *(Fig. (18))* e.g., "This means that in principle any three independent potential differences will give all available information and that additional leads will give only redundant information"[19].

2.3d The Arguments Against a Single, Fixed Location Dipole Theory

Support for Frank's proposals was by no means universal and there were many criticisms - notably those from Katz[24] who pointed out that the heart was in fact a large organ - not a point, that it varied in size and position from person to person and even during each cardiac cycle. He also pointed out that the body is not a linear, isotropic homogeneous infinite conductor being in fact, very much the opposite, and so to draw conclusions rejecting the 12 lead electrocardiogram was not correct. Studies by Helm & Chou[25] and others[26],[27],[28] have shown that the cancellation tests are by no means a definite proof of dipolarity, and in fact, that good cancellation can be obtained between waveforms no matter what the spatial distribution of the electrodes and source potentials[16]. It was also found that for people with cardiac disorders, cancellation became more difficult to obtain, and did not apply to P and T waves.

Another interesting point was that the subject used by Frank was discovered to be above average in his dipolarity[29], and the results could not be duplicated with as much success on other patients.

One major point of disagreement between Frank and others concerned proximity potentials. The precordial leads are assumed to be mainly relevant to the tissue beneath them, *(Fig. (19))*, but the dipole hypothesis rejects this quite definitely.

In *Fig. (19)* we have a heart vector $\overline{H}$ which is shown in a suitable medium and we can find the potentials between electrodes at points a, b, c, etc. by using the lead vector theorem

$$\begin{aligned} V_a &= \overline{C}_a.\overline{H} \\ V_b &= \overline{C}_b.\overline{H} \\ V_c &= \overline{C}_c.\overline{H} \\ V_d &= \overline{C}_d.\overline{H} \end{aligned} \qquad \overline{H} \text{ is common to all,}$$

Thus, by taking many leads all we obtain is information concerning the different lead vectors $\overline{C}_a, \overline{C}_b, \overline{C}_c, \overline{C}_d$, etc. Frank sums this up by saying "Admittedly it would be extremely valuable clinically to glean information concerning local regions of heart muscle that are close to an exploring precordial electrode, but this is a fleeting hope that melts in the face of experimental facts"[19].

Other workers, however, claim that proximity potentials exist, and are very important. Taccardi[30], Heppner[31] and others, have used excised turtle hearts in tanks of conducting solutions, and found that within a distance of less than two diameters of the heart, proximity effects are very evi-

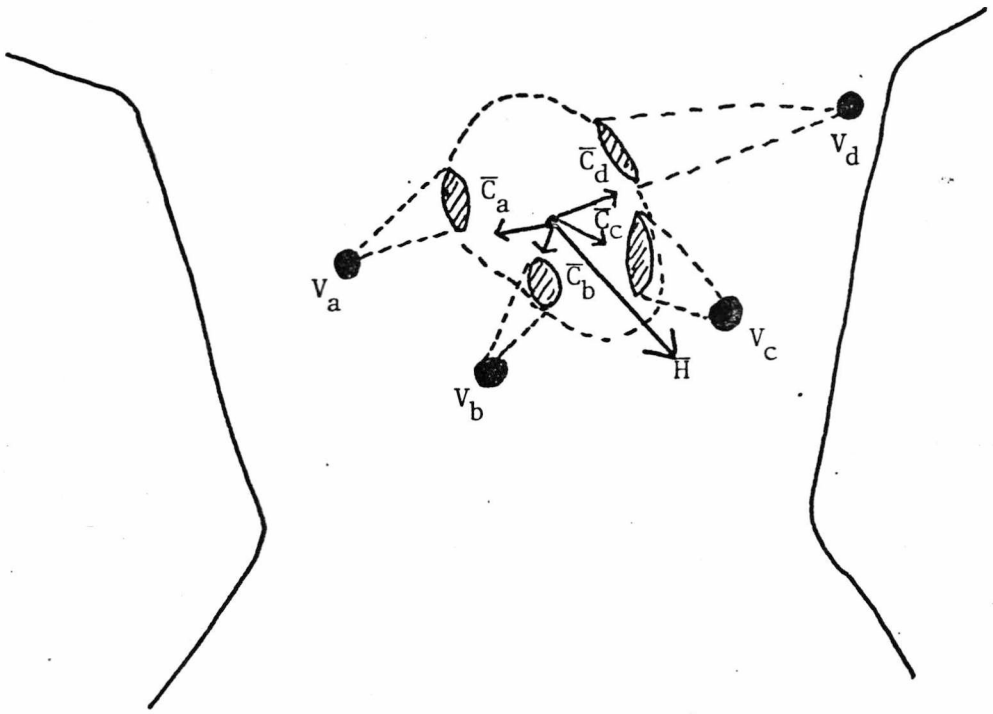
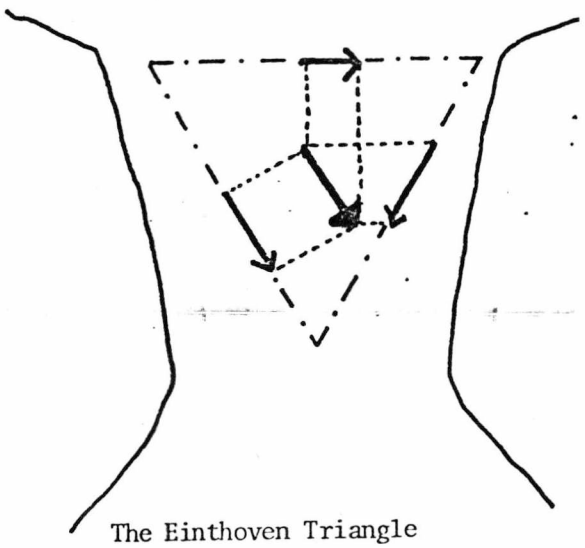
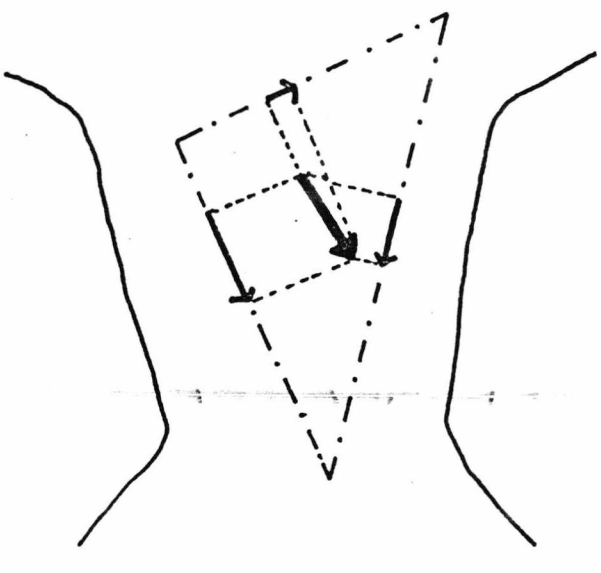


FIG. (19) : The two differing opinions on precordial leads are illustrated.



The Einthoven Triangle



The Burger Triangle which accounts for inhomogeneties in the conductivity of the torso.

FIG. (20)

dent. Katz[24] reports on work by Hartmann et al with an excised cat's heart in a goldfish bowl which confirmed the work of Taccardi et al, and then asks if these reports are true, then how can the simple dipole view of vectorcardiography be accepted? Geselowitz[29] in a review concludes that a fixed dipole hypothesis is unrepresentative of the situation, and if strictly adhered to, then clinically significant information will be lost.

Burger and Van Milaan had performed a set of experiments[17] on models to try to determine the effect that anatomical features such as the lungs, and bones and muscle had upon the fixed dipole theory. Using torso models they performed extensive tests to investigate the effects of imhomogeneties on the waveforms that a dipole produced at the surface. They found that the presence of areas of varying conductivity within the torso could not be ignored and whilst modifying Einthoven's triangle to a new shape *(Fig. (20))* to account for the lung tissue etc. (Burger's triangle) they concluded that the dipole hypothesis was true enough to support the idea of Wilson's Central Terminal, but not so true as to exclude precordial leads.

Geselowitz and others[29],[32] have calculated that the presence of a mass of highly conducting material near a non-dipolar source can make that source appear dipolar in behaviour. This is the situation within the body, since the ventricles contain volumes of blood - which is a much better conductor that the tissue itself[33]. Geselowitz himself, though, does state that it is an approximation that "does leave something to be desired"[29].

The discussion as to the nature of an exact equivalent

generator for the heart continues, and also as to the extent to which a pure dipole is representative of the situation or whether a multiple source has to be invoked. The parties involved each have their preferences as to vectorcardiographic leads or to the 12 lead electrocardiogram including precordial leads. McFee and Baule appear to sum up the situation in the following words "the main advantage of VCG leads relative to the 12 lead system appears to be that there are only three lead voltages to record, analyse, etc. rather than twelve"[16].

2.4 THE IMPORTANCE OF THE ECG IN CLINICAL DIAGNOSIS

2.4a Cardiovascular Disease

Whatever the final conclusions of the theoreticians as to the best model to explain the surface potentials generated by cardiac activity, the place of the electrocardiogram in routine clinical practice is well established, and unlikely to be displaced. The number of routine recordings taken at the Kent & Canterbury Hospital has increased from 1200 in 1970 to 6150 in 1975 and is expected to reach 7300 by the end of this year (1976)[34]. One of the reasons for McFarlane and Lawrie's[35] work on automatic routine diagnosis of the ECG was the large numbers of recordings being taken at Glasgow Royal Infirmary, and the expected burden that they would place upon the medical staff interpreting them (16,435 in 1964 to 25,318 in 1972).

Cardiovascular diseases account for 50% of the deaths in the Western World[36], of these half are due to ischaemic heart disease, and hypertension accounts for the rest. Rheumatic and congenital heart disease together account for 3% of deaths[36]. In view of these figures, it is perhaps

worthwhile to examine the ECG as it appears during some cardiac disorders to illustrate the reasons for its widespread acceptance as a diagnostic technique.

2.4b The Variation of the ECG with Disease

Patients with Angina Pectoris (pains in the chest and arms due to temporary inadequacy of the blood flow to the myocardium) frequently have a normal ECG inbetween attacks[37]. During the critical periods - sometimes induced for the purpose of diagnosis - the ECG undergoes changes in the form of S-T segment depression. Sometimes it is a horizontal depression *(Fig. (21))* or sometimes the S-T segment slopes downwards instead of following the isoelectric line *(Fig. (22))*.

Frequently, acute myocardial infarction (the necrosis of part of the cardiac muscle due to inadequate blood supply) follows attacks of severe angina, and here the effects upon the ECG are two-fold. Firstly, there will be changes in the basic patterns which are time dependent and, secondly, there will be an arrythmia - in 95% of the cases ; induced by the infarction[37]. The characteristic ECG changes are shown in *Fig. (23)*. S-T segment elevation occurs, and the Q wave widens and deepens. Then, after a time, T wave inversion occurs and the S-T segment returns to its normal place. Finally, the T wave returns to normal, but the Q wave usually stays the same[37]. The times of these changes are over several days, and are dependent upon the healing and accommodation properties of the heart, as it adjusts itself to the new situation.

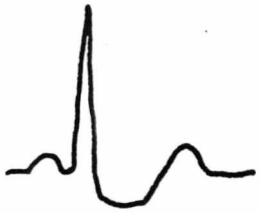


FIG. (21)

Horizontal S-T depression in Angina Pectoris.

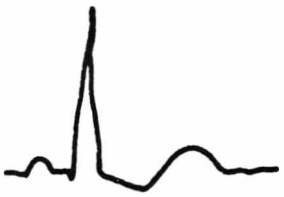
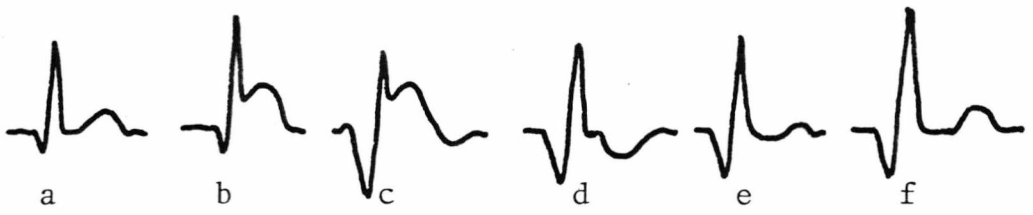
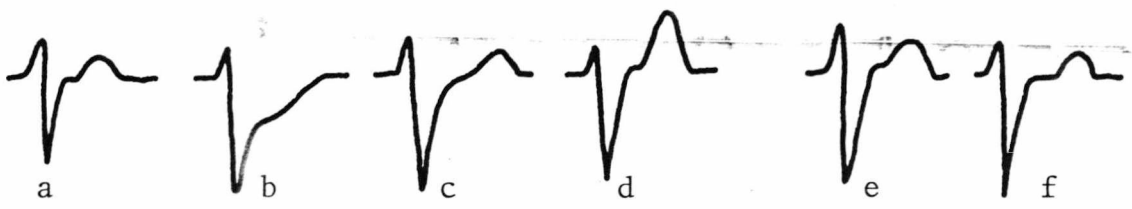


FIG. (22)

Sloping S-T segment in Angina Pectoris.



(a)



(b)

FIG. (23)

Changes in the ECG over a period of several days after myocardial infarction : (a) proximal electrode (b) distal electrode

The ECG can also be used to determine the extent of damage to the myocardium, and to give information to the clinician to modify his treatment to result in the smallest possible permanent infarct regions. It has been found that whilst different drug treatments can each save a person's life, and allow them to leave hospital; the extent of permanent myocardial damage differs between them[38]. Some treatments can reduce the infarct area, some have no effect on it at all, and some even increase the area. A map is plotted of 35 leads from the chest, and it is assumed that each recording gives information mainly from the area underneath the electrode[33]. The S-T segment elevation is measured in each case, and the map is plotted *(Fig. (24))*. Several of these maps are, in fact, plotted throughout the course of the treatment to see how the infarct size is varying *(Fig. (25))*.

As has been previously stated, 95% of acute myocardial infarction cases develop an arrythmia of one form or another, and its is these arrythmias that are the main causes of death - the infarction not necessarily being a cause of death in itself.

Atrial flutter *(Fig. (26))* denotes a very high atrial depolarisation rate - approximately 300 bpm. This is much more than the slowly acting A-V node can conduct, and so not every atrial beat is accompanied by a QRS complex. Flutter is fast, but reasonably regular whereas atrial fibrillation is the contraction of different parts of the atria completely asynchronously. The ECG is more reminiscent of a noisy baseline than a signal *(Fig. (27))*. The accompanying ventricular beats do not have any regular rhythm either.

(3)

MYOCARDIAL INFARCTION SIZE MEASUREMENT

NAME OF PATIENT ..DADD..RAYMOND..... AGE ....45....

WARD ....WEST..... HOSPITAL NO. .....

DATE ....3.:.12.:.74..... TIME ....4 P.M.....

NO. OF HOURS AFTER ONSET OF SYMPTOMS .....42 HRS.....

ENZYME LEVELS ON THE DAY OF THE RECORDING ..LDH..954...SGOT..295..

CLINICAL COMMENTS, i.e. drugs, etc.

PRE INFARCTOR.

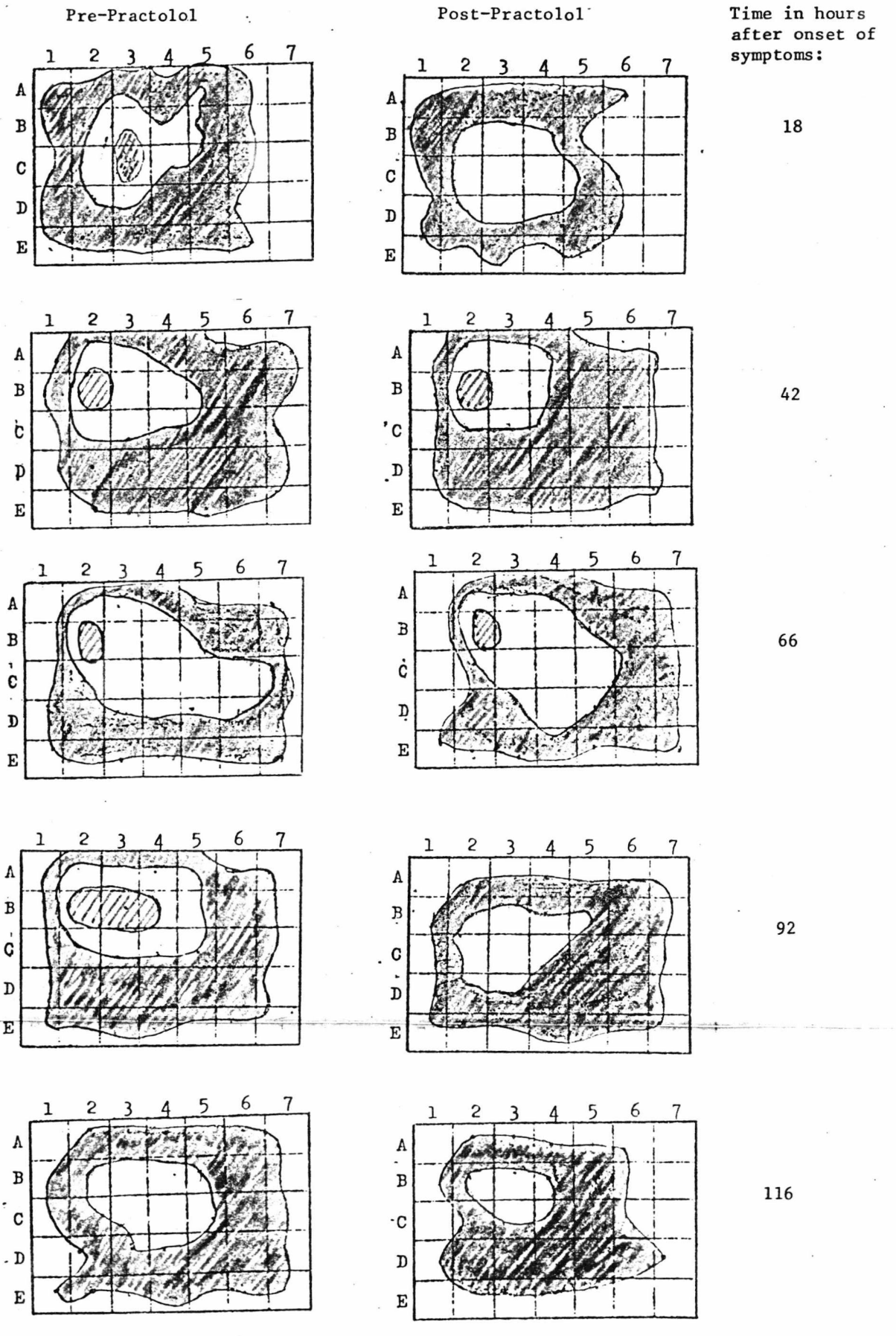
FURTHER PAIN IN PREVIOUS 2 hours

Total number with ST elevation greater than 6 mms
Total number with ST elevation between 4-6 mms
Total number with ST elevation between 2-3.9 mms
Total number with ST elevation between 0-1.9 mms
Total ST elevation

1	
10	
23	
56	

FIG. (24) : A typical map obtained from a patient with a myocardial infarction.

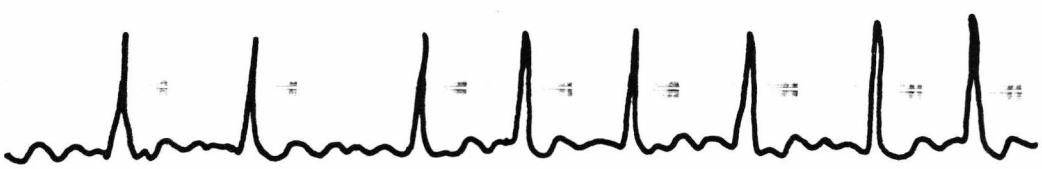
FIG. (25) : The set of maps obtained from a patient during the course of treatment.



Flutter

FIG. (26) : Atrial ~~Fibrillation~~

(a)



(b)

FIG. (27)

Figure (27) shows the irregular baseline in condition of Atrial fibrillation. (a) is for a low ventricular rate and (b) is for a high ventricular rate.

Ventricular ectopic ('extra') beats *(Fig. (28))* occur spontaneously within the ventricles and are occasionally present in some healthy individuals. After a myocardial infarction, however, the incidence increases enormously. Should an ectopic beat fall in the early part of a 'T' wave, then ventricular fibrillation occurs *(Fig. 29))*. The latter is a serious arrythmia as the pumping ability of the heart disappears. The ventricles contract irregularly and ineffectually at rates from 180 to 300 times a minute - with different parts of the ventricles beating independently of each other. If this is not treated within 4 minutes then serious brain damage occurs, and subsequent death if left unattended any longer[39]. It is here that the ECG is particularly useful as it enables fibrillation to be detected immediately (if the patient is being continuously monitored) rather than having to wait for other clinical signs to develop. Monitoring also provides staff with evidence of the rate of occurrence of ectopic beats so that if it is high, corrective action can be taken before fibrillation starts.

Metabolic changes such as hypokalaemia or hyperkalaemia (disturbance of the levels of serum potassium) *(Fig. (30))* provide a characteristic change in ECG[40]. The Q-T interval lengthens in both, with the T wave going biphasic in the former and a drooping of the S-T segment in the latter. Hyperthyroidism causes an unstable tachycardia with a rate in the range 100→150 b.p.m.[41] with a slight depression of the S-T segment *(Fig. (32))* and an enlarged T wave. Hypothyroidism *(Fig. (31))* gives rise to an ECG which has low values of Q.R.S. amplitude, flattened or

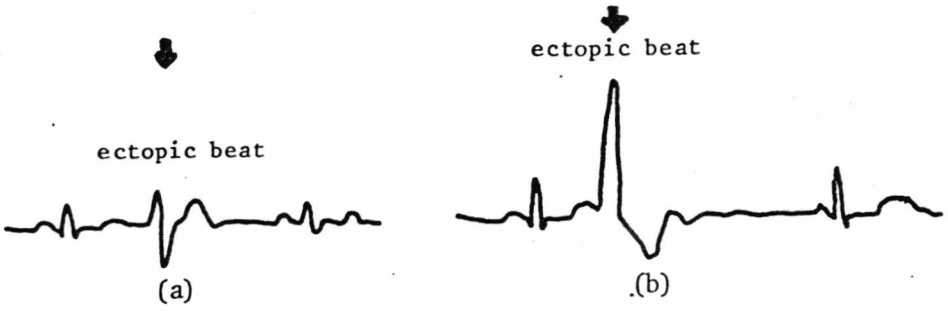
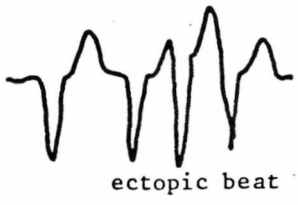


FIG. (28) : Examples of ventricular ectopic beats. Figures (b) and (c) show the ectopic beat occurring on the 'T' wave.



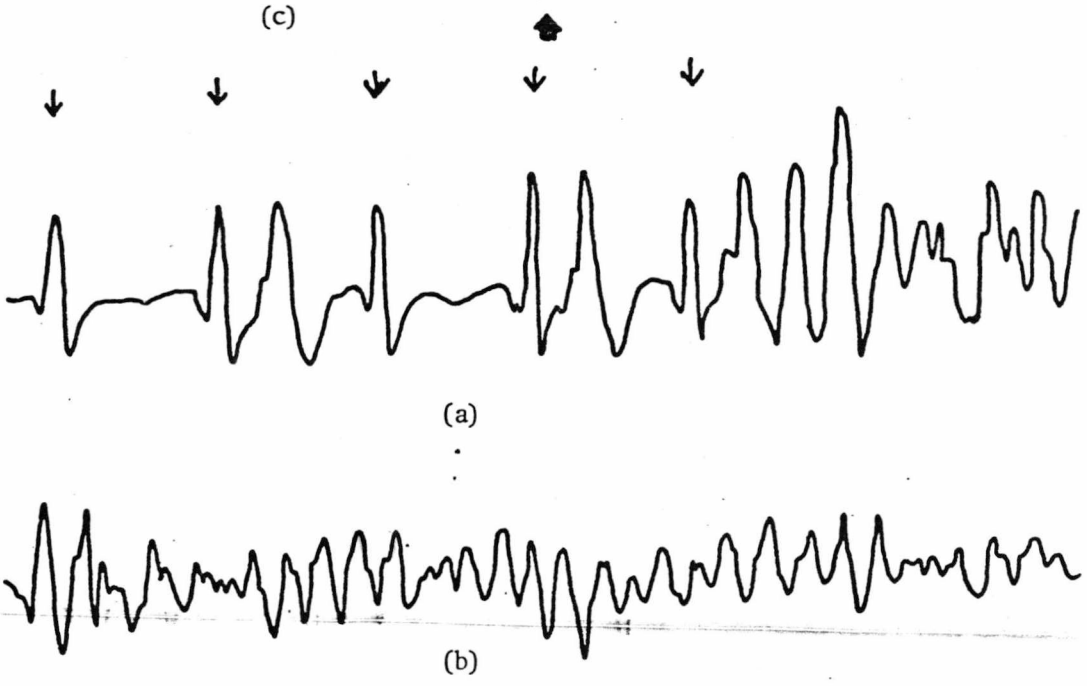


FIG. (29) : Figure 29(a) shows ectopic beats occurring in the electrocardiogram, with the normal complexes marked with arrows. After the last marked QRS complex, the ectopic occurring on the 'T' wave initiates ventricular fibrillation.

Figure 29(b) is a continuation of 29(a).

(a)

(b)

FIG. (30) : Examples of the ECG in (a) hypokalaemia, (b) hyperkalaemia.

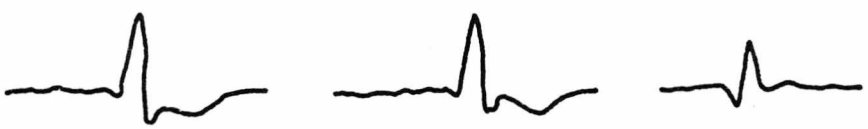


FIG. (31) : The ECG shapes of leads I, II and III in hypothryroidism are shown and are accompanied by sinus bradycardia.

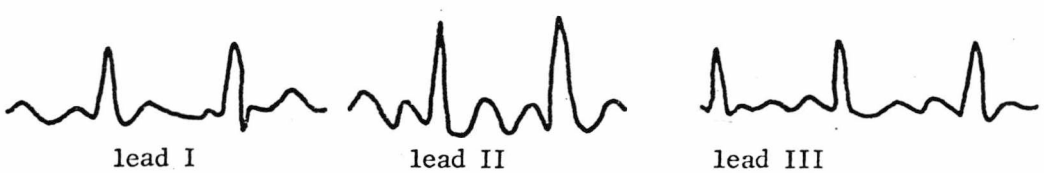


FIG. (32) : Hyperthyroidism is associated with unstable tachycardias, and the unstable baselines of leads I, II and III.

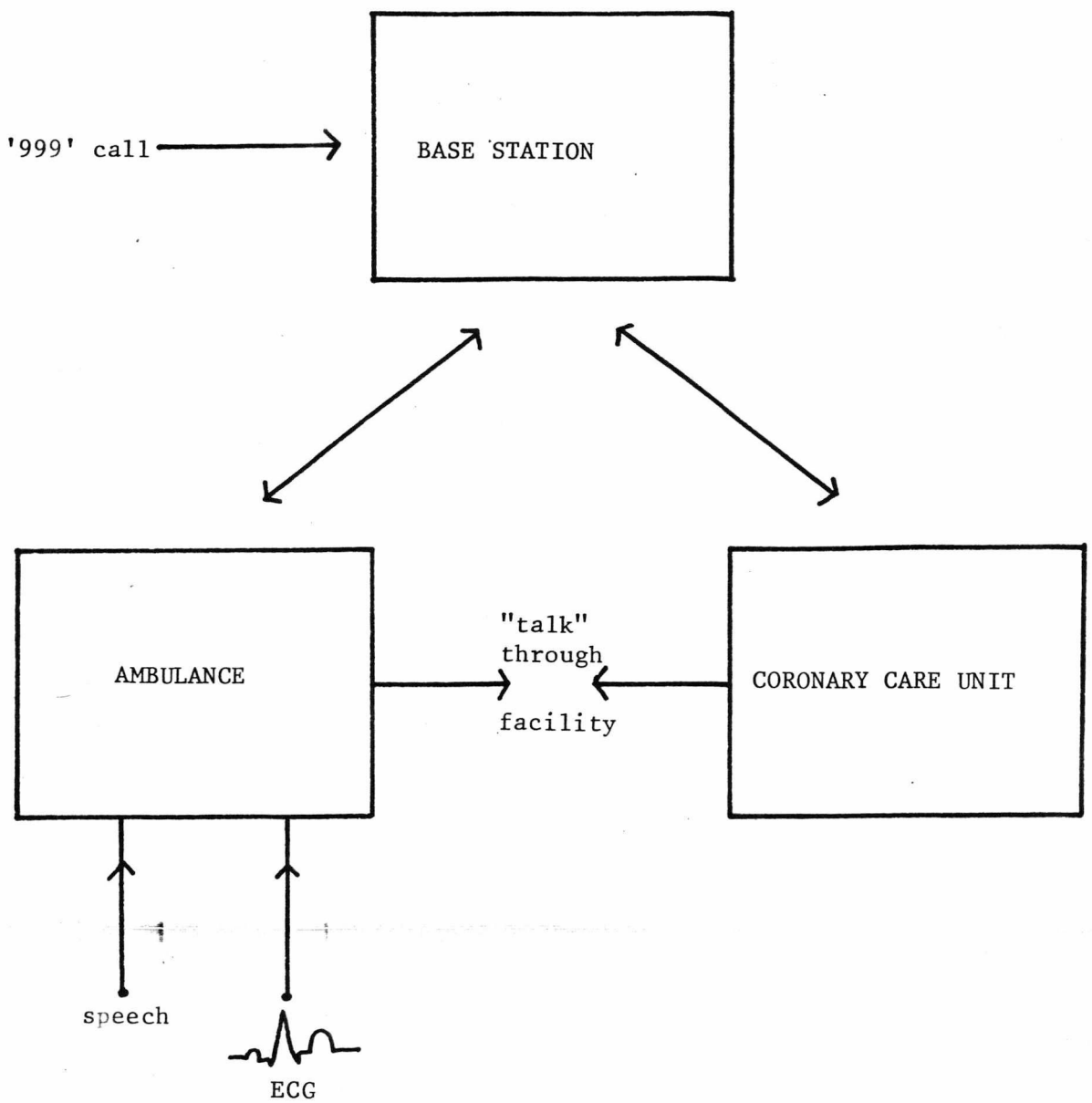

'999' call
BASE STATION
AMBULANCE
"talk"
through
facility
CORONARY CARE UNIT
speech
ECG

FIG. (1)

inverted T waves, and slight S-T segment depression.

2.4c Summary

The preceeding section gave but a very few examples of the wide variety of changes in the ECG that occur during illness (or even variations in normals). Owen et al[4] state "the value of electrocardiography as a diagnostic aid can hardly be overstated; it has a record of 95% accuracy in cardiac infarction, the commonest fatal disease of the Western World, while there is hardly any form of heart disease in which it is not at times of great help - not to mention occasional contributions to endocrine and metabolic problems".

It is because of its great importance in the treatment of cardiac disease that the ECG is highly valued, and this is why, in the remainder of the thesis, attention is concentrated on the transmission of the ECG from one place to another.

REFERENCES

(1) T. RUCH & H. PATTON "Physiology and BioPhysics" 1966. 19th Edition. Saunders and Company, Chapter 30.

(2) E.E. SELKURT (Editor) "Physiology 10 Authors" 1966. 2nd Edition. Little, Brown & Company.

(3) D.J. JULIAN "Cardiology" 1973. Published by Baillière Tindall, London, Chapter 3.

(4) S.G. OWEN, T.B. STRETTON & J. VALLENCE-OWEN "The essentials of Cardiology" 1968. Lloyd-Luke (Medical Books) London, Chapter 7.

(5) D.J. JULIAN, op.cit. p.35

(6) W. EINTHOVEN, G. FAHR & A. DeWAART "On the direction and manifest size of the variations of potential in the human heart and on the influence of the position of the heart on the form of the electrocardiogram" 1913. Translated from the original German by E. HOFFH & P. SEKELJ: American Heart Journal, 40, p. 163, 1950.

(7) F.D. JOHNSON "The spread of currents and distribution of potentials in homogeneous volume conductors" 1957. Annals New York Academy of Science, Vol. 65, p. 963.

(8) F.N. WILSON et al "Electrocardiograms that represent the potential variations of a single electrode" 1934. American Heart Journal, 9, p. 447.

(9) E. GOLDBERGER "A simple indifferent electrocardiographic electrode of zero potential and a technique of obtaining augmented unipolar extremity leads" 1942. American Heart Journal, 23, p. 483.

(10) E.E. KING "Errors in voltage in multi-channel ECG recordings using newer electrode materials" 1964. American Heart Journal, 18, pp. 295-297.

(11) L.A. GEDDES "Electrodes and the measurement of bioelectric events" 1972. Wiley Interscience, N.Y.

(12) M.F. DIPROSE & D.J. TAYLOR "Unpublished work" 1976. Kent & Canterbury Hospital Cardiac Department.

(13) H. SCHAEFER & H.G. HAAS "Electrocardiography" 1962. In handbook of Physiology, Vol. 1, (Dow & Hamilton Editors), American Physiological Association, Washington D.C.

(14) P. HAMMOND "Applied electromagnetism" 1971. Pergamon Press 1st Edition. Commonwealth and International Library Series.

(15) H.P. SCHWAN & C.F. KAY "The conductivity of living tissues" 1957. Annals of the New York Academy of Sciences, Vol. 65, p. 1007.

(16) R. McFEE & G. BAULE "Research in electrocardiography and magnetocardiography" 1972. Proceedings of the IEEE,

Vol. 60, No. 3, March, pp. 290-321.

(17) H.C. BURGER & J. VAN MILAAN "Heart vector and leads I" 1946. British Heart Journal, Vol. 8, p. 157.
- "Heart vector and leads II" 1947. Ibid, Vol. 9, p.154
- "Heart vector and leads III" 1948. Ibid, Vol.10, p.229

(18) W.J. DUFFIN "Electricity and Magnetism" 1965. McGraw-Hill, London.

(19) E. FRANK "Spread of current in volume conductors of finite extent" 1957. Annals New York Academy of Science, Vol. 65, p. 980.

(20) E. FRANK "Measurement and significance of cancellation potentials on the human subject" 1955. Circulation Vol. 11, p. 937.

(21) S.R. MOORE & P.H. LANGNER "Location of electrical centre of ventricular depolarisation" 1956. American Heart Journal, Vol. 51, p. 405.

(22) O.H. SCHMITT et al "Electrocardiographic mirror pattern studies" 1953. Parts I, II & III, American Heart Journal, Vol. 45, p. 416, p. 500 and p. 655.

(23) G.E. SIEDEN "Anatomic location of the electrical heart centre in patients" 1955. Circulation Vol. 12, p. 773.

(24) L.N. KATZ "The physiological basis of the spread of cardiac action currents through the body" 1957. Annals

of the New York Academy of Sciences, Vol. 65, p. 960.

(25) R.A. HELM & T. CHOU "Electrocardiographic cancellation - a study of a single dipole at variable locations" 1966. American Heart Journal, Vol. 72, p. 218.

(26) D.A. BRODY & G.D. COPELAND "Electrocardiographic cancellation - some observations concerning the non-dipolar fraction of precordial electrocardiograms" 1958. American Heart Journal, Vol. 56, p. 381.

(27) R.H. OKADA "An experimental study of multiple dipole potentials and the effect of inhomogeneities in volume conductors" 1957. American Heart Journal, Vol. 54, p. 567.

(28) R. McFEE & H. PARUNAGO "On the interpretation of cancellation experiments I" 1959. American Heart Journal, Vol. 58, p. 582.

- "On the interpretation of cancellation experiments II" 1960. Ibid, Vol. 59, p. 433.

(29) D.B. GESELOWITZ "Dipole theory in electrocardiography" 1964. American Journal of Cardiology, Vol. 14, p. 301.

(30) B. TACCARDI "La distribution spatiale des potentials cardiaques" 1958. Acta Cardiology, Vol. 13, p. 173.

(31) D.B. HEPPNER "Dipole and quadripole measurements of an isolated turtle heart" 1968. IEEE Trans. on Bio-

Medical Engineering, Vol. BME 15, p. 298.

(32) D.A. BRODY et al "The approximate determination of lead vectors and the Burger triangle in normal human subjects" 1956. American Heart Journal, Vol. 51, p. 211.

(33) H.H. HECHT "Some observations and theories concerning the electrical behaviour of heart muscle" 1961. American Journal of Medicine, Vol. 30, p. 720.

(34) Yearly records and statistics, the ECG Department - Kent & Canterbury Hospital, Canterbury, Kent.

(35) P. McFARLANE & T. LAWRIE "An introduction to automated electrocardiogram interpretation" 1974. Butterworth Press.

(36) D.G. JULIAN "Cardiology" 1973. Baillière Tindall, London. Introduction.

(37) D.G. JULIAN "Cardiology" 1973. Baillière Tindall, London. Chapter 3.

(38) P.R. MAROKO et al "Precordial ST segment mapping. An automatic method for assessing alteration in the extent of myocardial ischaemic injury. The effects of pharmocologic and hermodynamic intervention" 1972. American Journal of Cardiology, Vol. 29, p. 223.

(39) D.G. JULIAN "Cardiology" 1973. Baillière Tindall, London. Chapter 4.

(40) "Notes on Electrocardiography" 1969. Available from Selpharm Laboratories, Harrow, pp. 140-141.

(41) "Notes on Electrocardiography" 1969. Available from Selpharm Laboratories, Harrow, pp. 147-149.

CHAPTER 3

3.1 INTRODUCTION TO MOBILE CORONARY CARE UNITS

3.1a The MCCU Concept

The coronary care unit (CCU) was introduced into hospital practice as a means of giving cardiac patients a level of intensive care that would not otherwise have been available to them in the general medical wards. The importance of the CCU is now well established, and most hospitals in Great Britain have a unit. As experience was gained in this sector of medical care, it became apparent that the sooner the patient was treated the better. The majority of fatalities appeared to arise from the results of cardiac arrhythmias (which can be both treated and prevented) rather than from the myocardial infarction itself. These arrhythmias can occur at any time after the first infarction[1] and are most likely to occur within the first hour[2],[3]. Various studies of the admission pattern of CCU's showed the disturbing fact that the average time between onset of symptoms in a patient and the beginning of therapy was several hours - in some cases up to 12 hours or more[4],[5]. Clearly, by this time most patients had passed the most critical time of the illness and any further advance in coronary care would involve not just improvements in therapy but the decrease in admission times as well.

Moiseev in Moscow[6] and Pantridge in Belfast[7] examined this problem and offered mobile coronary care units (MCCU) as a possible solution. These were vehicles that had all the necessary equipment and drugs for administration of intensive coronary care on board and which were manned by a doctor as well

as the normal ambulance crew. As soon as any emergency call came through, the ambulance would move immediately to the scene, and the patient would be under intensive care. When the patient was in a suitable condition, with the risks of arrhythmia minimised, and not before, he would be moved to the hospital.

The initial results as shown by comparing "dead on arrival" rates before and after the introduction of MCCU's in Belfast, were dramatic. In the study by Pemberton and McNeilly[8] of the 414 patients that were alive when the ambulance reached them, 102 (25%) were dead on arrival at hospital. With the new mobile service provided by Pantridge, of the ~~32~~ 312 patients, all reached hospital alive[1] (0%). There is also evidence to suggest that the earlier the treatment is given, the greater the patients overall chance of leaving hospital alive - "It is, therefore, of interest that among patients seen early, in the majority of whom dysrhythmias were either prevented or quickly corrected, the incidence of shock and pump failure was low and the in-hospital mortality low"[9] and from Adgey et al[9] "Early initiation of intensive care not only prevents death from ventricular fibrillation, but also diminishes the incidence of shock and pump failure by preventing and immediately correcting dysrhythmias and limiting the size of the myocardial infarct. The resources for coronary care should be directed towards the pre-hospital phase of the coronary attack".

In spite of these encouraging results, there were disagreements about the concept of pre-hospital treatment. The most obvious factor was that not all hospitals could afford to buy, equip and maintain a special vehicle, nor have the

staff available to allow them to leave the hospital for, perhaps, several hours at a time, at a moments notice. The other objections were based on a rejection of the belief that substantial reductions in mortality can, in fact, be achieved by MCCU's.

3.1b The Arguments Against

A study by Smyllie et al in Doncaster[10] is an example of the method of thinking behind the rejection of the idea. Whilst gathering information needed to calculate the size of their new CCU[11], experiments were run simultaneously to evaluate the usefulness of a mobile unit. Their conclusions were completely at variance with those of Pantridge i.e. "We argue that the provision of MCCU's on request from general practitioners is unlikely to have any appreciable effect in preventing deaths from acute myocardial infarction outside hospital"[10].

This conclusion was based upon a study of admission times of patients to their CCU. They found that the median time that elapsed before the ambulance was called was 2 hrs. 23 mins. after onset of symptoms, and that the median times for collection of the patient by ambulance and their delivery at the hospital was just 25 mins.

Smyllie et al argued that since 60% of deaths occur in the first hour[2],[3], and that the median time for calling the ambulance was over two hours, therefore the extra delay due to ambulance travel was small and negligible and the time saved by a MCCU in getting a patient under care is small and has little effect upon the course of the illness. Therefore, it did not matter whether the ambulance brought corony care to the patient, or the patient was taken to the coronary care.

3.1c A Discussion of the Objections

This argument, however, shows a misunderstanding of the basic concept of mobile units. Arrhythmias and in particular ventricular fibrillation, are likely to occur at any time, and in these circumstances five, ten minutes, or longer in an ambulance whilst the patient is still at high risk is dangerous. Even the motion of the ambulance could perhaps trigger off an arrhythmia in a patient. The concept of the MCCU is not just to save time, but to get intensive therapy to the patient as soon as possible, and to make sure that the heart rhythm is stable before any attempt to transport the patient is made. That this is effective becomes apparent from the comparison mentioned earlier, between the figures of Pemberton and McNeilly[8] and Pantridge and Geddes[1].

It is misleading, also, to make too rigid a criterion of the median times between events in making an evaluation of a MCCU. They are, after all, just median times, and although it is true that half of the patients take 2 hrs. 23 mins. or more to call an ambulance - what about the other half? These are the ones who take less than two hours before summoning help. That this group of people must be considered can be seen from a further study of Pantridge and Adgey[9]. In this they discuss the results of reaching 284 patients (out of 1150 i.e. 25%) in less than one hour after onset of symptoms - in 79 of these cases they were treating the patient within 30 minutes of the first attack. This number is definitely not negligible and interestingly enough the median time (in Belfast) before calling the ambulance was found by Pemberton and McNeilly to be 3 hrs. 30 mins.[8].

3.1d MCCU's at the Present Moment

Since these early trials that have been described, the number of units has multiplied throughout the world. It is especially popular in the United States, and the Soviet Union (who would like 1 ambulance per 10,000 population[12]!) and countries such as Sweden[13], Germany[14], Poland[15], Australia[16], are among other countries, apart from the U.K., who are operating MCCU's. The idea now seems to have gained firm acceptance with the majority of the medical profession, although the types of MCCU's actually operating are very diverse.

3.2 DIFFERENT SYSTEMS OF MOBILE CORONARY CARE

The Mobile Coronary Care Unit (MCCU) was first introduced into regular Health Service operation in Moscow in 1962[8],[17],[18] by Moiseev, and this was followed by the trials of Pantridge and Geddes[1],[7] in Belfast in 1966. Following on from the results of these pioneers several other hospitals in the United Kingdom and the United States of America are now operating mobile units[25]-[19]. Each group has developed its own operating system depending upon ~~this~~ their particular circumstances but two main classes of pre-hospital care have emerged:-

(a) Those which transport qualified medical staff to the patient.

(b) Those which rely upon semi-skilled personnel for administration of treatment as directed by expert personnel at the hospital, through radio communication.

The former service (a) offers an excellent standard of pre-hospital care for a patient - although it is expensive in

terms of qualified manpower. The latter scheme (b) however, is more attractive to small hospitals who have to consider cost and personnel carefully. Five basic schemes of mobile coronary care have been selected for discussion, and are listed below.

(1) Ambulances with doctors and nurses.

(2) Ambulances with telemetry and no treatment of patients.

(3) Ambulances with telemetry and nurse and semi-skilled ambulance men.

(4) Ambulances with telemetry and well-trained ambulance men.

(5) Ambulances with highly trained ambulance men.

The first of these schemes is as used by Pantridge and Geddes, Kernohahn and McGuchen and Walsh et al, all in Northern Ireland[7],[20],[22], and Palm et al, Denmark[12] and Sandler et al, Barnsley[26]. An ambulance or van is fully equipped with cardiac monitoring and pacing equipment, a portable defibrillator, oxygen, entonox and a range of drugs. It is kept on standby in close proximity to the hospital coronary care unit (CCU), and when an emergency call is received, a doctor and nurse go to the ambulance which can be on its way in a few minutes from the receipt of the call. As soon as the ambulance arrives, the patient is under intensive care and is not moved until the doctor has decided the heart rhythm is stable and it is safe to do so.

This type of care is the best that can be provided, but it does depend upon the hospital having enough staff to be able to send with the mobile unit at any time of day or night. All of the hospitals serve a large population (500,000, 250,000,

165,000 respectively) and consequently have large staffs to be able to run such a service.

The first of the telemetry methods listed - that as used by Woodwark and Gillespie[23] and Uhley[24] both in the U.S.A. just uses radio equipment to send the patients ECG to the Coronary Care Unit in the hospital. Although no treatment is given to the patient, this method does forewarn the hospital of the patient's condition and facilitates admission.

This method now seems to have virtually disappeared, at least in America. It grew up in the early days when non-professionally qualified people were disallowed from giving injections and other forms of treatment. In the United States now the laws have been amended in several States[27] and para-medical personnel (often fireman[28]) are now able to give intravenous injections of lidocaine, atropine, epinephrine, and perform intubations, cardiopulmonory resuscitation and any other necessary tasks[29].

For the patient to receive the benefit of treatment prior to their admission to a hospital, some method of administering it must be found, and the remainder of the schemes (numbers 3, 4 and 5) are different ways of doing this. The difference between them arise in the degree to which the ambulance crew are trained to treat patients with real or suspected myocardial infarctions. If a nurse is sent out with the ambulance then drugs and injections can be given by her, and the ambulance men can assist the nurse when required[30]. The nurse would be in radio contact with specialist staff in the CCU and these would diagnose the patient's condition and recommend a course of treatment. It would be necessary to give the ambulance men training and knowledge of the methods of intensive care of patients but this need not be too elaborate since the main

responsibility for the administration of treatment would lie with the nurse. This method does depend upon a nurse being available to travel with the vehicle at all times and would rely upon a large number of nursing staff who had experience of intensive care of patients, working in the hospital.

To avoid the necessity of having a nurse travel with the MCCU it is possible to train the ambulance men to be proficient in treating patients themselves under direction from the hospital by telemetry, and this is how the Bristol[31] team and several American teams work[28],[29]. They would have to be able to cope with injections, intubations, defibrillation and all other aspects of intensive care of a patient. This method would make the ambulance more independent of the hospital, in that it need not be stationed adjacent to it but the responsibility of diagnosis and decisions still lies with the hospital staff. It would mean, however, that the ambulance men would have to be very well trained in these techniques, and hospital staff would have to plan and execute a comprehensive programme of instruction. This would have to allow the ambulance men to have training periods both in the Coronary Care and Intensive Care Units (ICU) as well as attending lectures and demonstrations.

The final method to be discussed is that of Chamberlain et al at Sussex[21] and Gearty et al in Dublin[25]. The telemetry link with the hospital is discarded and the ambulance crews are trained to examine patients' ECGs, make diagnosis and to give treatment. The removal of the data link to the experts in the CCU does put responsibility on the man in the ambulance, and an indirect one upon the hospital to train the men sufficiently. It seems to be rather a waste of consultant staff to try to duplicate their skills when all that is really

needed is an "extension of their hands" - which can be provided by semi-skilled men via the radio. Training becomes comprehensive and the men at Sussex undertake at least a six month course. Chamberlain et al claim that telemetry is not comprehensive since there may not be anyone in the CCU able to read the ECGs and instruct the mobile unit. This seems an unlikely situation, as if it were so, nobody would be available to treat the patient when he arrived either, i.e. a complete breakdown of the CCU concept.

3.3 THE SITUATION IN CANTERBURY

3.3a Staff and Area of Service

The Kent and Canterbury Hospital serves a population of approximately 250,000 both in the City itself and the surrounding rural areas. The hospital has a six bed CCU and a six bed ITU, and after 6 o'clock at night it is the only centre for emergency admissions in South East Kent. From discussions with senior staff[32], it would appear that the manpower is not available to introduce coronary ambulances in the style of Pantridge and Geddes. This appears to be the situation for the near future, and so any MCCU operated by the hospital would have to use trained ambulance men and telemetry, with or without an attendant nurse.

The large rural area for which the hospital has to provide an emergency cardiac service, must be taken into consideration when planning a MCCU. The delays that occur in bringing cardiac patients from outlying districts to the CCU could be well balanced by giving the patient intensive treatment as soon as the ambulance reaches him.

3.3b The Mobile Unit and Equipment

It would be unnecessary to equip all ambulances with the essential equipment, since only one is required. At present the Canterbury Ambulance Station has only one emergency ambulance and this must be used for all types of emergency medical treatment, not just for cardiac cases, and must be kept at readiness at the Northgate centre at all times. This means that whatever the final solution, whilst the system is being implemented and personnel trained, another separate ambulance must be used. It would be preferable to have this stationed near the CCU at the hospital - not only for when a nurse has to be collected but also to enable the crews to carry on training and gaining experience in the hospital, whilst waiting in between calls. (If an ambulance is not available, a small van, as used by Walsh et al[20], could be used).

This situation would allow the MCCU to operate for cardiac cases only and would not interfere with the normal emergency services that are provided by the Ambulance Corps.

The medical equipment in the unit must include a portable defibrillator and a portable ECG machine. The radio for telemetry will already be in the ambulance and the necessary encoder for sending the ECG signals will have to be provided. The CCU will have to contain a radio transreceiver to enable conversation to be carried on with the ambulance and a decoder for the ECG with the necessary recording devices. It would be difficult to use the telephone as a link to the ambulance, since the electronics that will have to be in the ambulance centre radio department would have to be duplicated between Canterbury and Thanet since after 6 o'clock each night, Thanet becomes the centre of ambulance operations for the area.

3.3c Training

Since the MCCU will have to be operated on a 24 hour basis, this implies three crews (8 hour shift each), and there will have to be others in case of accident, illness or holiday. This means that at least a dozen men will have to be trained by the hospital to be able to operate the mobile unit continuously. Arrangements must not only be made by the hospital to provide the instruction and teaching, but the ambulance staff will have to be released from regular duties to undertake the course. Continuation training can be undertaken during quiet periods if the MCCU is kept at the hospital.

3.3d The Legal Situation

At the time of writing the liberal laws operating in the U.S., giving wide powers of treatment to para-medical personnel whilst under direction, do not operate in the U.K. Thus it is against the law for British and many European ambulance men to give injections[33]. This is a problem which can only be overcome by legislation. The immediate consequence of this is that any MCCU operating in this area must have qualified medical staff present, since the injections of drugs such as lignacaine or atropine is a vital part of anti-arrythmic therapy[34].

3.3e Telemetry and Communications

The success of the venture will not only depend upon the training of the crews, but also upon the reliability of the telemetry link between the hospital and the MCCU. The present equipment utilises the ambulance radio, by processing the ECG into a form suitable for transmission. The policy of the GPO authorities at present seems to be against the issuing of

licences for separate channels for ECG transmission[31], and this means that although it would be highly desirable to have simultaneous voice communication and ECG transmission[24], this will be impossible. When there is wider use of MCCUs amongst hospitals there will be a case for the GPO to review its policy. Until then, however, use must be made of the existing ambulance radio. It has been said, that a dangerous situation could occur if, during a transmission, a life threatening arrhythmia occurs. The hospital would be unable to warn the ambulance and give instructions for emergency treatment until the ECG transmission had ceased. This may not be such a serious problem in practice, however, since there will be an ECG monitor in the ambulance. It is highly likely that the attendants themselves will recognise the advent of dangerous arrhythmias and interrupt the transmission to obtain the hospital's opinion. In America[23] it is the practice to have a small ECG transmitter, which is strapped to the patient wherever he is and the ECG is radioed to the ambulance, and thence re-routed to the hospital. This is useful if it is impossible to move the patient at all, although it does not seem to be the practice of any of the U.K. teams, and there is no mention or comment on it in their papers. This has, however, been independently suggested by the ambulance men from Northgate, during discussions of the project with them. It is a refinement which can easily be added to the system at a later date, should the need be found to arise.

3.4 A PROPOSAL FOR A TYPE OF MCCU FOR CANTERBURY

From the preceding discussion and in the present circumstances of the Canterbury Area, it would appear that the best method of mobile coronary care that can be offered is to have

a fully equipped ambulance at readiness at the hospital, manned by a series of crews who have had training in intensive coronary care techniques, and in contact with the consultants of the CCU via a radio telemetry link.

In most cases that the ambulance attends, the request for assistance comes from a doctor[8],[25] and so there is someone on hand, who can administer drugs if required, and a nurse need not travel in the ambulance. One of the major considerations of pre-hospital care, however, is to get treatment to the patient as soon as possible. There is often a delay in patients calling a doctor, and then in the doctor arriving[8],[10]. One of the aims of a MCCU service to the community should be a degree of public education so that the unit can be called directly by the patient or his relatives. This would obviously result in more false alarms but would considerably shorten the time before treatment and hence increase the recovery chances of many patients.

In the initial stages of the scheme, however, the cooperation of the local GPs would be called for, and most of the calls would have to come from them[25]. This would mean that no nurse would be required to travel in the ambulance, since the former will be present to administer necessary therapy, a useful feature from the hospital's point of view.

Should the staffing situation improve, then nurses and perhaps doctors can be sent out with the ambulance, to make the system more sensitive to calls from the general public, but at present, the success of the scheme will depend upon the ability of the remote consultants to correctly diagnose the patient's condition and to recommend the correct course of treatment. To do so, they must have reliable ECG data from the

telemetry link, and so a method must be found of relaying ECGs from the ambulance to the CCU with no degradation of quality. The method of doing this forms the subject of the next chapter.

REFERENCES

(1) J.F. PANTRIDGE & J.S. GEDDES "A mobile intensive care unit in the management of myocardial infarction" 1967. The Lancet, Vol. 2, p. 271.

(2) W.M. YATER, A.H. TRAUM et al 1948. American Heart Journal, Vol. 36, p. 334.

(3) C.R. BAINTON & D.R. PETERSON 1963. New English Journal Medicine, Vol. 268, p. 569.

(4) B. MITTRA 1965. The Lancet, Vol. 2, p. 607.

(5) B. LOWN et al 1967. Journal American Medical Association, Vol. 199, p. 188.

(6) S.G. MOISEEV 1962. Sovetsk. Medicine, Vol. 26, No. 6, p. 30.

(7) J.F. PANTRIDGE & J.S. GEDDES "Cardiac arrest after myocardial infarction" 1966. The Lancet, Vol. 1, p. 807.

(8) J. PEMBERTON & R.H. McNEILLY "Duration of last attack in 998 fatal cases of coronary artery disease and its relation to possible cardiac resuscitation" 1968. British Medical Journal, Vol. 3, p. 139.

(9) J.F. PANTRIDGE, A.A.T. ADGEY, et al "Acute phase of myocardial infarction" 1971. The Lancet, p. 501, 4th Sept.

(10) H.C. SMYLLIE et al "Acute myocardial infarction in Doncaster - delays in admission and survival" 1972. British Medical Journal, Vol. 1, p. 34.

(11) H.C. SMYLLIE et al "Acute myocardial infarction in Doncaster - estimating the size of the coronary care unit" 1972. British Medical Journal, Vol. 1, p. 31.

(12) T. PALM et al "A mobile emergency care unit - 2 years of experience" 1974. Acta Anaesthesiology Scandinavia, Vol. 18, p. 195-200.

(13) F. DANNERT et al "Ambulance Transport and Nursing in the Ambulance" 1975. Lakartidningen 72(45)4345-6.

(14) E. ROTTER et al "The medical emergency ambulance service of the fire brigade - Lower Austria" 1974. Anaethetist 23(2), p. 95.

(15) J. MATCZAK "3 years experience with the organisation of intensive extra hospital care by the Ambulance Service in Lödz" 1971. Polski Tygodnik Lekarski, Vol. 26, p. 810-812.

(16) F.W. FREEMAN et al "A coronary care ambulance controlled by radio telemetry" 1975. Medical Journal of Australia 1(5) p. 132-4.

(17) E.J. CZAZOV "Experience with the work of specialised first aid service and specialised units for treating patients with myocardial infarction" 1969. Arch. Klin.

Med. 216, p. 258-266.

(18) A. SMETNEV "Mobile coronary care unit" 1969. W.H.O. CCU Study Group, Moscow.

(19) M.J. WALSH et al "Mobile coronary care" 1970. The Lancet, 18th July.

(20) M.J. WALSH et al "Mobile coronary care" 1972. British Heart Journal, Vol. 34, p. 701.

(21) D.A. CHAMBERLAIN, N.M. WHITE et al "Mobile coronary care provided by ambulance personnel" 1973. British Medical Journal, 22nd Sept., Vol. 3, p. 618.

(22) R.J. KERNOHAN & R.B. McGUCKEN "Mobile Intensive Care in myocardial infarction" 1968. British Medical Journal, Vol. 3, p. 178.

(23) G.M. WOODWARK & I.A. GILLESPIE "Monitoring of ambulance patients by radio telemetry" 1970. Canadian Medical Association Journal, Vol. 102, p. 1277, 6th June.

(24) H.N. UHLEY "Electrocardiographic telemetry from ambulances - a practical approach to MCCU" 1970. American Heart Journal, Vol. 80, No. 6, p. 838.

(25) G.F. GEARTY et al "Pre-hospital coronary care service" 1971. British Medical Journal, Vol. 3, p. 33-35.

(26) W. ROBINSON & G. SANDLER "Mobile coronary care in Barnsley" 1973. Nursing Times, March 29, p. 401.

(27) M.I. SHANHOLTZ & D.K. WIEKING "Law Affecting Emergency Medical Services and Rescue Squads" 1974. Virginia Medical Monthly, Vol. 101, July, p. 577-79.

(28) E.L. NAGAL et al "Telemetry medical command in coronary and other mobile emergency care systems" 1970. J.A.M.A., Vol. 214, p. 332-338.

(29) R.R. LIBERTHSON et al "Pre-hospital ventricular defibrillation" 1974. New England Journal of Medicine, Vol. 291, Aug., p. 317-21.

(30) W.S. GRAF et al "Community programme for emergency cardiac care" 1973. J.A.M.A., Vol. 226, Oct., p. 156-60.

(31) DR. P. BASKETT "Personal Communication" 1974. Anaesthetics Department, Frenchay Hospital, Bristol.

(32) CONSULTANT ANAETHETISTS & PHYSICIANS 1973/74 "Personal Communications" Kent and Canterbury Hospital, Kent.

(33) C.R.A. MARTIN "Mis-use of drugs Act 1971" 1973 in Law Relating to Medical Practice, Pitman Medical Series.

(34) J.P. SHILLINGFORD "The management of acute myocardial infarction" 1971. Postgraduate Medical Journal, Jan., Vol. 47, p. 23-26.

CHAPTER 4

THE TELEMETRY OF THE ECG FROM AN AMBULANCE

4.1 THE COMPLETE AMBULANCE ECG TELEMETRY SYSTEM

The complete communication system for an MCCU, as suggested at the end of the previous chapter is shown in *Fig. (1)*. The initial emergency call goes to the Ambulance Base Station, and the MCCU is dispatched immediately. The base station will then call up the CCU on its radio and alert the ward. Upon arrival at the patient the ambulance is put in direct contact with the CCU by the "talk through" facility controlled by the base station, and so the dialogue concerning the treatment and the transmission of ECG's can commence.

With the exception of ECG transmission, the equipment for the above system is already available and in service, and so only the circuitry to adapt the ECG's into a suitable form need be developed.

4.2 THE DESIGN REQUIREMENTS FOR ECG TRANSMISSION FACILITIES

The ECG as detected on the body suface by standard electrodes is a signal with a peak amplitude (the R-wave) of, on average, 1mV. The actual size varies from person to person and also according to which lead is selected, with a range of values from $\frac{1}{2}$mV to 2mV. In the final operational version it will be necessary to transmit the full 12 lead ECG, but for the evaluation experiments only leads I, II and III were made available. To provide all the leads, only the input switching to the ECG preamplifier will need to be altered - no other changes in the circuitry will be required.

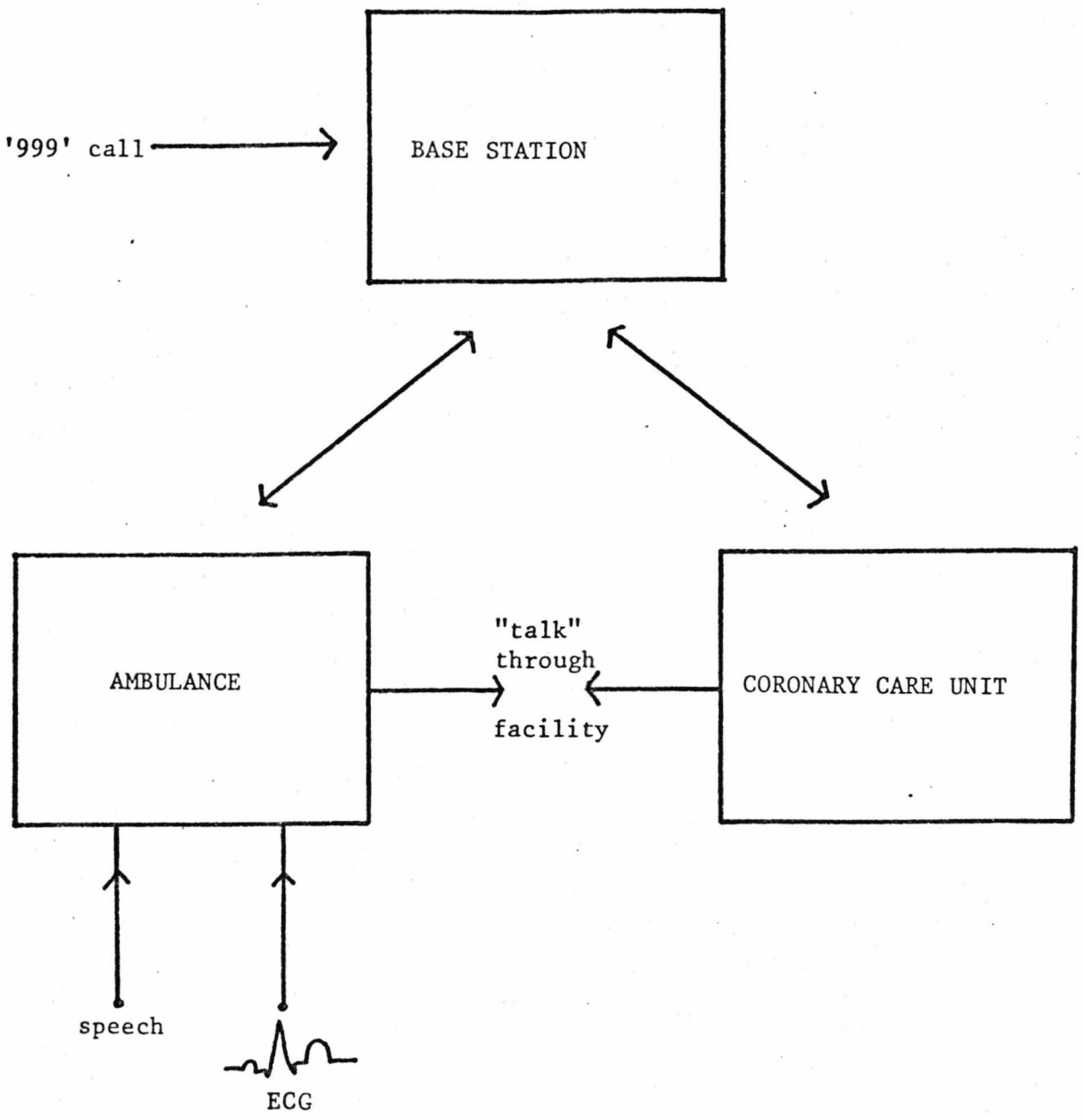

'999' call
BASE STATION
AMBULANCE
"talk"
through
facility
CORONARY CARE UNIT
speech
ECG

FIG. (1)

The bandwidth of the amplifier used for ECG work (as measured between the 3dB points) is now required to be from 0.05 Hz to 100 Hz[1]. The lower frequency limit is usually expressed as a time constant of 3 secs. Feldman et al[2] have performed Fourier analysis on the ECG and found that most of the information is contained in frequencies below 30 Hz. Nelson[3] uses a bandpass filter to detect the Q.R.S. complex and this has a centre frequency of 8 Hz, and the work of Morriss et al[4] show that the frequencies with greatest energy are in the 8 to 12 Hz region and so all agree with the requirements of[1].

Although some groups[5],[6] claim that significant information is to be found in the 100 Hz to 5 KHz region, for most practical purposes, 100 Hz is taken as the upper limit. This is consistent with clinical practice since most of the recorders in general use have a hot stylus marking heat sensitive paper, and this combination has a low writing speed, often with an upper frequency limit of 50 Hz. The frequency spectra of pathological ECG's are not significantly different from those of normal ones[7],[8].

The transmissions must be continuous and in real time, and provision must be made for simple switching between voice and ECG signals with the existing 2-way radios in the ambulances being used. The latter has an audio input bandwidth of 300 Hz to 3.5 KHz, thus the ECG must be transposed to a suitable frequency.

This is effected by having a subcarrier signal generated by a voltage controlled oscillator whose centre frequency of

2 KHz is frequency modulated by the incoming ECG signal. When the signal is received at the CCU, the subcarrier is decoded, and the ECG is extracted and suitably displayed to the clinician.

4.3 THE ELECTRONICS FOR TRANSMITTING THE ECG

4.3a The Circuitry Developed

A diagram of the device used to encode the ECG signal is shown in block diagram form in *Fig.* (2) and the elements of the decoder are shown in *Fig.* (3). The circuit diagrams are shown in *Figs.* (4(i), (ii)) and *Figs.* (5(i), (ii)), respectively.

4.3b The Encoder

The ECG preamplifier (*Fig.* (4(i))) is a differential type using an F.E.T. input stage with both defibrillation and patient protection. The patient lead is selected by S_2 which has four positions - one for a 1mV calibration input and the other three for the standard leads I, II and III. The defibrillation protection is provided by D_1 and D_2 and R_1 and R_2. Should a large voltage be present on the leads when the patient is defibrillated, then either D_1 or D_2 conducts and clamps the input to the F.E.T. at 0.6 volts, thus protecting it from damage. R_1 and R_2 are current limiting resistors to protect the diodes. Should anything go wrong with the equipment itself, then once again D_1 and D_2 prevent harmful effects; this time, to the patient. If a worst case fault is assumed i.e. one gate goes to +9 volts and the other to -9 volts (due to device breakdown or wires touching) then D_1 or D_2 becomes

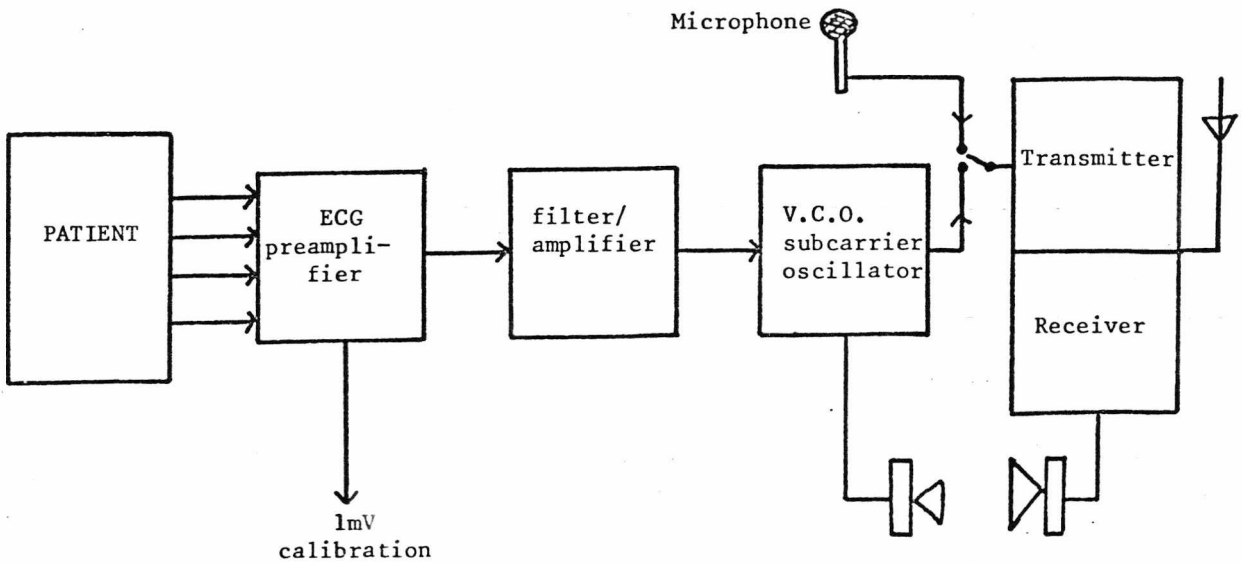

Microphone
PATIENT
ECG preampli-fier
filter/ amplifier
V.C.O. subcarrier oscillator
Transmitter
Receiver
1mV calibration

FIG. (2)

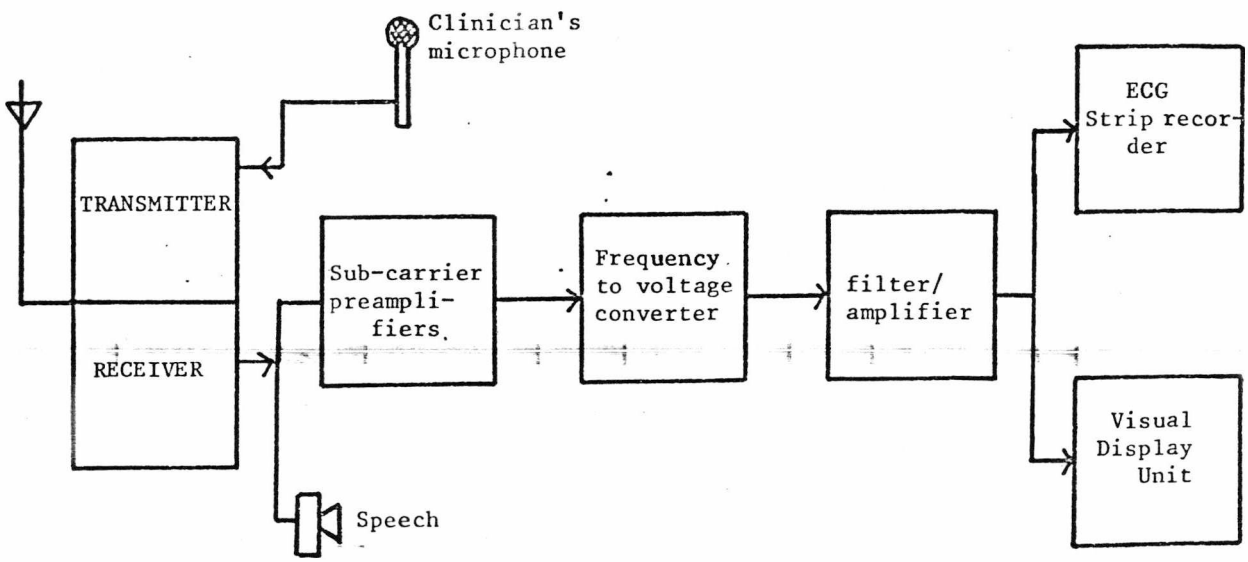

Clinician's microphone
TRANSMITTER
RECEIVER
Sub-carrier preampli-fiers.
Frequency to voltage converter
filter/ amplifier
ECG Strip recor-der
Visual Display Unit
Speech

FIG. (3)

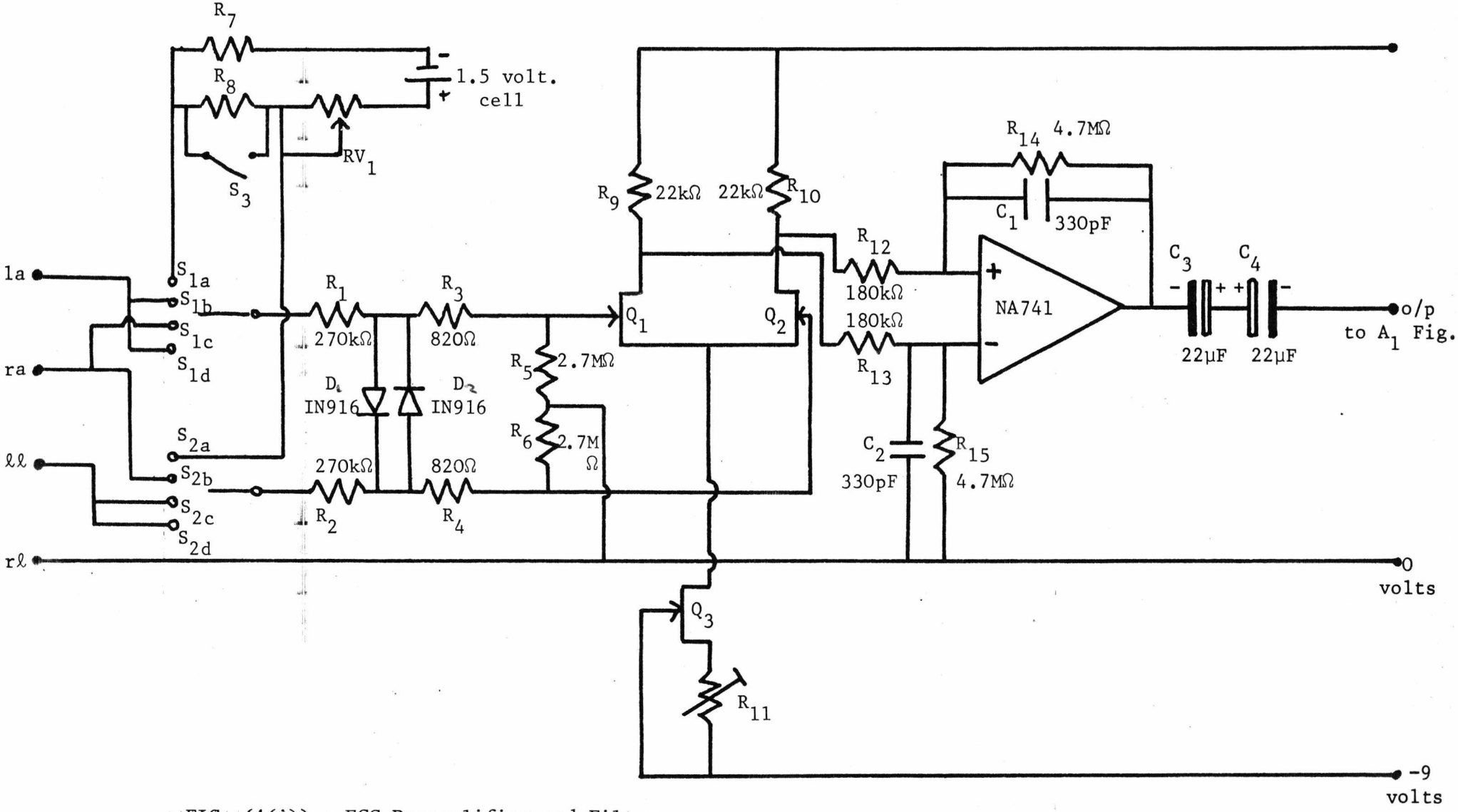


FIG. (4(i)) : ECG Preamplifier and Filter

forward biased with R_3 and R_4 limiting the current to 10mA. If it is assumed there is a short circuit path through the patient via the electrodes, then due to R_1 and R_2 only 1.2μA flows in the patient - which is well within safety limits[1].

The F.E.T. is a dual matched type in a long tailed pair differential amplifier circuit (Q_1,Q_2) and a constant current is supplied by Q_3, the value of which is determined by the source resistor, R_{11}. An integrated circuit differential amplifier is used to follow the first stage, and uses 741 type as the active element; capacitors C_1 and C_2 in parallel with the feedback resistors R_{14} and R_{15} are included to give high frequency attenuation with the -3dB point at 100 Hz. The lower 3dB point is determined by the coupling capacitors C_3 and C_4 and the input resistance of the next stage R_{16}. Two electrolytic capacitors are placed back to back to give a polarity independent one of value 11μF. The value of R_{16} is 270kΩ to give a time constant of ≃3 secs as required.

The voltage controlled oscillator (VCO) *(Fig. (4(ii)))* is an integrated circuit type which produces a square wave output whose frequency depends upon the control voltage applied to pin (5) and the values of C_5 and R_{20}. The control voltage is taken from the collector of Q_4. With no input to the amplifier A_2, its output and hence the base voltage of Q_4 is zero, and so the emitter is slightly below this. Variation of VR_2 sets the required current through the device Q_4 and hence its collector voltage and the basic frequency of the VCO - chosen to be 2 KHz. Inputs from the ECG preamplifier are amplified by A_2 and used to modulate the current through Q_4 and produce variations of the frequency of the oscillator.

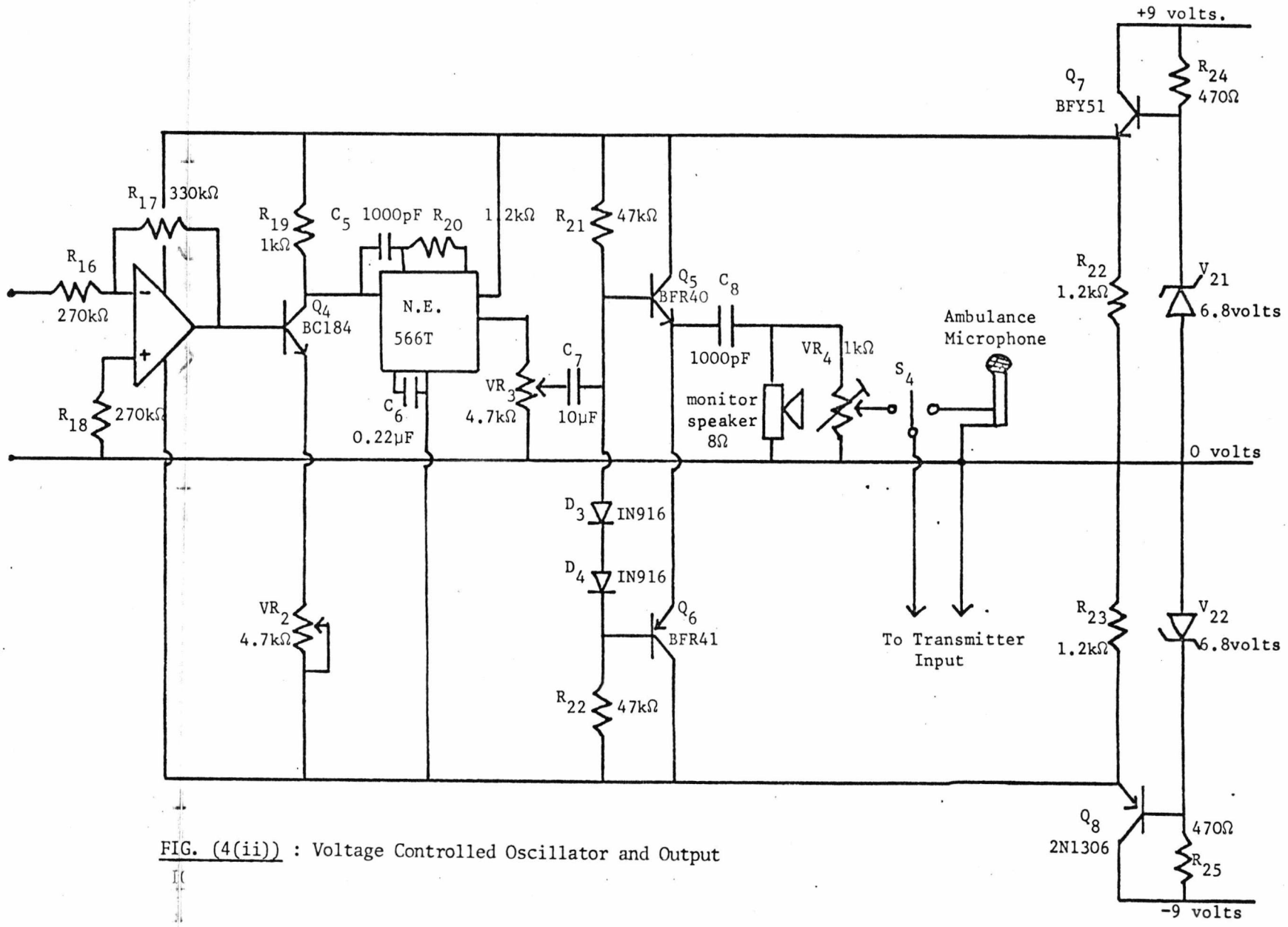


FIG. (4(ii)) : Voltage Controlled Oscillator and Output

A simple push-pull amplifier Q_5,Q_6 is used to drive a small loudspeaker as an audible indication of the output of the VCO, and the volume is simply controlled by VR_3. A portion of the output voltage across the speaker is selected by VR_4 (a few mV) and is sent to the transreceiver via S_4 which selects either the ECG or voice transmission mode.

4.3c The Decoder *(Fig. (5))*

The original decoder circuit was designed to have a microphone for detecting the modulated audio-tone from the loudspeaker of the transreceiver in the CCU. The microphone had an impedance of 600Ω and so the input resistor R_4 of A_2 *(Fig. (5))* was chosen to be 560Ω for best matching. Subsequent testing showed that this idea was inadequate since a lot of spurious background noise from the surrounding area was detected e.g. doors closing, which led to considerable interference in the ECG output. A buffer amplifier A_1 was then added to enable direct connection to be made to the output of the transreceiver. Both A_1 and A_2 are standard feedback operational amplifier circuits, and to avoid having offset trimming controls with the possibilities of drifting, the output of A_2 was a.c. coupled to A_3. When A_1 was added to the circuit, its gain could not be too high since any d.c. offset voltage produced at the output is then multiplied by the gain of A_2 and so saturation of the latter's output signals could occur. Another amplifier A_3 feeds a level detector A_4, which squares up the signal for reliable triggering of the following monostable. The level detector A_4 is connected in the open loop configuration, and

each operational amplifier has a ±9 volt supply, the connections for which have been omitted for clarity.

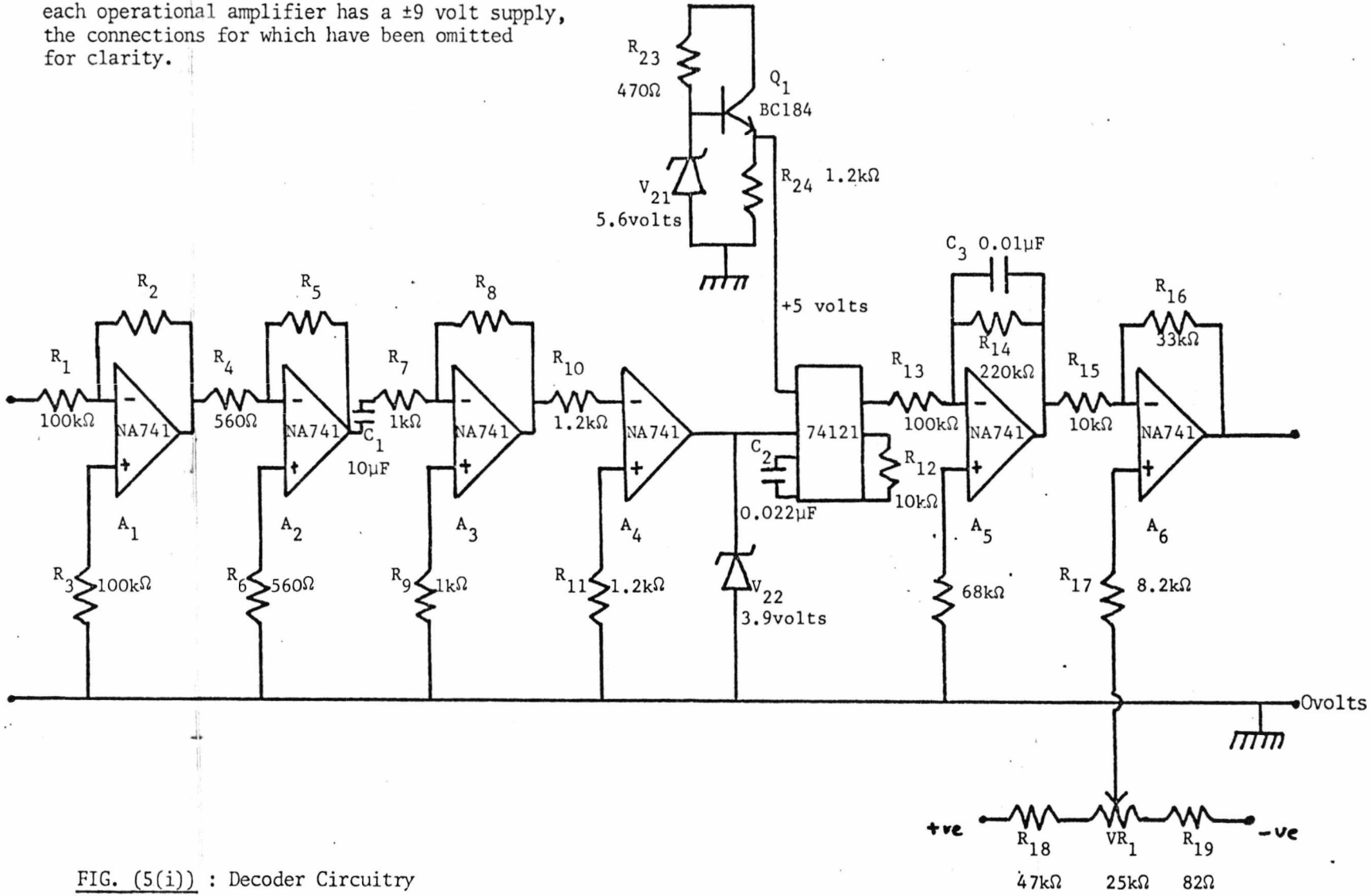


FIG. (5(i)) : Decoder Circuitry

This amplifier has a ±9 volt supply which has been omitted for clarity.

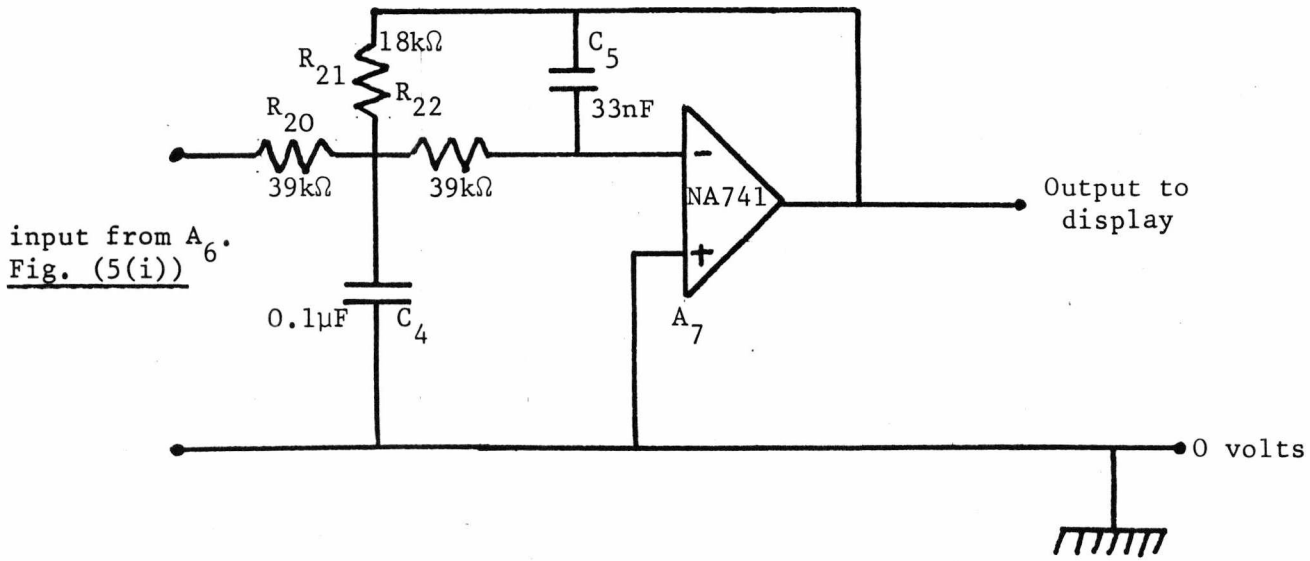


FIG. (5(ii))

Decoder Circuit - Final Filter

its output would swing from one supply rail to the other when driven but for the zener diode across the output, which clamps it to values compatible with the input requirements of the TTL monostable MI. This monostable produces pulses of constant height and width and only the rate is varying with the frequency of the input to the decoder. These pulses are filtered by the circuit action of A_5 (a low pass filter) the output of which is a voltage whose mean d.c. level is proportional to the pulse frequency.

(Fourier analysis of a train of pulses of width τ, and interval T, and height A, yields a frequency spectrum given by[9] *(Fig. (6))*,

$$V(t) = \frac{A\tau}{T} + \frac{2A\tau}{T} \sum_{1}^{\infty} \frac{\sin \frac{n\omega\tau}{2}}{\frac{n\omega\tau}{2}} \cdot \cos n\omega t \ .$$

The d.c. component $\frac{A\tau}{T}$ can be seen to be proportional to the frequency of the pulses ($f = \frac{1}{T}$), and the rest of the components of the signal are removed by the low pass filter A_5).

This is passed to A_6 which provides some amplification for the decoded signal, and also contains an output voltage shift control. This provides a d.c. voltage via R_{18},R_{19} and VR_1 which is opposite in polarity to the output voltage from A_5. When there is no ECG, the pulse frequency is constant at 2 kc/s, and VR_1 is adjusted so that the output of A_6 is zero volts. An active low pass filter A_7 removes any high frequency ripple that may be left in the output, and after this the ECG output is taken to a visual display and strip recorder.

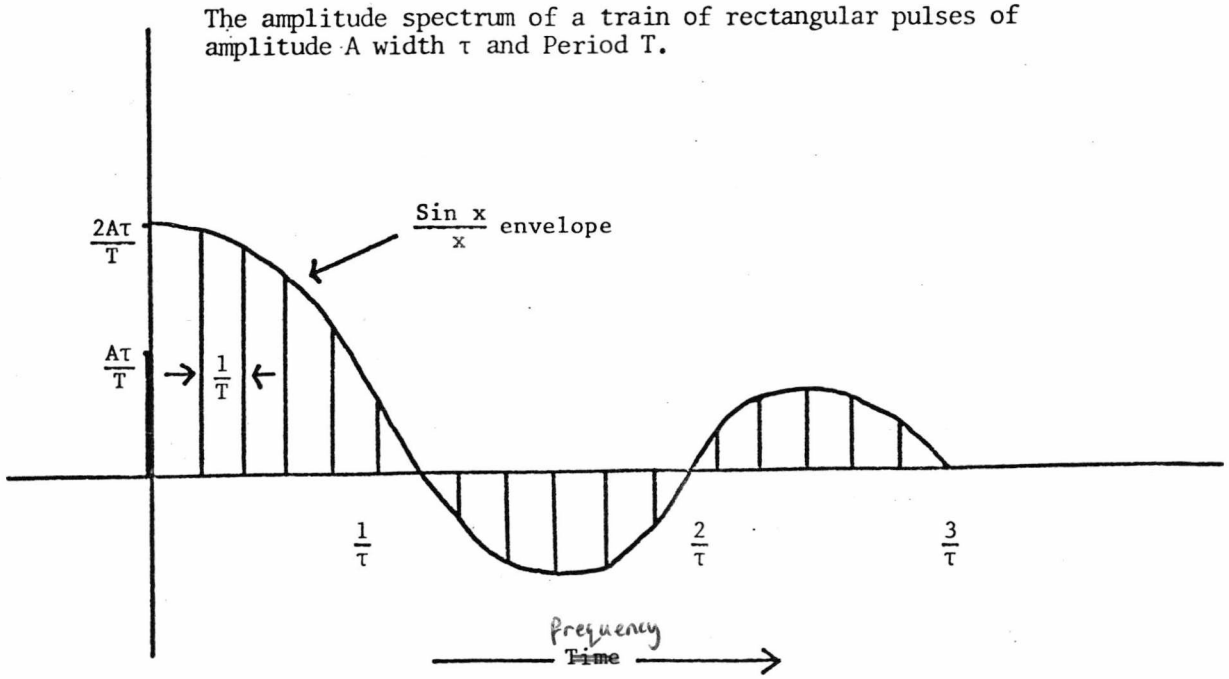

The amplitude spectrum of a train of rectangular pulses of amplitude A width τ and Period T.
2Aτ/T
Aτ/T
1/T
Sin x / x envelope
1/τ
2/τ
3/τ
Frequency
Time

FIG. (6)

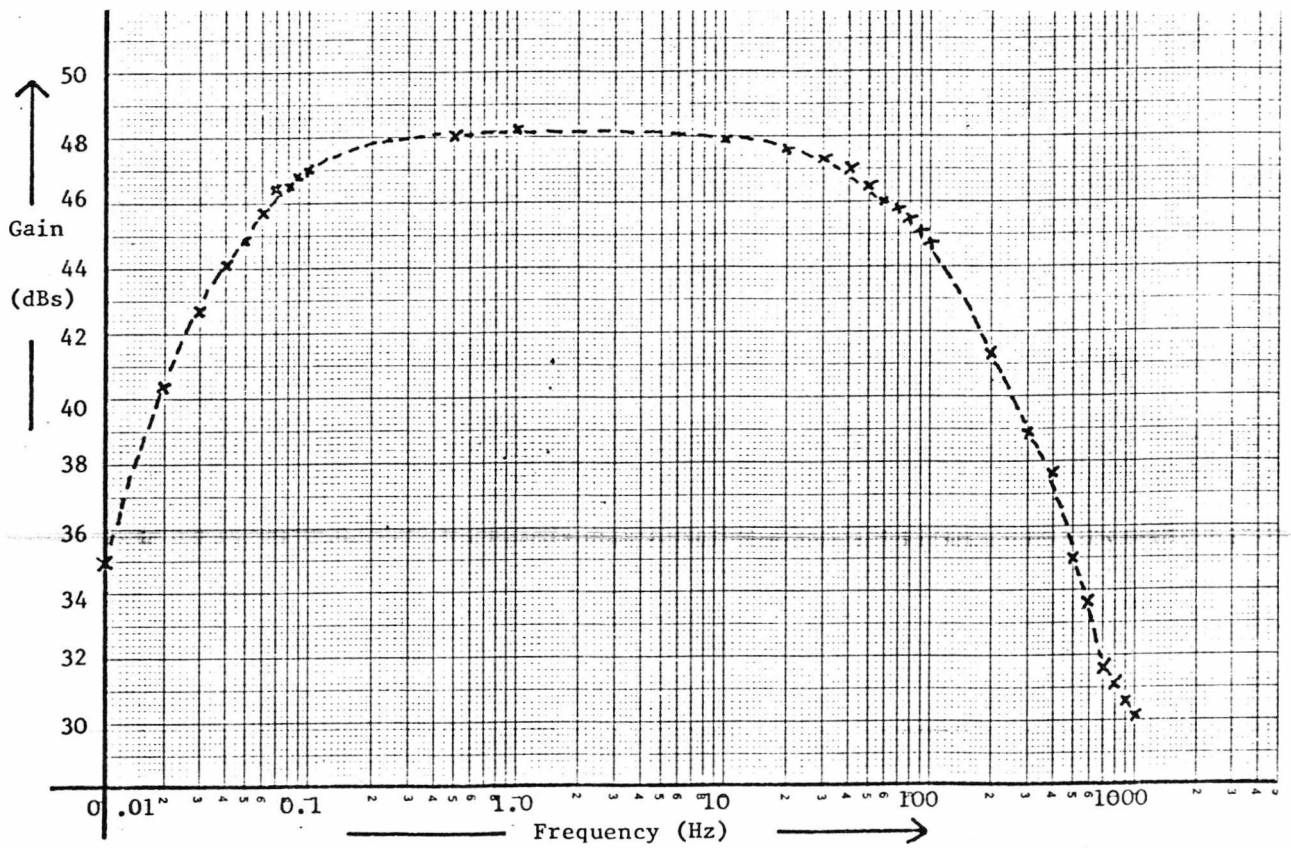

Gain
(dBs)
50
48
46
44
42
40
38
36
34
32
30
0
.01
0.1
1.0
10
100
1000
Frequency (Hz)

FIG. (7)

4.3d Circuit Performances

The circuits were built, and the individual sections were tested.

The gain of the ECG preamplifier of *Fig. (4(i))* was 250, and its bandwidth was from 0.05 Hz to 90 Hz *(Fig. (7))*. Its common mode rejection was found to be 600:1 (56 dBs) when using two separate TIS 88 F.E.T.s and 2000:1 (66 dBs) when a dual matched pair (2N 5045) was used.

The response of the voltage to frequency converter is as in *Fig. (8)* and is linear over the input voltage range to it of ±1 volt. This corresponds to an input to the ECG pre-amplifier of ±4mV which is within the specifications stated in section 4.2.

The frequency to voltage characteristic of the decoder is plotted in *Fig. (9)* and is linear over the operating range of frequencies, (1 KHz to 3 KHz). The threshold voltage for reliable operation is 2.5mV pk to pk at the input, and the dynamic range is 1000:1 *(Fig. (10))*. Below 2.5mV the gain of amplifiers A_1,A_2 and A_3, was not enough to drive the squarer and monostable consistently. The frequency response of the final low pass filter (A_7) is shown in *Fig. (11)* and has an upper 3dB point of 110 Hz.

A bench test was performed by driving the coder with a signal from an ECG simulator, and a microphone was placed over the loudspeaker to provide an input to the decoder. The results of this can be seen in *Fig. (12)* where the input signal (~~top~~ bottom trace) is compared with the decoder output signal (~~bottom~~ top trace). This was repeated for three sine wave inputs

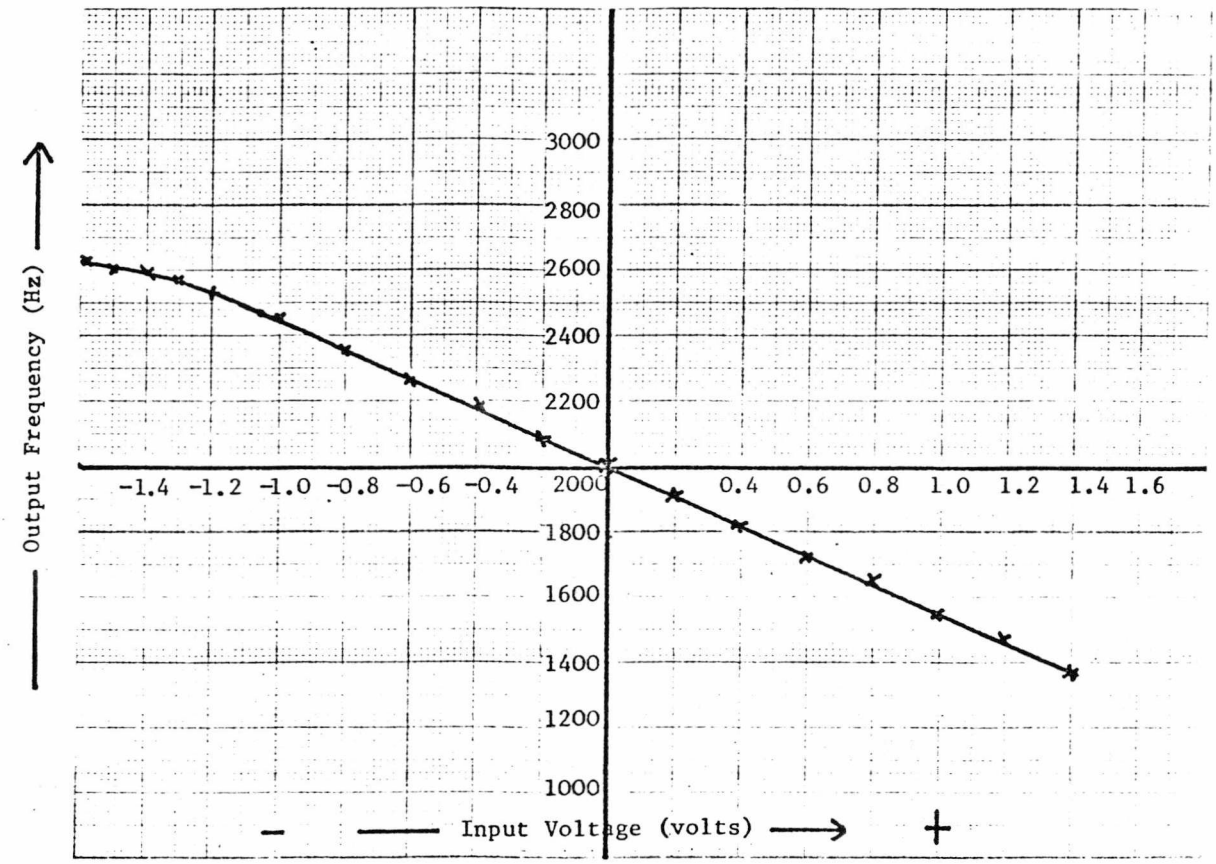


FIG. (8) : Voltage to Frequency Response of Encoder

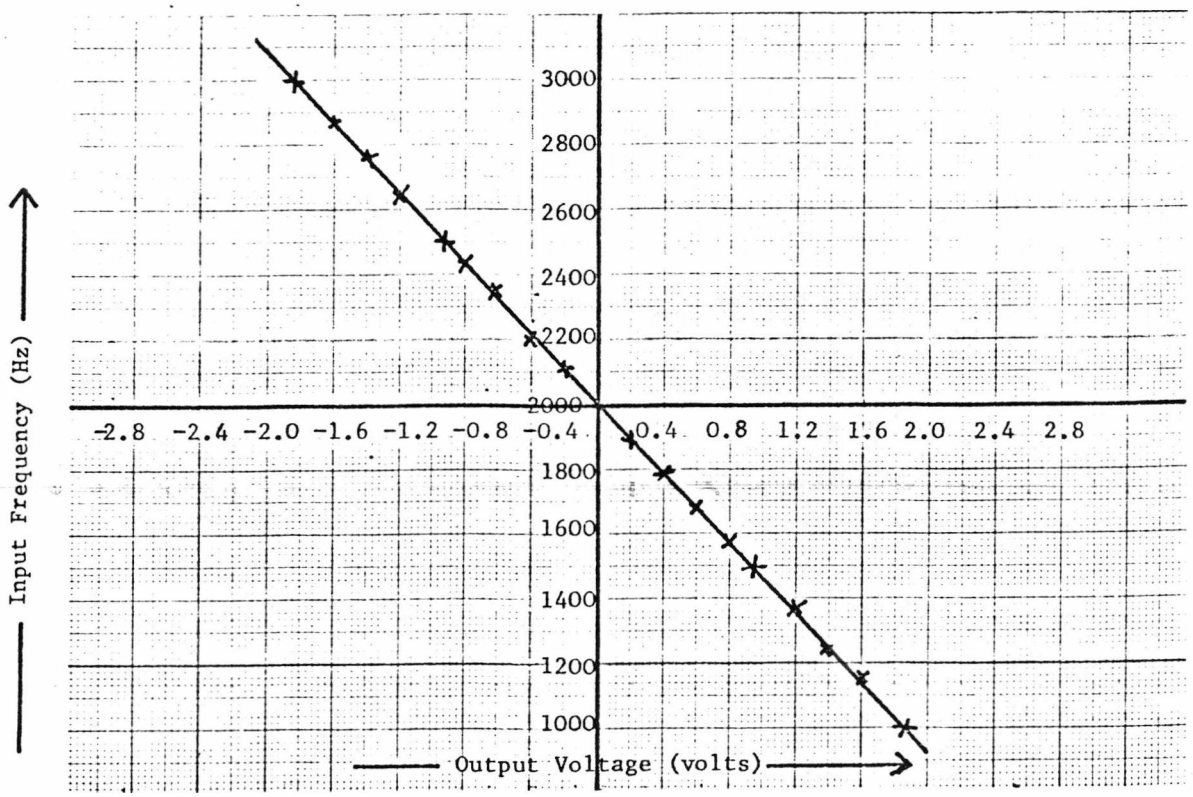


FIG. (9) : Frequency to Voltage Response of Decoder

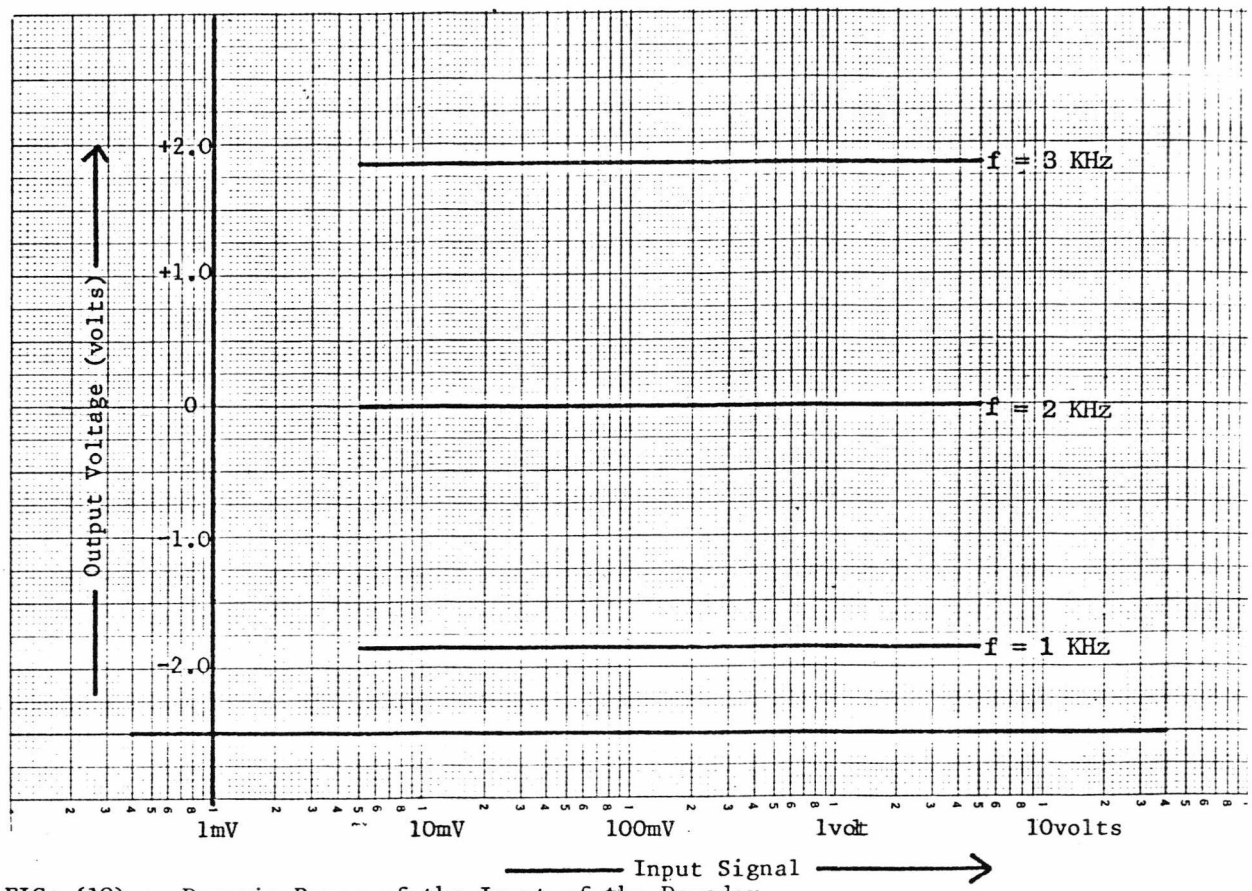


FIG. (10) : Dynamic Range of the Input of the Decoder

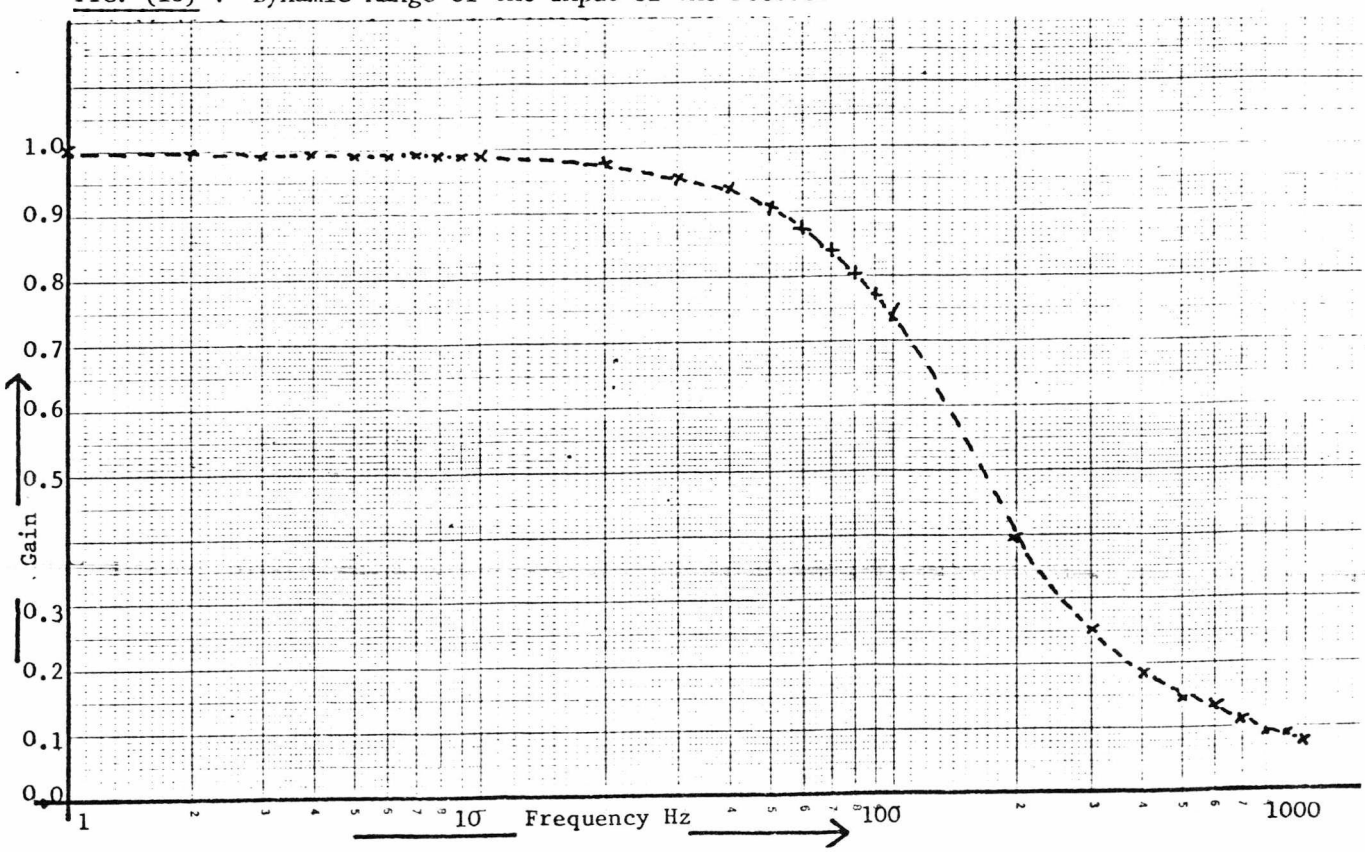
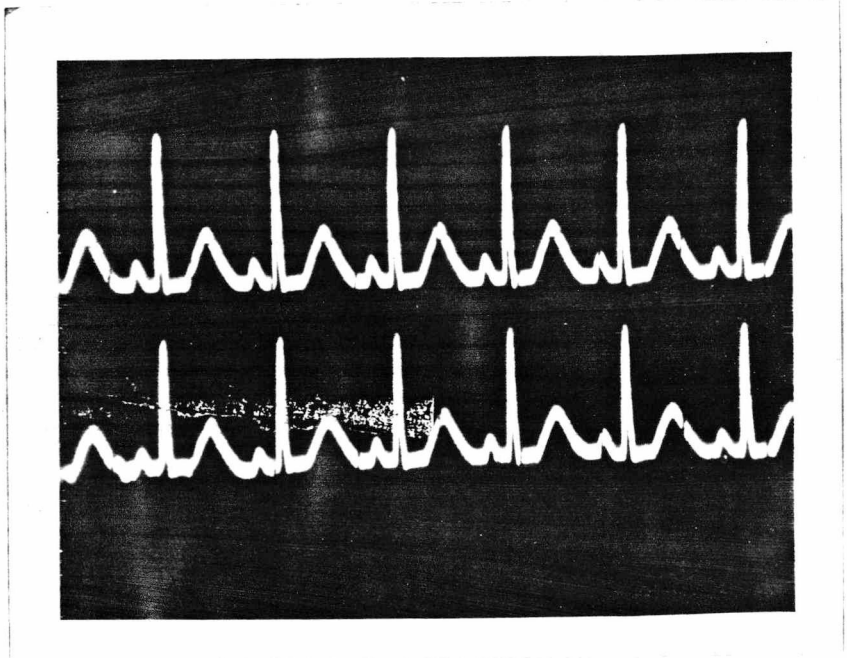


FIG. (11) : Frequency Response of the Decoder Filter

Top Trace

Bottom Trace



0.2volts/div ↑

0.2 secs/div →

2mV/div ↑

0.2 secs/div →

The received signal

The transmitted signal

FIG. 12. The e.c.g. transmitted from encoder to decoder directly

of 1, 10 and 100 Hz and the results can be seen in *Figs. (13), (14)* and *(15)*. A final test was the transmission of a simulated ECG over an internal telephone link in the Hospital, and *Fig. (16)* is the result of this.

These tests showed that the electronic circuits were working, and the next stage is the actual testing of the transmission of ECGs over a radio network.

4.4 TESTING THE WHOLE SYSTEM

The radio systems used by the various Ambulance Services fall into two main categories, described by the method used for modulation of the carrier wave - either frequency modulated (F.M.) or amplitude modulated (A.M.). An examination of the two different systems[10],[11] shows that F.M. is the best to have since it has much lower noise levels than A.M. At present the local ambulances are fitted with A.M. radios, but it is planned to change to F.M. transreceivers as soon as possible. Accordingly, the tests of the ECG telemetry system were carried out on an F.M. radio. The network used belonged to the Electronics Laboratories with the base station on the campus operating at 156 MHz, and the mobile unit was in the Laboratories' land-rover.

The "patient" sat in the front of the land-rover and was connected up to the ECG encoder and a portable ECG writer. This was to enable a permanent record to be kept, of the ECG being transmitted for comparison with that received by the base station. At first, a live person was used as a signal source, but due to the cramped conditions of the vehicle it

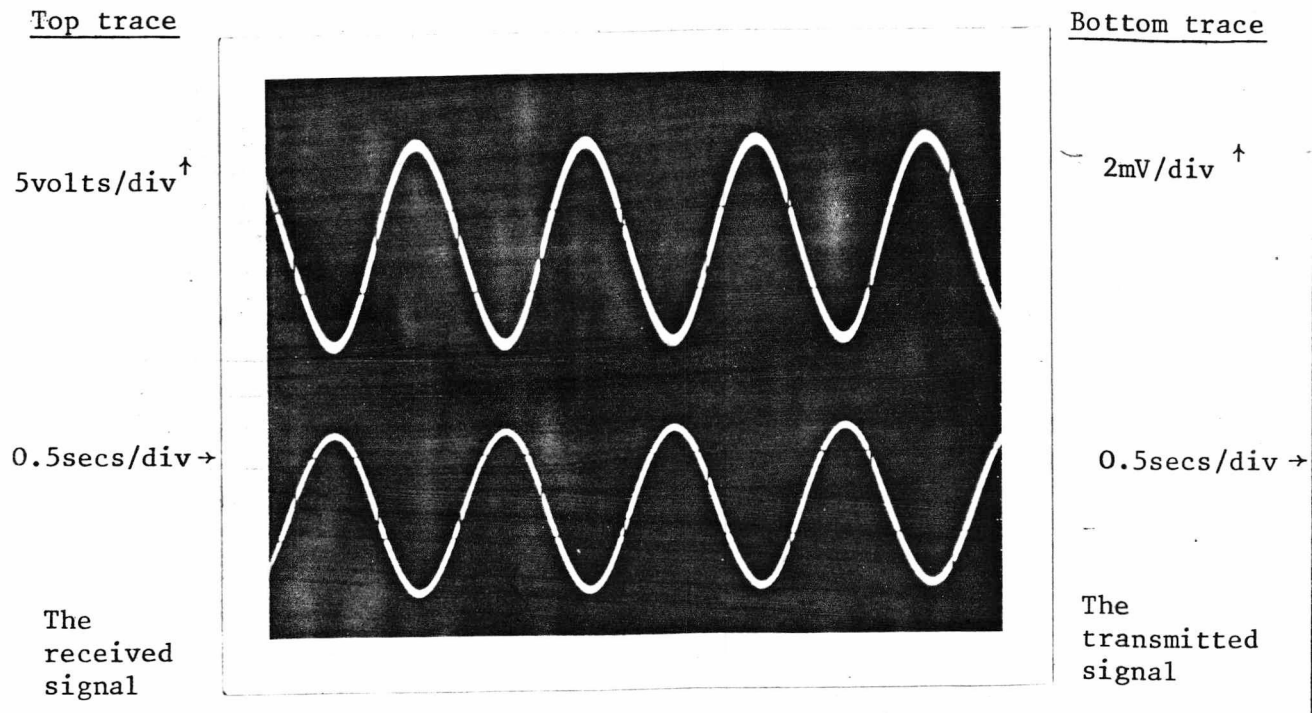


FIG. 13 A 1Hz signal

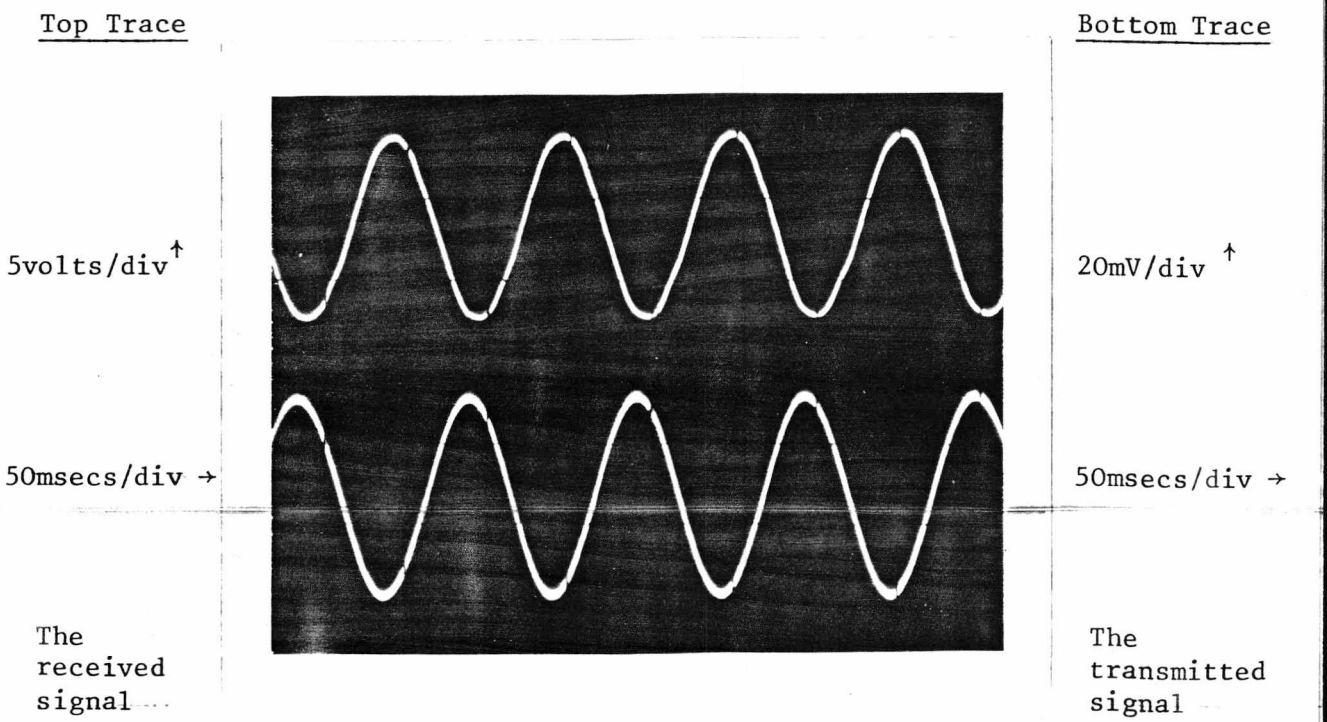


FIG. 14 A 10Hz signal

The position of these diagrams should be reversed.

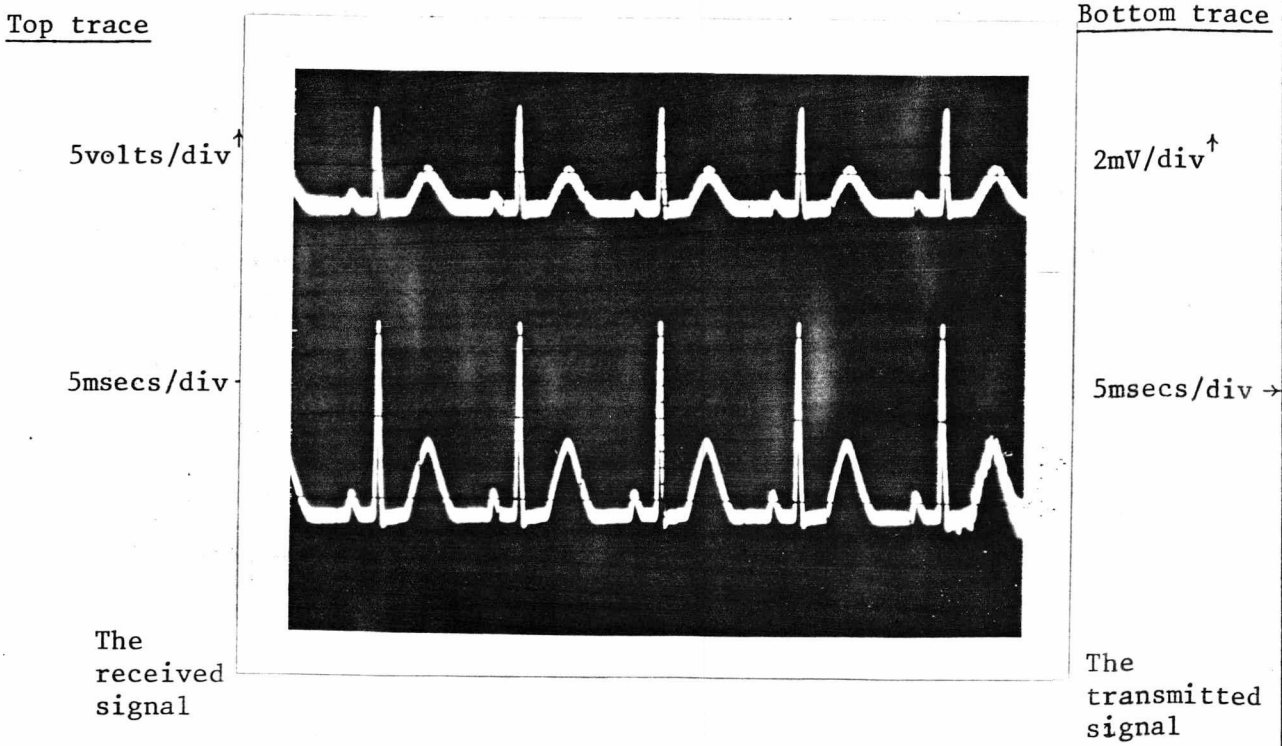


FIG. 15 A 100Hz signal

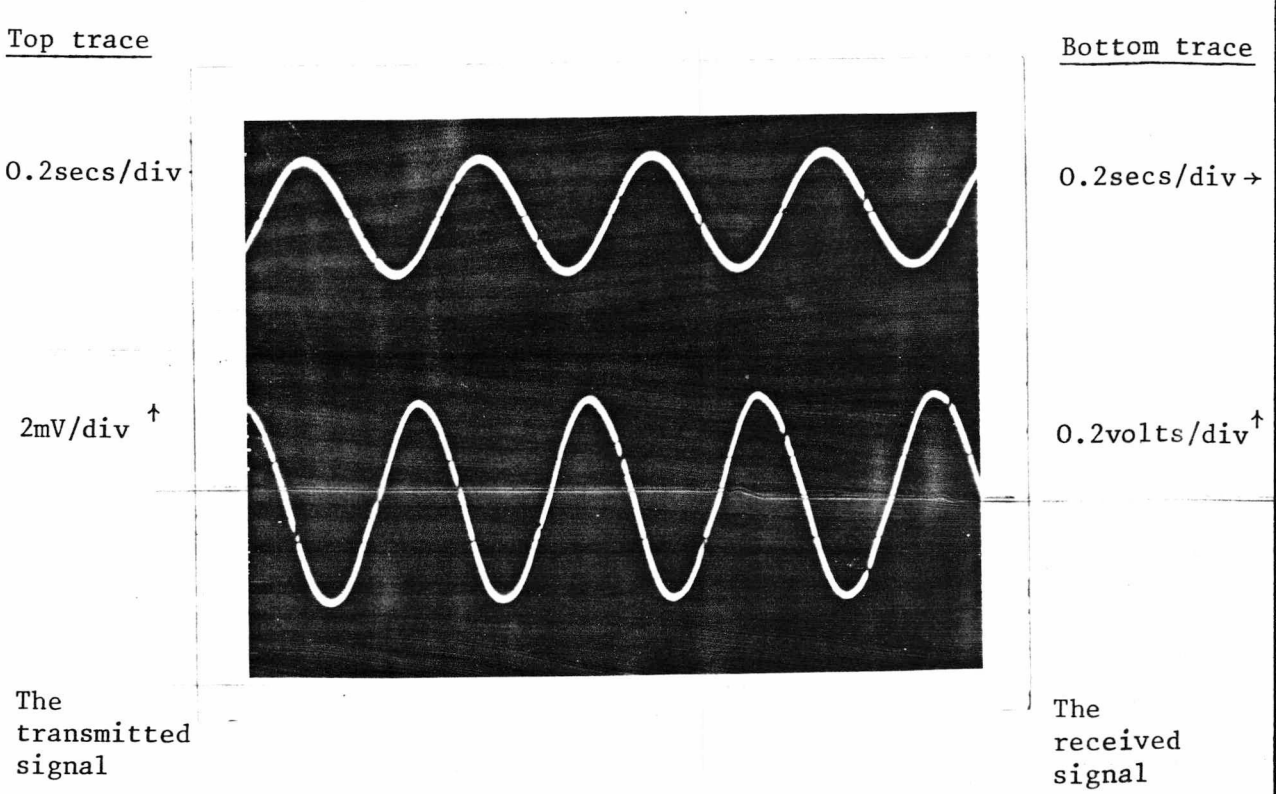


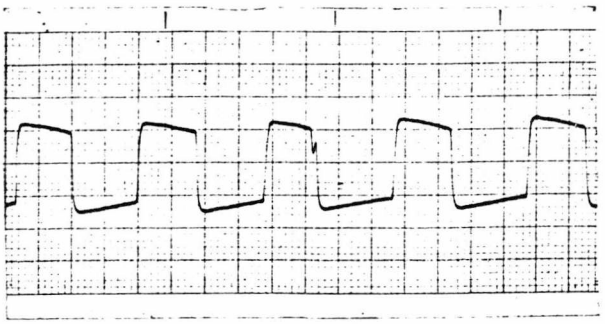
FIG. 16. The e.c.g. transmitted over a telephone link.

proved impossible to obtain a good ECG as so much muscle tremor was present *(Fig. (17))*. Thus, an ECG simulator was used instead, and this was quite satisfactory since it gave a nice, stable baseline for comparison purposes. At the University, the decoder was connected directly across the loudspeaker terminals of the receiver, and the ECG output went to an oscilloscope display and another ECG writer, for the recording of the received signal.

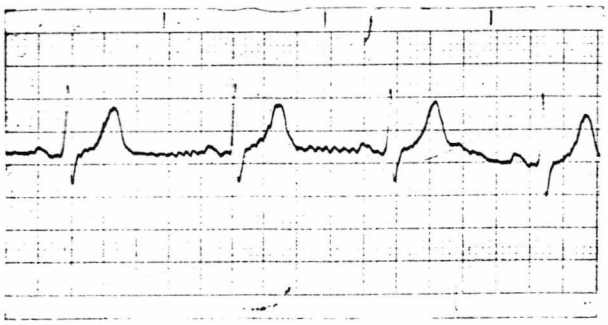
The testing consisted of the land-rover driving away from the University, and at certain distances stopping to transmit ECG signals. When in Ramsgate, which was 14½ miles away, the land-rover made some transmissions whilst starting the engine, and driving down the road.

4.4a Results

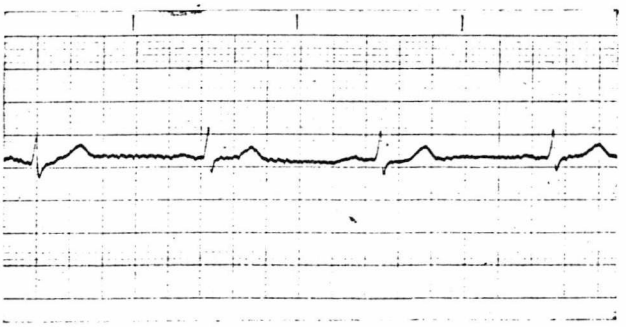
Fig. (17) shows the results when a live subject was used. There is considerable artefact on the baseline of both the transmitted and the received trace which makes any assessment difficult. The noise on the baseline is of the same order of magnitude as the 'P' wave of the ECG approximately 0.1mV. *Figs. (18), (19)* and *(20)* are some of the results of the second set of experiments and the baselines can be seen to be much steadier. Each of these pairs of results is from a different distance. It can be seen that there is no difference in quality between 20 yds and 14½ miles, since there seems to be no degradation of the baseline or signal complexes in any of the results. *Fig. (20)* is what happens when the engine is started and *Fig. (22)* is the transmission from the moving



Decoded
Calibration Signal

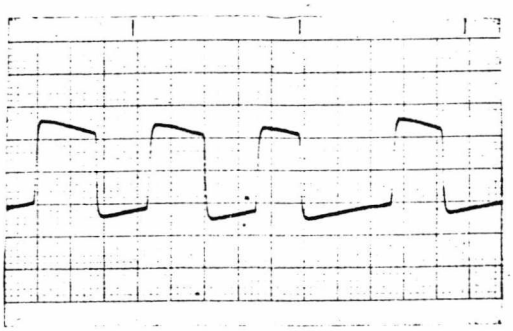


Decoded at University

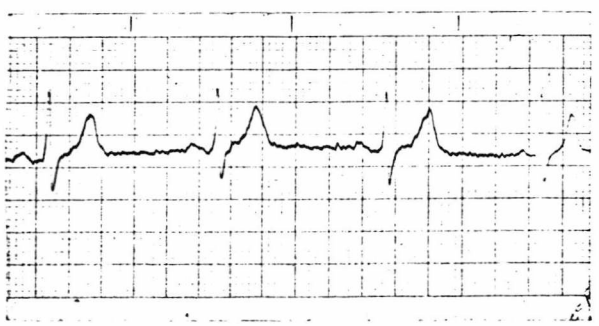


E.C.G. recorded in the Land Rover

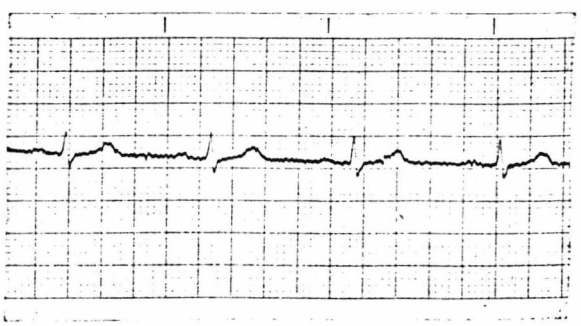
FIG. 17(i) Live Patient Transmissions. Range = 11 miles



Decoded Calibration Signal



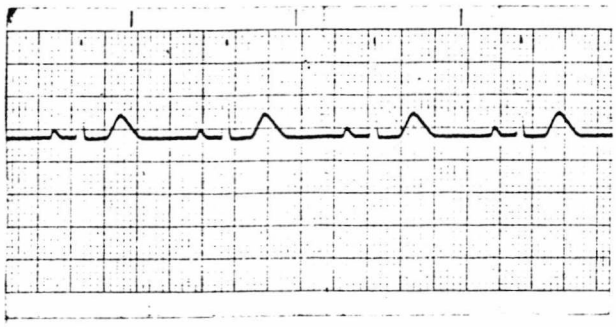
Decoded at University



E.C.G. rcorded in the Land Rover

FIG. 17(ii) Live Patient Transmission. Range = $3\frac{1}{2}$ miles

TRACE RECORDED IN LAND ROVER



DECODED AT UNIVERSITY

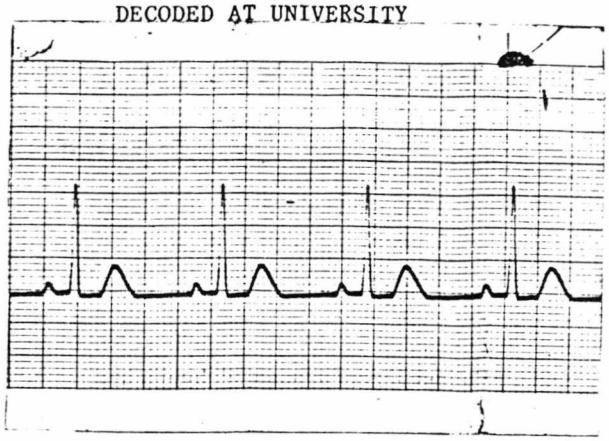
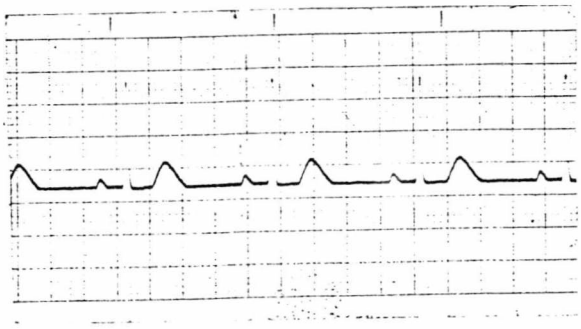


FIG. 18(i) Range = 200 yards



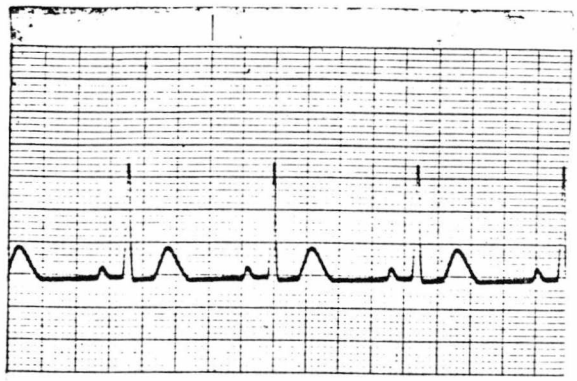
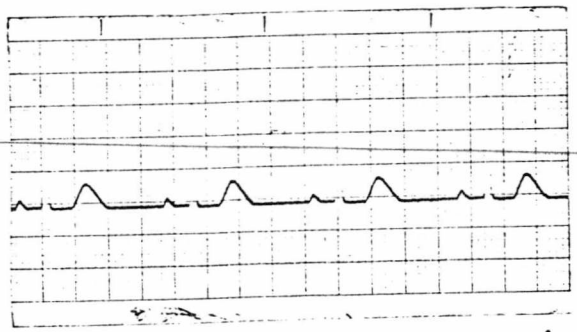


FIG. 18(ii) Range = $3\frac{1}{2}$ miles



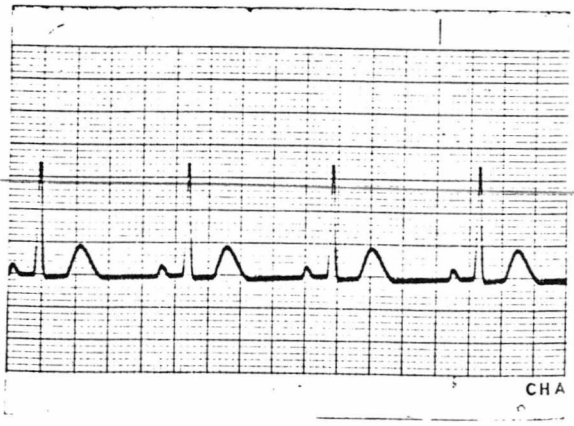
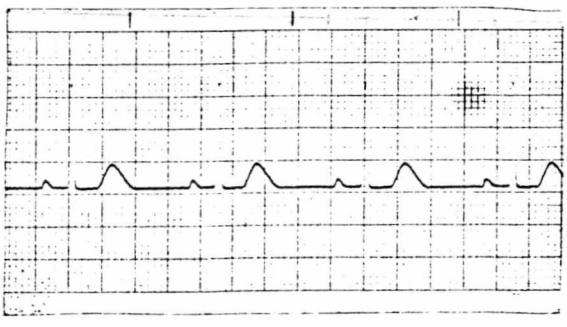


FIG. 18(iii) Range = 6 miles



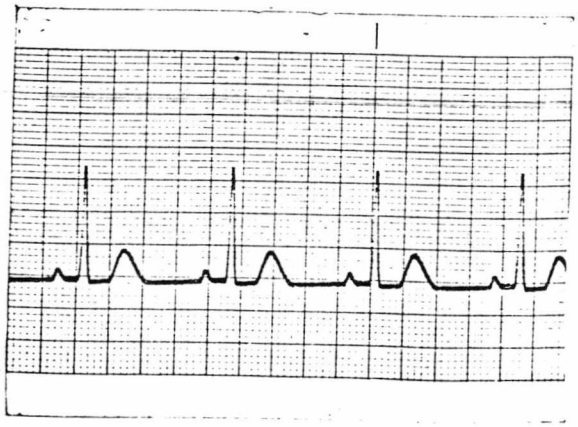


FIG. 18(iv) Range = $8\frac{1}{2}$ miles

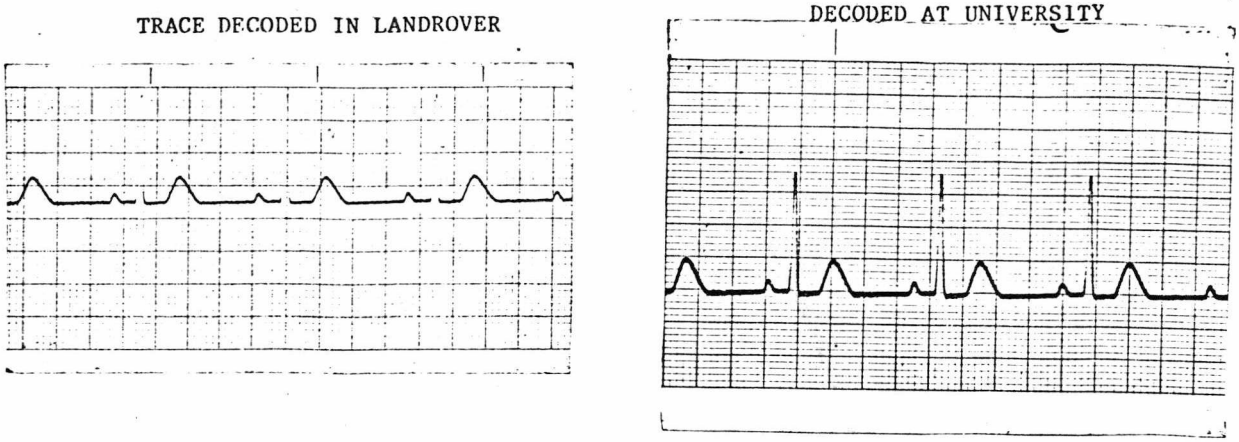


FIG. 19(i) Range = 12 miles

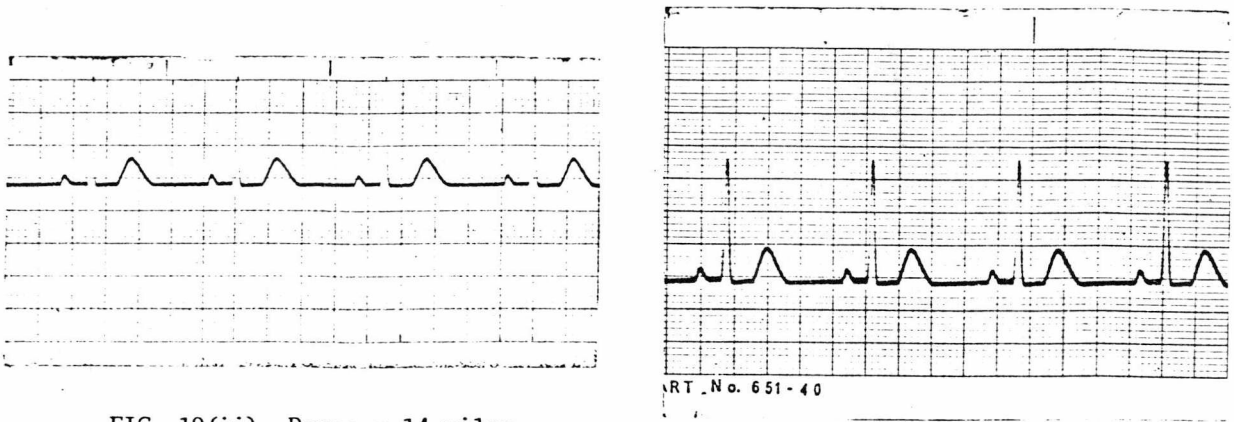


FIG. 19(ii) Range = 14 miles

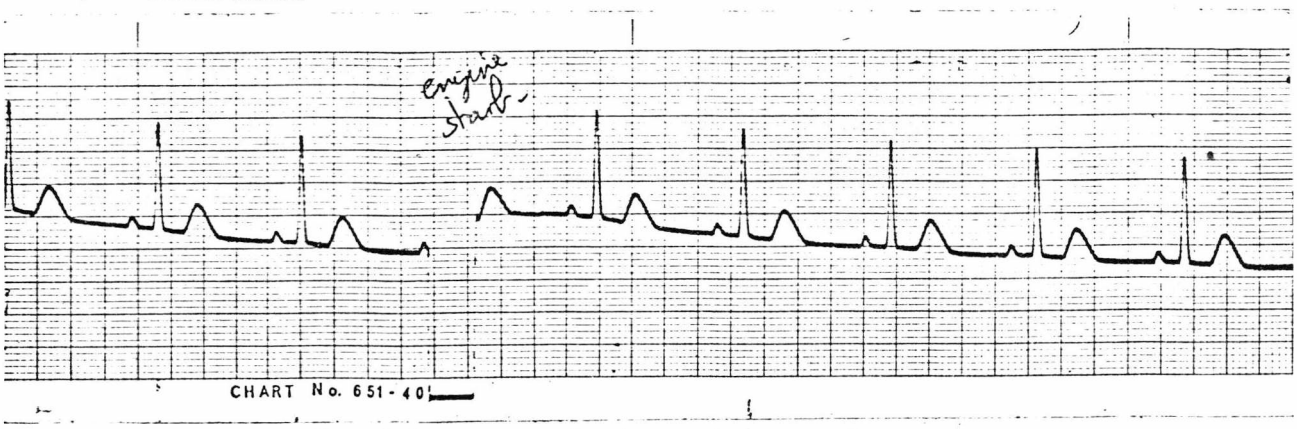


FIG. 20 Decoded trace received at University when engine starting and continued running. Range = 14½ miles.

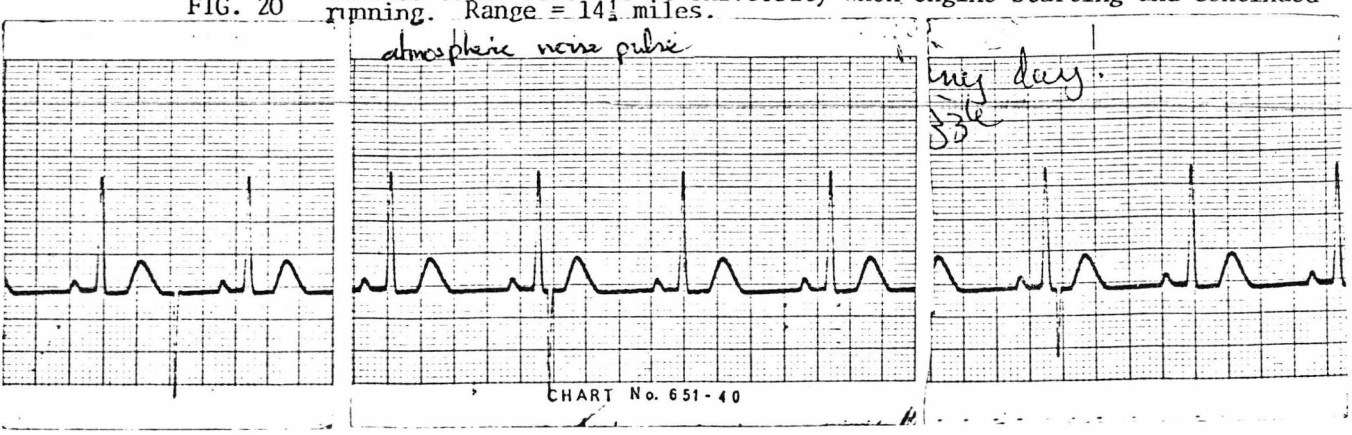


FIG. 21 Effect of various atmospheric "clicks" upon decoded trace.

vehicle a little later on. The last two results were both from Ramsgate. *Fig. (21)* shows some recordings of the interference caused by loud atmospheric 'clicks' which were heard from the receiver's loudspeaker at different times.

The drop out in *Fig. (20)* was when the engine started, and this is due to the starter motor reducing the battery voltage of the vehicle (usually to about 7 volts instead of 12), and so the transmitter stopped working. Once the engine had started, the transmitter came on, and no interference can be seen after this point. *Fig. (22)* shows what happens when the vehicle was driven further down the road. There is no noticeable interference until about halfway, and then several drop outs occurred. When the driver tried to speak, however, he was unintelligible as well, with a great deal of noise and distortion. This went up and down as he passed houses, buses and lorries.

It was noticed that all the interference produced -ve pulses, not +ve going ones at all. This is believed to be a combination of the muting control on the receiver and the ECG decoder. When the signal became weak and noisy, the muting control cuts in and the receiver output falls thus the input to the decoder disappears, and so it's output falls, hence a -ve pulse.

4.5 CONCLUSIONS

From these results it was concluded that the telemetry of ECGs is a feasible proposition. The ECG recordings obtained were of good quality irrespective of the transmitted distance

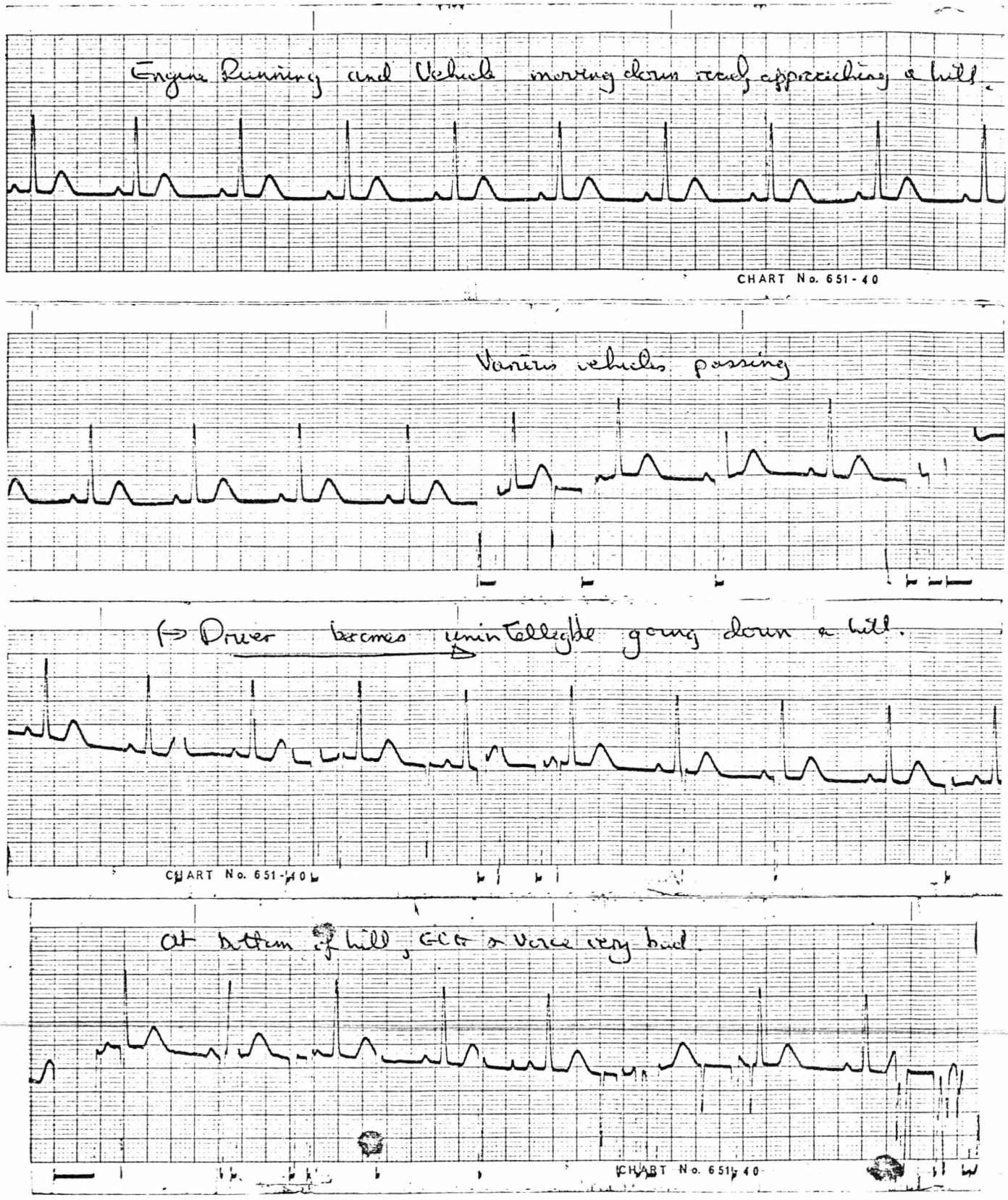


FIG. 22 This is a continuous recording, as received at the University, with the Land Rover driving along the road. Range = $14\frac{1}{2}$ miles

(compare *Figs. (18(i)* and *(19(ii))*). When degradation did occur (as in *Fig. (22)*) it was accompanied by loss of voice transmission as well, and these results indicate that the ECG depends upon the state of the transmission link rather than upon the coding and decoding apparatus. This is supported by the results of the laboratory tests as seen in *Figs. (12), (13), (14), (15)* and *(16)*. The beginning of *Fig. (22)* shows that quality ECGs can be received from a moving vehicle as well as a stationary one. When the ambulance service is equipped with its new F.M. radios, then they will be powerful enough to give good coverage over all the East Kent Area, and will enable high standard ECGs to be transmitted.

This means that MCCU's operating without a doctor, and relying upon telemetry for diagnosis of the patient's condition, ~~is~~ are technically feasible.

REFERENCES

(1) REPORT OF THE COMMITTEE OF ELECTROCARDIOGRAPHY - AMERICAN HEAT ASSOCIATION 1967 "Recommendations for standardisation of leads and of specifications for instruments in electrocardiography and vectorcardiography" IEEE Transactions in Bio Medical Engineering, Vol. BME 14, pp. 60-68. Jan. 1967.

(2) C.L. FELDMAN et al "Digital filtering of the electrocardiogram" 1971. Proceedings Conf., Computers for Analysis and Control in Medical & Biological Research, p. 50, Sheffield.

(3) J.M. NEILSON "A special purpose hybrid computer for analysis of ECG arrythmias" 1971. Proceedings (Conf.) on Computers for Analysis & Control in Medical & Biological Research, p. 151, Sheffield.

(4) R.E. MORRIS, S. LITMAN & D. GROOM "Energy spectrum of the cardiac cycle" 1966. Proceedings 19th Annual Conf. Engineering in Medicine & Biology.

(5) P. LANGNER, D.B. GESELOWITZ & F.T. MANSURE "High frequency components in the electrocardiograms of normal subjects and of patients with coronary heart disease" 1964. American Heart Journal, Vol. 62, pp. 746-753.

(6) O.P. LANGNER & J.A. LAUER "The relative significance of high frequency notching in the electrocardiogram" 1966. American Heart Journal, Vol. 71, pp. 34-42.

(7) W.D. BUSSMANN et al "On-line analysis of ECG arrythmias with a digital computer" 1975. Medical & Biological Engineering, Vol. 13, No. 3, p. 382.

(8) P.W. McFARLANE & T.D.V. LAWRIE "An introduction to automated electrocardiogram interpretation" 1974. Published by Butterworths, p. 26.

(9) J. BROWN & E.V.D. GLAZIER "Telecommunications" 1966. Chapman & Hall Ltd. (Publishers), Chapter 2.

(10) J. BROWN & E.V.D. GLAZIER "Telecommunications" 1966. Chapman & Hall, Chapter 6.

(11) A.M. ROSIE "Information and Communication theory" 1966. Publishers Blackie & Son, Chapter 7.

CHAPTER 5

WARD AND EXERCISE TELEMETRY

5.1 MONITORING IN THE ITU

The previous work illustrated how electronics can be utilised to enable people suffering from acute ischaemic heart disease to have a much better chance of reaching hospital alive.

When they arrive in the Intensive Care Unit (ITU) or Coronary Care Unit (CCU) a variety of instruments, machines and therapies are available for use in sustaining the patient and overcoming any arrythmias that may have arisen. The ones that are used depend upon the clinician's assessment of the situation and particular requirements of the patient. One instrument that is essential is the ECG machine, since it is easy to collect the signals, and as was shown in Chapter 2, can provide extensive information to the trained observer for the diagnosis and prognosis of the patient's condition.

The ECG is monitored continuously, and in most machines facilities are available to display the patient's heart rate with alarms included to sound if the rate goes outside certain limits as defined by the physician. In a survey by Rawles & Crockett[1], it was shown that a lot of inconvenience arises with the use of the alarm systems. Of the alarms sounded during the survey 90% were false, with the principle causes of these being the electrodes and the leads. Often the leads pulled away from the electrodes, or the wires were broken in the cables or the electrodes were pulled off the body surface or badly disturbed by movement of the leads. This tendency

has been confirmed by Jewitt et al[2] and Pentecost and Mayne[3] both of whom state that the rate alarms are seldom used due to their unreliability. When the ECG cables, of necessity must be as long as they are, and because the patient has to be moved regularly to avoid bedsores, and when several people at a time may be in attendance around the bed, then these problems are almost inevitable. This chapter investigates the possibilities of developing a telemetry system for recording the patient's ECG to overcome these problems as short electrode leads can be used, with the transmitter strapped to the patient's chest.

Other dangerous situations can arise when using normal cable ECG monitoring. One of them is the possibility of electric shock if several other pieces of equipment are also connected to the patient. Pocock[4] has shown how this can happen. If a fault occurs in the earth wire of the ECG machine then it is possible for leakage currents to find a path through the patient and out via another machine. In the case of the other machine being an intra cardiac catheter, then currents in excess of the danger levels (10μA rms[5]) can easily flow through the myocardium and cause fibrillation *(Fig. (1))*. Although this is unlikely to happen, the more mains powered equipment used the greater the dangers become. Telemetry of the ECG can help in this situation as a battery powered transmitter does not have an earth line - or a mains input. Thus, it cannot generate or pass leakage currents.

Kornfield[6],[7] has claimed that in the U.S. all the equipment that is connected to a patient can have deletirious psychological effects. He found that patients in the recovery room often showed severe psychotic-like symptoms, and that those in ITU's and CCU's found that the equipment prevented sleep and caused irritation and worry. In studies in the

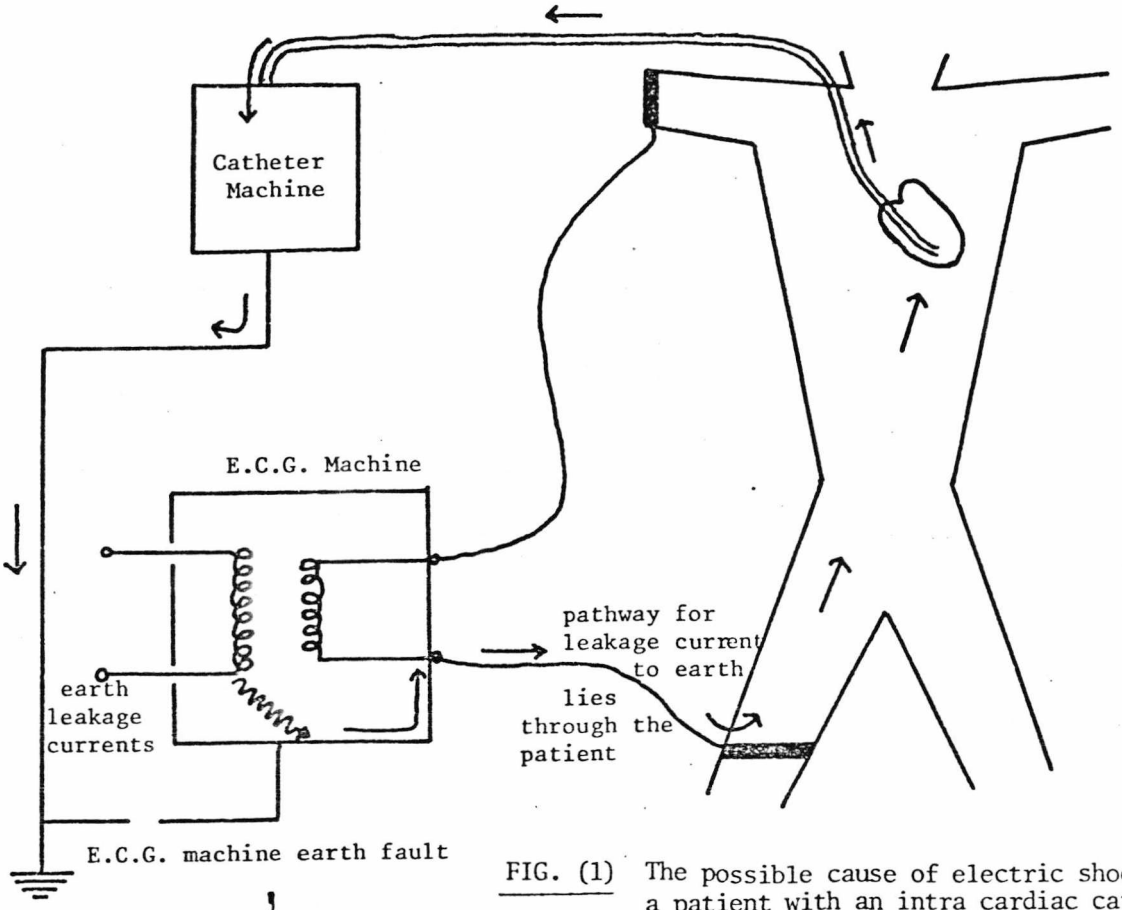


FIG. (1) The possible cause of electric shock to a patient with an intra cardiac catheter

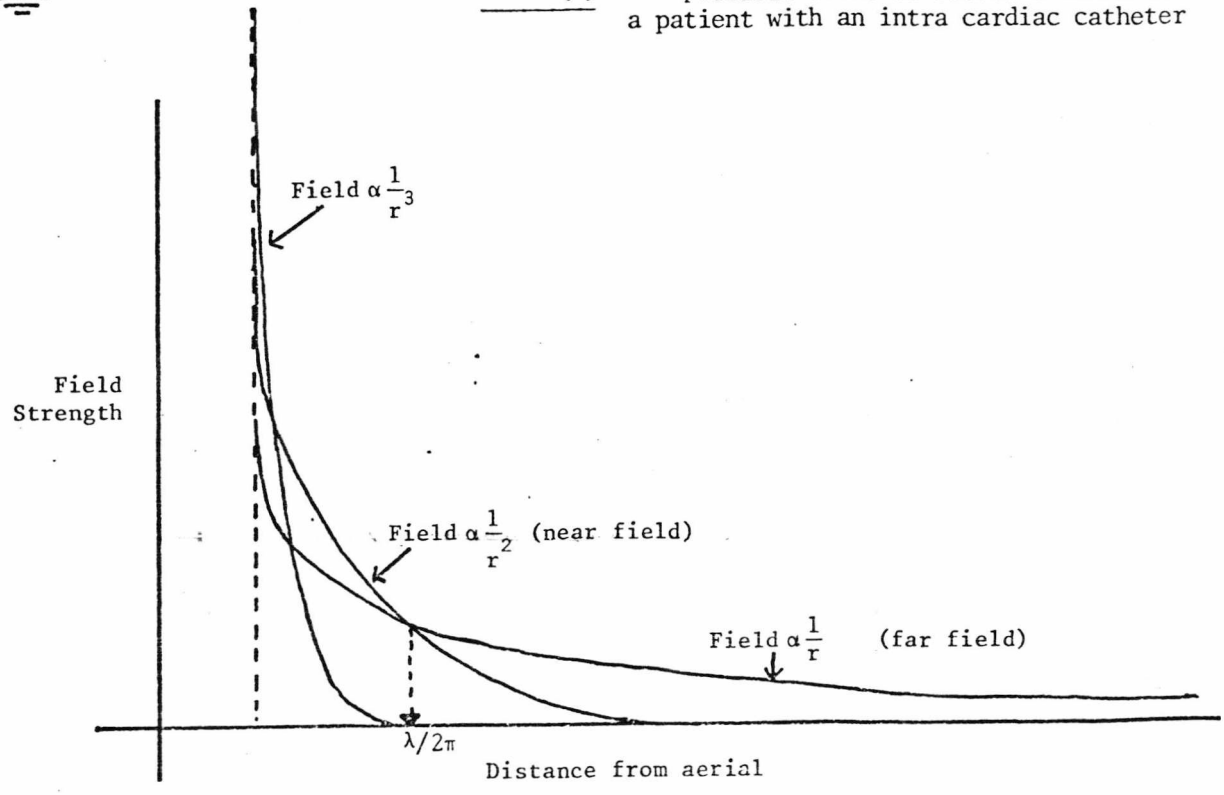


FIG. (2) An illustration of how the field strengths change with distance from a Hertzian Dipole

U.K.[8],[9] it has been found that the same symptoms do not appear to exist - most people are unworried by the equipment - some indeed derived comfort from its presence. It was agreed, however, that problems can arise when the patient leaves the I.T.U. for the general wards. Removal from the equipment can cause anxiety during this phase of recovery. Telemetry could assist in several ways. by being reasonably small and unobtrusive so that the patient may be alarmed less by this, than by a set of long trailing leads, and also, as recommended by the previous studies[6],[8], the monitors can be placed out of sight of the patient. Also during the critical phase of first mobility and transfer to a general ward, it is possible to monitor the patient's ECG - a benefit to the clinical staff, and possibly to the patient with anxiety as he would still feel the presence of monitoring equipment.

After the patients' recovery it is necessary to establish how much work they are able to do upon going home - or whether they must rest in bed again for a further few days. One method of doing this is by using an exercise bicycle. Patients pedal against a variable load, and their ability to work against different loads indicates to the physician their capabilities. During the activity the patients' ECG is monitored for the presence of arrythmias or significant shape changes in the ECG pattern. The present method of doing this with a conventional machine is difficult, due to the artefact that is produced when the patient's pedalling movements move the leads. It is possible that the use of a small transmitter strapped to the chest and using two short signal leads can overcome the artefact problem, and enable more successful records of rate and rhythm to be obtained.

5.2 DESIGN CONSIDERATIONS

From discussions with the consultant staff of the I.T.U., it was decided that four beds would be required to have ECG monitoring by telemetry. The transmitters must be able to operate satisfactorily from any part of the unit and from a reasonable distance outside e.g. the toilets. A range of 60 feet from the central receiver was decided upon. The transmitter would have to reproduce a signal that was of the same quality as an ECG from a bedside monitor since it was to be used for diagnostic purposes; it was not sufficient to send signals for rate analysis alone. This determined the frequency response of the ECG preamplifier to be that recommended by the American Heart Association(Chapter 4(1)) of 0.05 Hz to 100 Hz. It would also need to be defibrillator proof, since its prime use was to be with patients most likely to need such assistance.

The battery life was required to be as long as possible preferably two weeks at least. This would mean that the - equipment could be left undisturbed on the patient, if necessary, throughout their stay in the I.T.U. It was also to be reasonably small and light to avoid undue patient discomfort from pressing heavy weights.

There was to be a single receiver at the nurses desk, with outputs to a four channel monitor, so that a nurse could watch all four patients at once.

Since the frequency bands allocated by the Post Office for medical use are limited, the transmitters would have to be stable in frequency - and this meant crystal control for those operating in the VHF band. In addition the four channel requirement and a single receiver for all, meant that each transmitter would have to contain its own subcarrier generator, to enable easy separation of the signals after reception.

There remained one more point to resolve- that of the exact frequency band to use. There are two main telemetry systems - those using the near field (induction), and those using the far field (radiation) of the carrier frequency. The difference between the two arises from the conditions that exist in an aerial when it is excited by a current. If we consider the smallest possible aerial - the Hertzian Dipole, then it can be shown[10],[11] that three types of field are produced upon excitation:-

(1) A field $\alpha \frac{1}{r^3}$ and a function of the charge on the aerial

(2) A field $\alpha \frac{1}{r^2}$ (induction field) and a function of the velocity of the charge

(3) A field $\alpha \frac{1}{r}$ (radiated field) and a function of the acceleration of the charge

The three are shown with their relative strengths in *Fig. (2)*, and it can be seen that for small distances ($< \lambda/2\pi$) the induced field is stronger than the radiated field although it is dropping off at a faster rate. One system, then, for ECG telemetry would be to use a low frequency transmitter of about 400 KHz and have the aerial around the bed. This would only be ~~alright~~ all right until the patient became mobile. To maintain reception during this latter period an aerial must be placed right around the room, but then the problem of reliability of reception occurs due to orientation effects between the transmitting and receiving aerials. Jacob[12] has investigated this, and shown that the further away from the receiver the transmitter is, then the more chance there is of losing the

signal (Table 1), when the transmitting and receiving aerials are randomly orientated to each other.

Distance from aerial in terms of its diameter of 120 mm.	Percentage probability of receiving a signal from a randomly oriented transmitter.
4.2	82%
3.2	91%
2.5	96%
1.0	97%

Table (1)

Another consideration is that in the I.T.U. at Canterbury it would have been difficult to install a complete loop aerial that would be needed, and also there was a lot of interference present due to power cable ducts in the wall to the kitchen below the unit. This became a considerable nuisance with some of the ECG machines (at times obliterating the trace at one location) and it was felt to run a loop aerial around the walls past these places could lead to similar problems. It was thus decided to operate in the allocated VHF frequency band of 102.2 MHz to 102.4 MHz.

Fig. (3) shows a block diagram of the system as it was finally decided upon, and more detailed explanations of the circuitry follow in the next section.

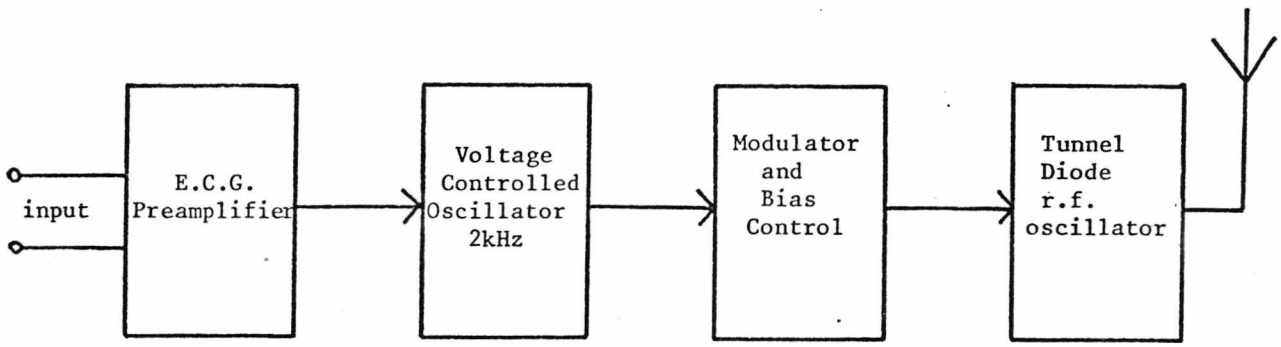


FIG. (3a) The block diagram of the transmitter

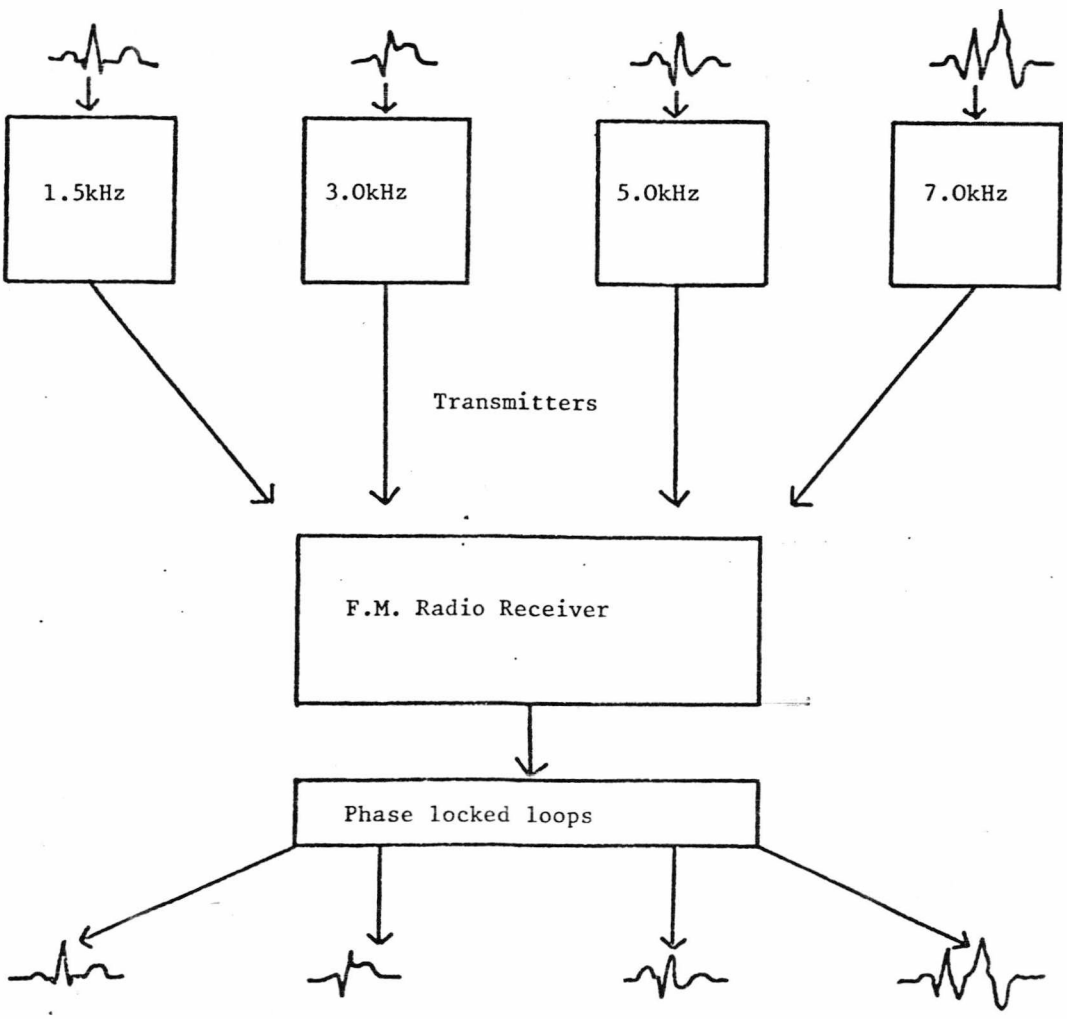


FIG. (3b) The four channel system, with each transmitter having its own sub-carrier frequency

5.3 THE CIRCUITS OF THE ECG TRANSMITTER

5.3a Introduction

Since battery life was a premium consideration, and battery size was to be kept as small as possible, then the circuits must be designed for minimum current consumption. Low power, programmable, operational amplifiers, working from supplies as low as ±1.5 volts have become commercially available in recent months[13], and it was decided to try to use these. For power considerations, again, and for simple circuitry, a crystal controlled tunnel diode oscillator was developed to generate the carrier frequency of 102 MHz.

5.3b The ECG Preamplifier

The ECG preamplifier used a standard instrumentation amplifier design[14] which gives a high input impedance, differential input amplifier with good common mode rejection. The final circuit can be seen in *Fig. (4)*. R_1 and R_2 act in conjunction with D_1 and D_2 to safeguard the amplifier during defibrillation. Any voltage difference between the electrodes of greater than ±0.6 volts is clamped at that value by the diode action, and any further increase in voltage only serves to increase the bias current through the diode and resistors R_1 and R_2 i.e. if a 100 volt pulse were applied, then the current through the diode = $\frac{100\text{volts}}{20\text{k}\Omega}$ = 5m/a. This is safe for the diodes, and the input of the amplifier is protected since 0.6 volts is well within the maximum limits of the operational amplifiers[13].

Resistors R_3 and R_4 are present to provide a path for the input bias currents for the bipolar transistors in the operational amplifiers. Although these bias currents are very

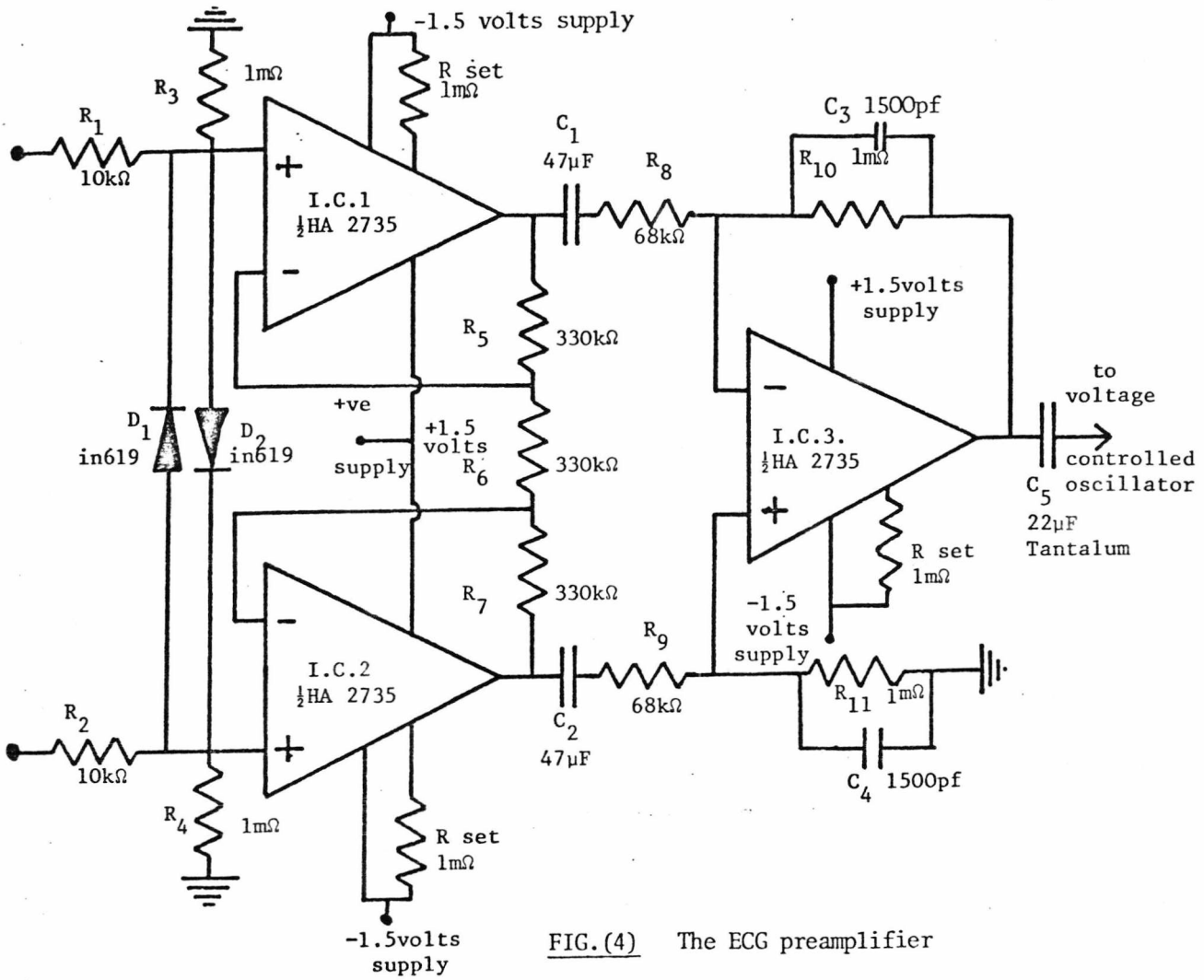


FIG. (4) The ECG preamplifier

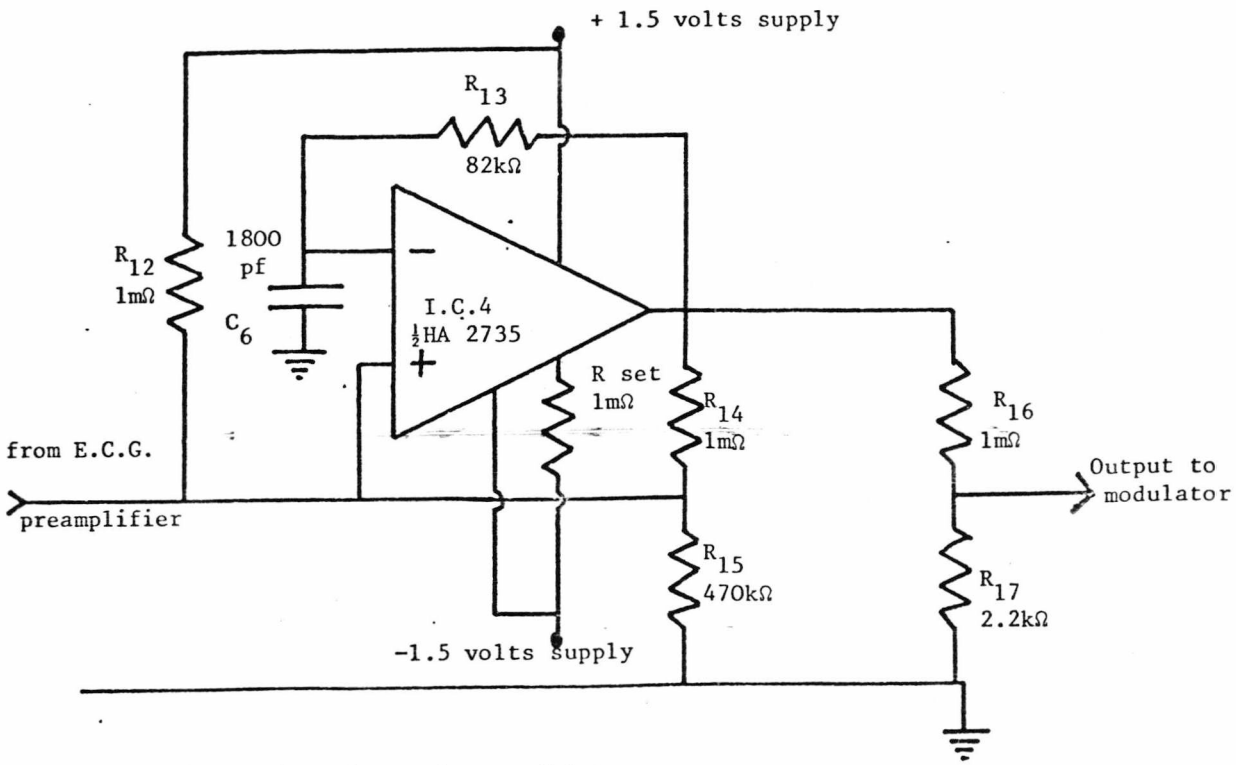


FIG. (5) The sub carrier oscillator

small (5nA with I_{set} = 1.5μA) they are necessary for stable operation of the circuit. Relying upon the bias through the electrodes was not satisfactory and also a third patient electrode was required to complete the current path.

Resistor sets R_1, R_2, and R_3,R_4, must be closely matched, otherwise they adversely affect the common mode rejection. Each pair R_1-R_3, R_2-R_4 acts as a potential divider, so any inequalities in the ratios will mean that, for a common input signal, there will be a difference in the values of signals at the inputs of IC1 and IC2 and hence an output. The gain of the first stage of this instrumentation amplifier is given by the formula $G = 1 + R_5/R_6 + R_7/R_6$. Since d.c. coupling is used at the front end, and d.c. offset potentials from the electrodes of up to ±100mV could be encountered in practice, then the gain of the first stage should be limited to avoid saturation of the output. Initially, a gain of 10 was chosen, but it was subsequently found necessary to reduce this and eventually all the resistors R_5, R_6 and R_7 were made equal in value to give a gain of 3. Capacitors C_1 and C_2 couple the signal to the next stage and block any d.c. levels present, and so enabling high gains to be used in this stage as required. The amplifier is a standard differential amplifier using a single active element (IC3) and its gain is determined by R_{10}/R_8 and R_{11}/R_9 with R_8 and C_1 and R_9 and C_2 determining the lower frequency cut-off point of the whole amplifier. To obtain a 3 second time constant C_1 and C_2 were chosen as 47μF and R_8 and R_9 were calculated to be 68kΩ each. Once again the common mode performance of the whole amplifier depends upon the matching of resistors R_8-R_9 and R_{10}-R_{11}. Capacitor C_3 and C_4 provide high frequency roll-off to limit the frequency response.

5.3c The Sub-Carrier Oscillator

This uses a single operational amplifier IC_4 in a standard square wave oscillator circuit[15] which is shown in *Fig. (5)*. If the output has just changed from its positive to its negative value, then due to R_{14} and R_{15} a negative voltage will be present on the non-inverting (+) input of IC4. The capacitor C_6 will be charged via R_{13} towards the negative rail and when the voltage across the capacitor is greater than that on the + input then the circuit quickly changes state and the output goes positive. This sets a positive voltage on the + input and C_4 now is charged up positively until once again it exceeds the + input value and the output changes state.

Modulation of the sub-carrier oscillator is achieved directly by the voltage from the ECG preamplifier, which comes via C_5 to the junction of R_{14}, R_{15} and the + input. This varies the voltage to which C_6 must charge, and so alters the frequency of the oscillator. To avoid having reverse polarity voltages applied to the capacitor C_5, R_{12} was included in the circuit to ensure that the potential of the + input was always positive.

5.3d The Radio Frequency Circuit

Circuits for crystal controlled oscillators at 100MHz using tunnel diodes as the active elements are available[16-18] and an example of one is shown in *Fig. (6)*. The tunnel diode D_a is biased by R_a and R_b via L_a, in the negative resistance region of its characteristic. The tuned circuit consists of L_a, in parallel with C_b and the varactor diode VC_a in series. At its resonance, the crystal has a very low impedance, and so the parallel resonance circuit is completed and is excited

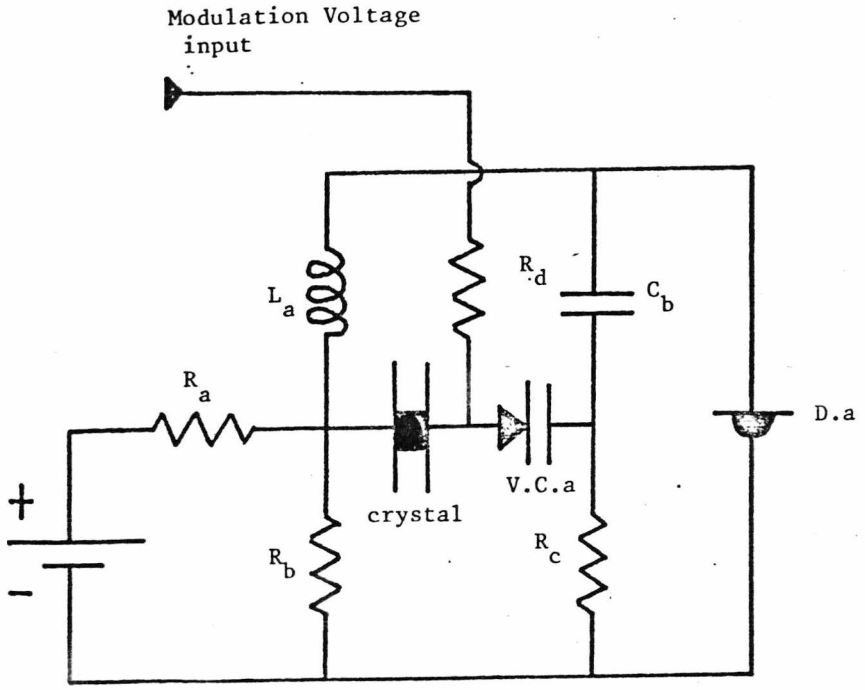


FIG. (6) The circuit of the tunnel diode oscillator as used by Kavanaugh

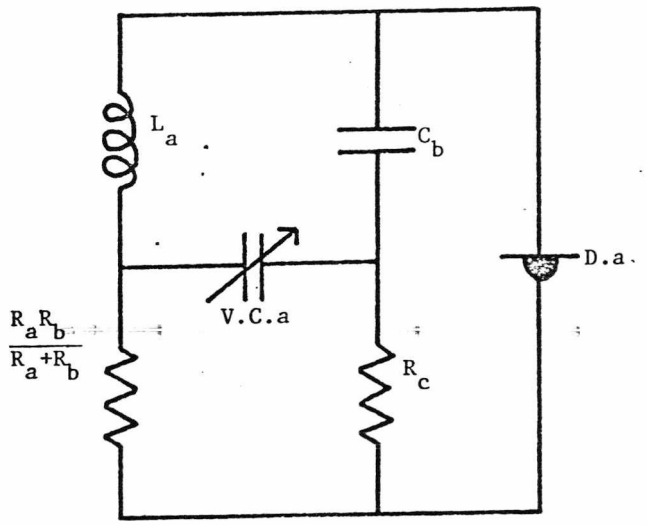


FIG. (7) The a.c. equivalent circuit derived from Figure 6.

by energy from the tunnel diode.

One of these was constructed, but several problems were encountered in its construction. The first arose with the values of the bias resistors R_a and R_b, since the values given in the circuit were found to be too high. It was necessary to reduce them by at least half and preferably by a factor of 10. This was because the bias resistors acted in series with the tuned circuit *(Fig. (7))*, and so large values damped the oscillations. A large value capacitor (4,700pf) was placed across R_b, and R_c was removed, and these modifications *(Fig. (8))* enabled the circuit to oscillate well with the original values of R_a and R_b.

This still meant, however, that approximately seven times the current that was used by the diode was being dissipated in the bias network (diode current = 250μA and bias current = 2mA). This could not be reduced either since the bias stability of such a circuit depends upon this inequality. Since battery life was an important consideration then a way to reduce the wasted power was needed.

The final circuit that was developed is shown in *Fig. (9)* and it can be seen that one of the lower power operational amplifiers[13] is used as the bias supply. This time the bias voltage is determined by the potential divider, R_{23}-V_{R1} and as it is at the input of the operational amplifier and the input bias currents are so small, only $2\frac{1}{2}$μA flows through them. The bias voltage is applied by the integrated circuit through L_1 to the tunnel diode oscillator section consisting of D_3, L_2, C_8 and the crystal, with C_7 and L_1 decoupling the bias supply. The resistance R_{22} determines the value of the standing current used by the device, and with the value of 470kΩ this current is only 50μA - very much less than the consumption of the bias

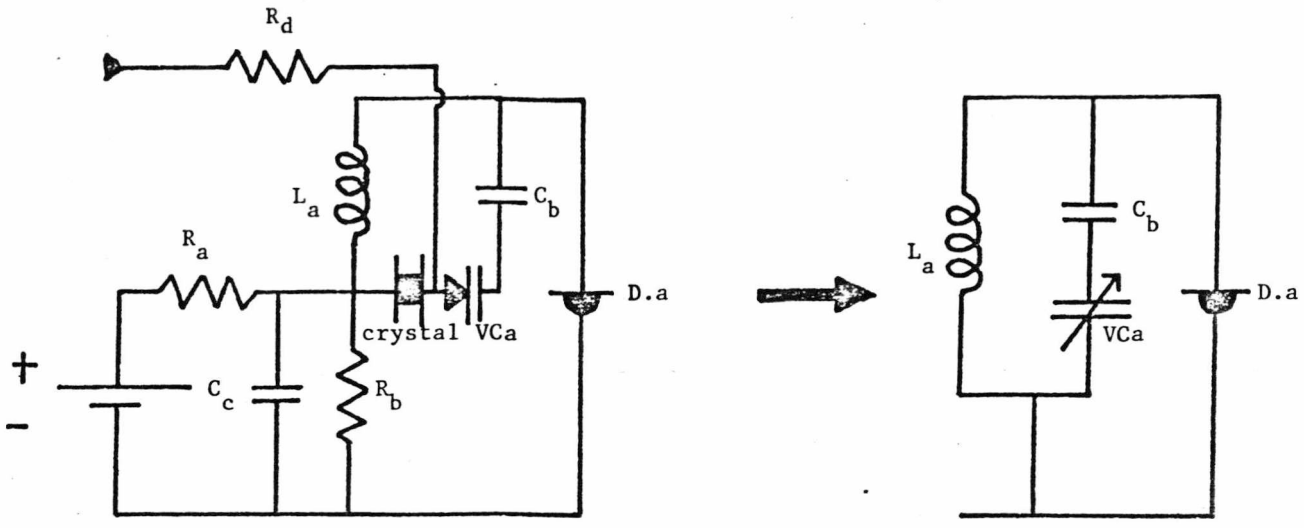


FIG. (8) The modified circuit of Figure 6 and its a.c. equivalent circuit

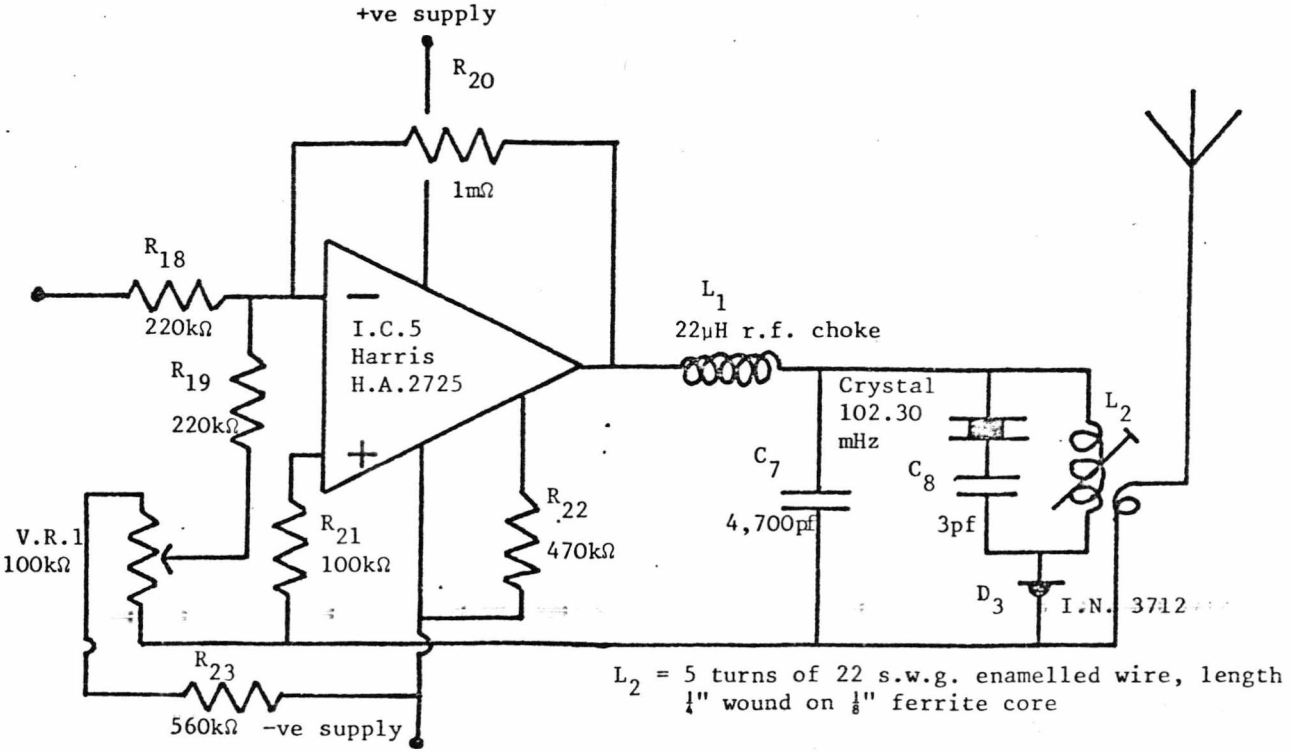


FIG.(9) The circuit developed for the tunnel diode oscillator, with its bias and modulation supplied by an operational amplifier

chain of *Fig. (6)*.

It was also found that the precise frequency of oscillation depended upon the actual value of bias. The diode appears in parallel with the tuned circuit and its junction capacitance thus affects the operating frequency by pulling the crystal slightly. Since the capacitance is voltage dependant, then varying the bias will vary the frequency. Thus, this property was used to modulate the carrier frequency (since in the FM-FM system linearity is not required) and the varactor diode is dispensed with.

The sub-carrier signal was attenuated to the correct level by R_{16} and R_{17} *(Fig. (5))* and applied to the input of the operational amplifier IC5 via R_{18}. Therefore, IC5 acted both as a modulator and a bias supply, giving a level of +195mV with a ±10mV variation for modulation.

5.4 THE PERFORMANCE OF THE CONSTRUCTED CIRCUITS

The bandwidth of the ECG preamplifier is plotted in *Fig. (10)* and the lower and upper 3dB points are 0.05Hz and 110Hz respectively and so are within the specified limits. It was originally thought that a gain of 10 would be needed in the first stage of the preamplifier, but this had to be reduced, because of the small range of linearity of the sub-carrier oscillator. Due to the necessity of close matching of components for common mode rejection, it was easier to increase the value of R_6 to reduce the gain, and this was done. The overall gain of the preamplifier was 45. The common mode rejection ratio is plotted in *Fig. (11)* and it can be seen that whilst it is good at low frequencies, it becomes small at the upper limits of the frequencies of interest. At 50 Hz it is

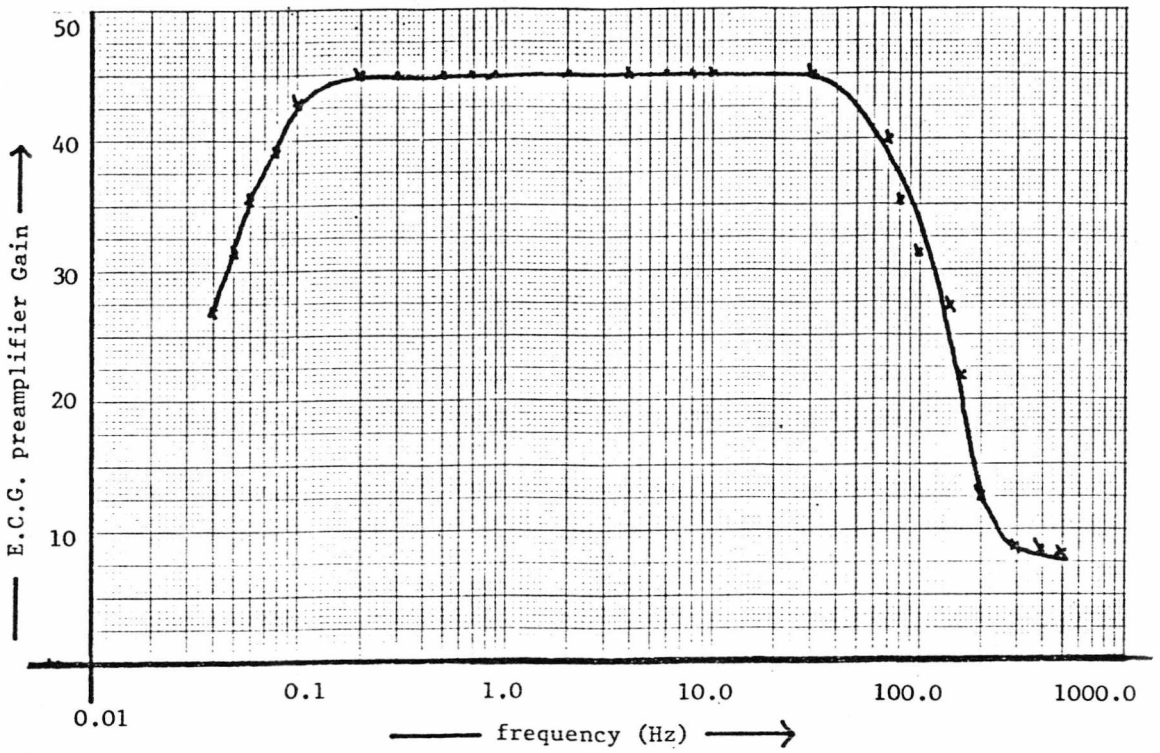

50
40
30
20
10
E.C.G. preamplifier Gain
0.01
0.1
1.0
10.0
100.0
1000.0
frequency (Hz)

FIG. (10)

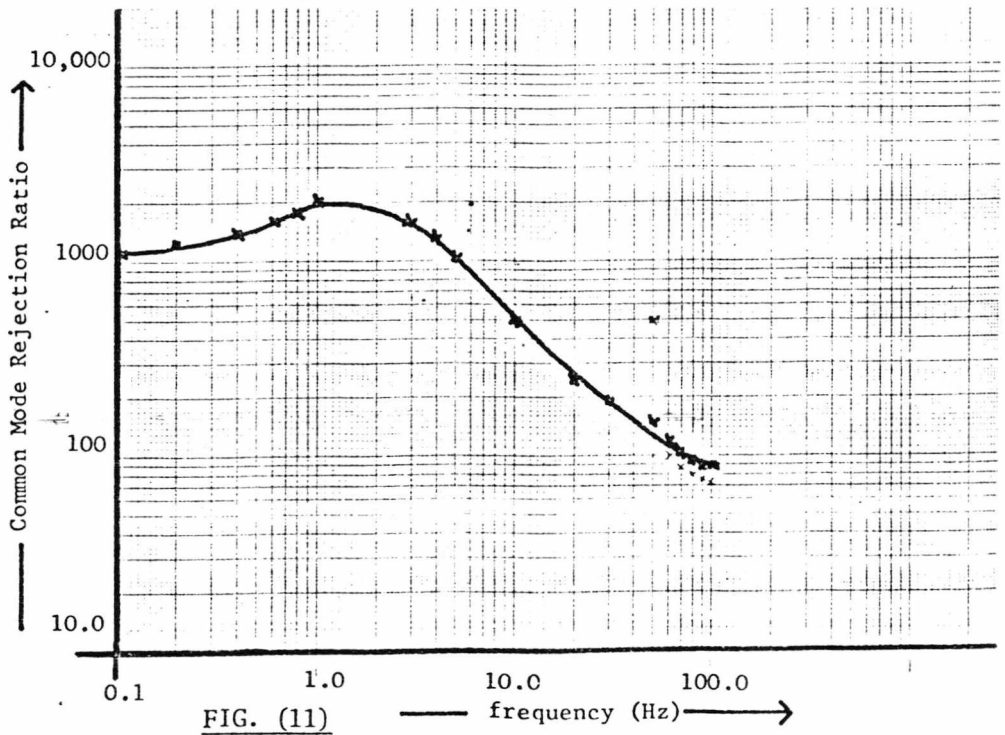

10,000
1000
100
10.0
Common Mode Rejection Ratio
0.1
1.0
10.0
100.0
frequency (Hz)

FIG. (11)

only just adequate with a value of 150:1 . Although care had been taken in the selection of the resistors and capacitors, the results reflect the difficulty of matching many components, especially the large value tantalum capacitors.

The sub-carrier oscillator oscillated freely and reliably and examples of the waveform can be seen in *Fig. (12)*. It was found that the stability of the oscillator with power supply changes was good - and can be seen in *Fig. (14)*. The exact frequency of oscillation was a little high (2.7 KHz instead of 2 KHz) but this can easily be remedied for four channel operation by increasing the value of C_6. The bandwidth of the modulator input was tested, and can be seen in *Fig. (13)*, and also only a few mV of signal were needed to modulate the frequency of the sub-carrier oscillator. Above inputs to it of ±60mV the modulation became non-linear due to the charging action of the timing capacitor. Since the current into it is determined by resistor R_{13} and the output voltage, the charging characteristic is not linear and follows the normal exponential shape *(Fig. (15))*. The voltage to which it has to charge is modulated by the ECG signal, but due to the non-linearities equal positive and negative voltage changes do not result in equal time changes in the period of oscillations. If the voltages are small, however, then the differences are negligible. In this case a limiting value between 60 to 80mV was found *(Fig. (16))*.

The bias and modulation circuitry developed for the tunnel diode was satisfactory in use and it was found that the oscillations stayed locked to the crystal over a bias range of 170mV to 205mV[19]. The presence of the crystal meant that the modulation depth of the carrier frequency was quite small - about 200 Hz/mV. Without the crystal the oscillator worked

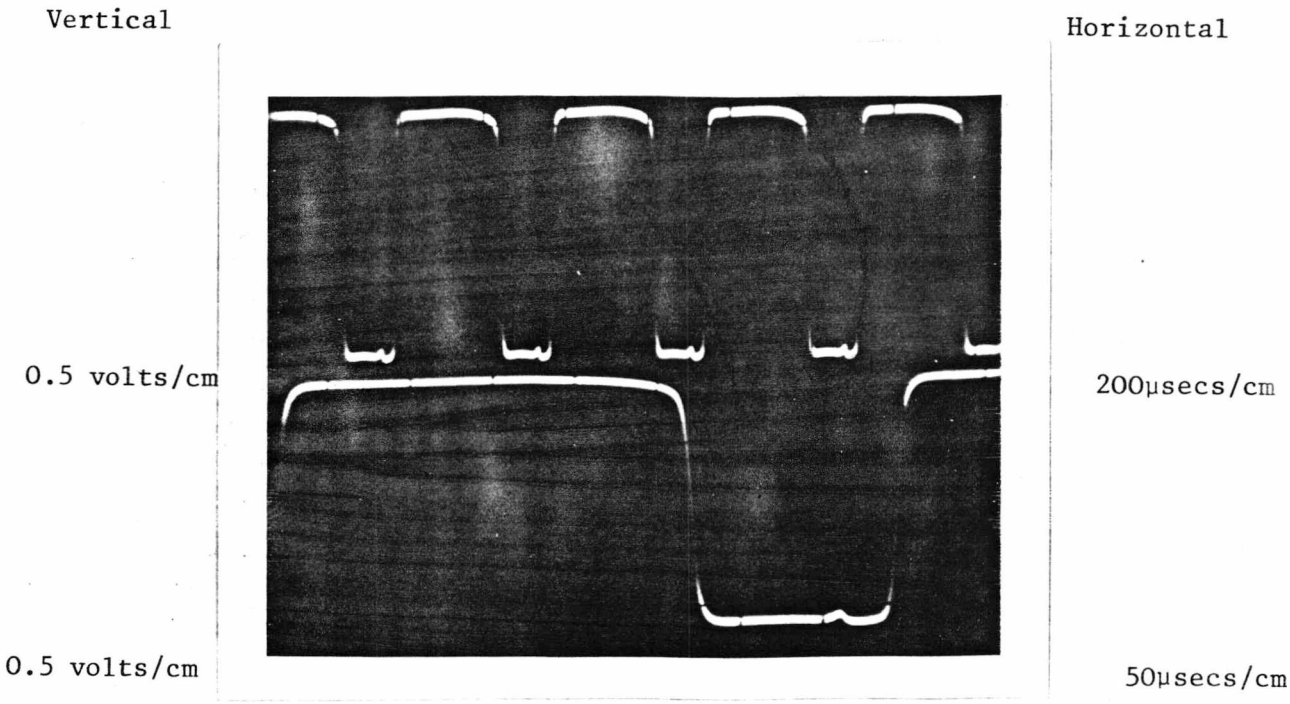


FIG. (12) The output waveform of the subcarrier oscillator

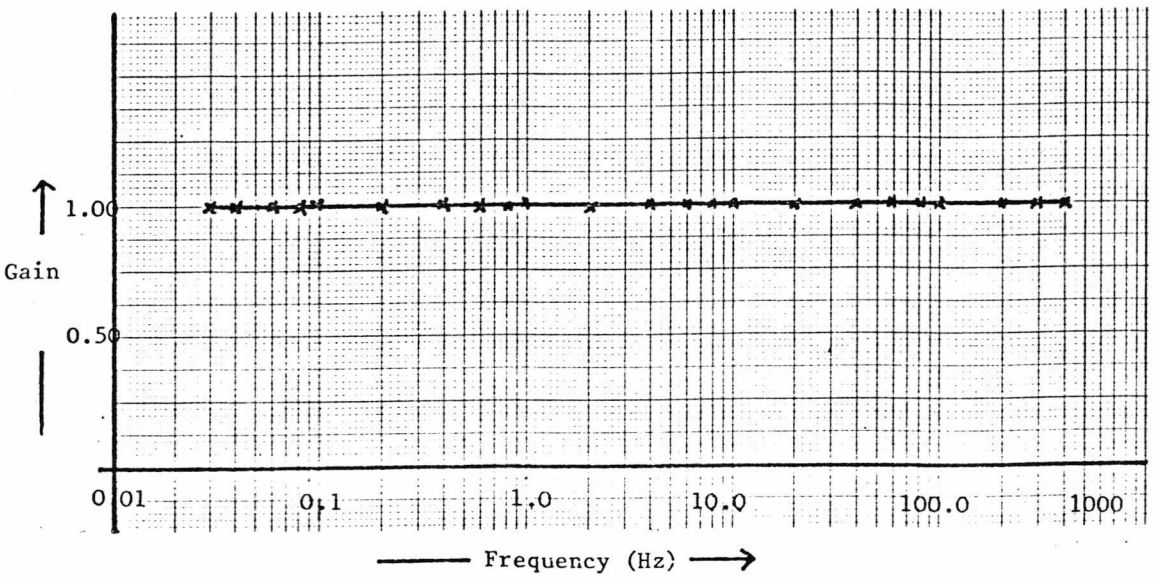


FIG (13) The bandwidth of the modulator input

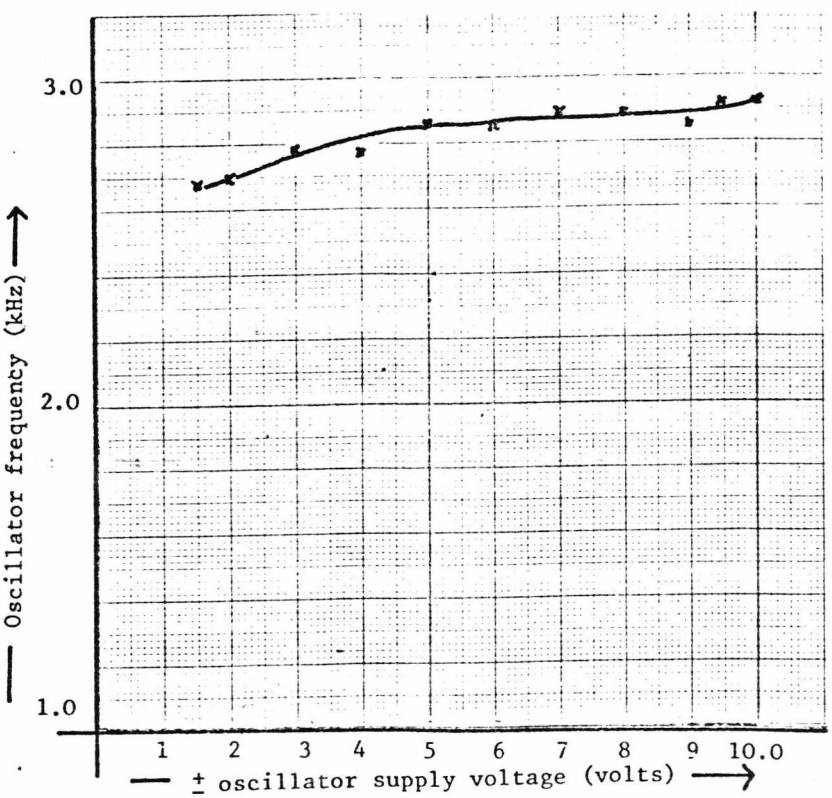


FIG (14) Stability of subcarrier oscillator against supply voltage

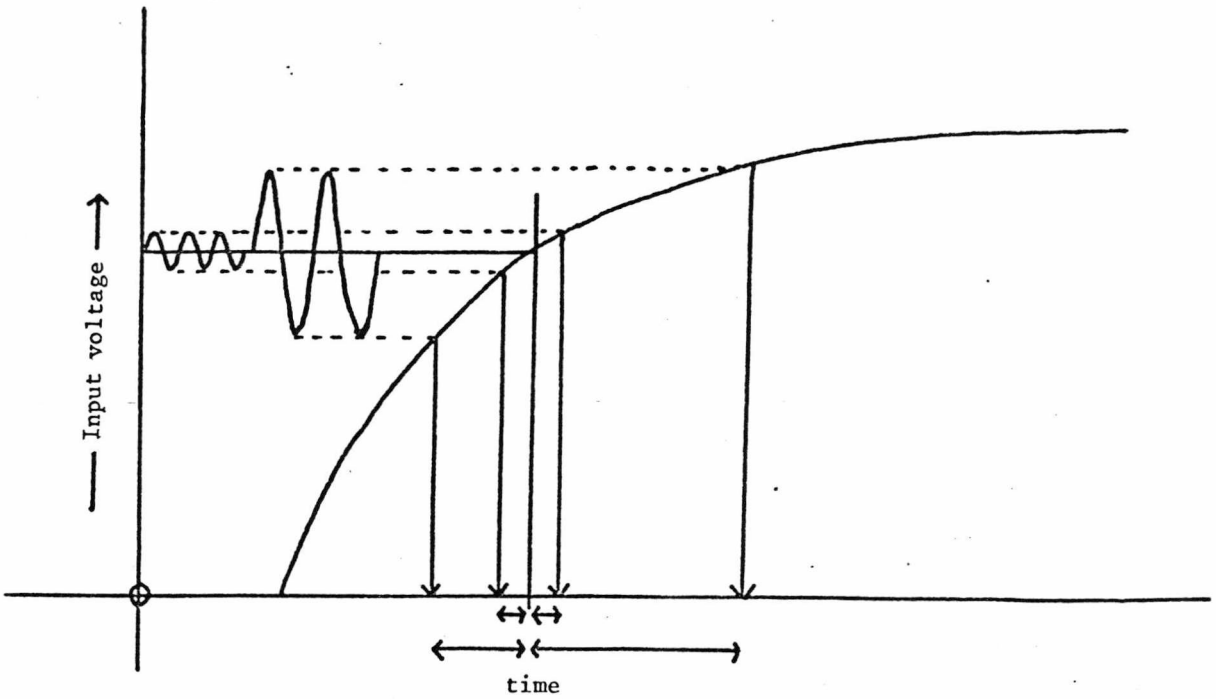


FIG. 15 The effect upon the modulation linearity of the subcarrier oscillator

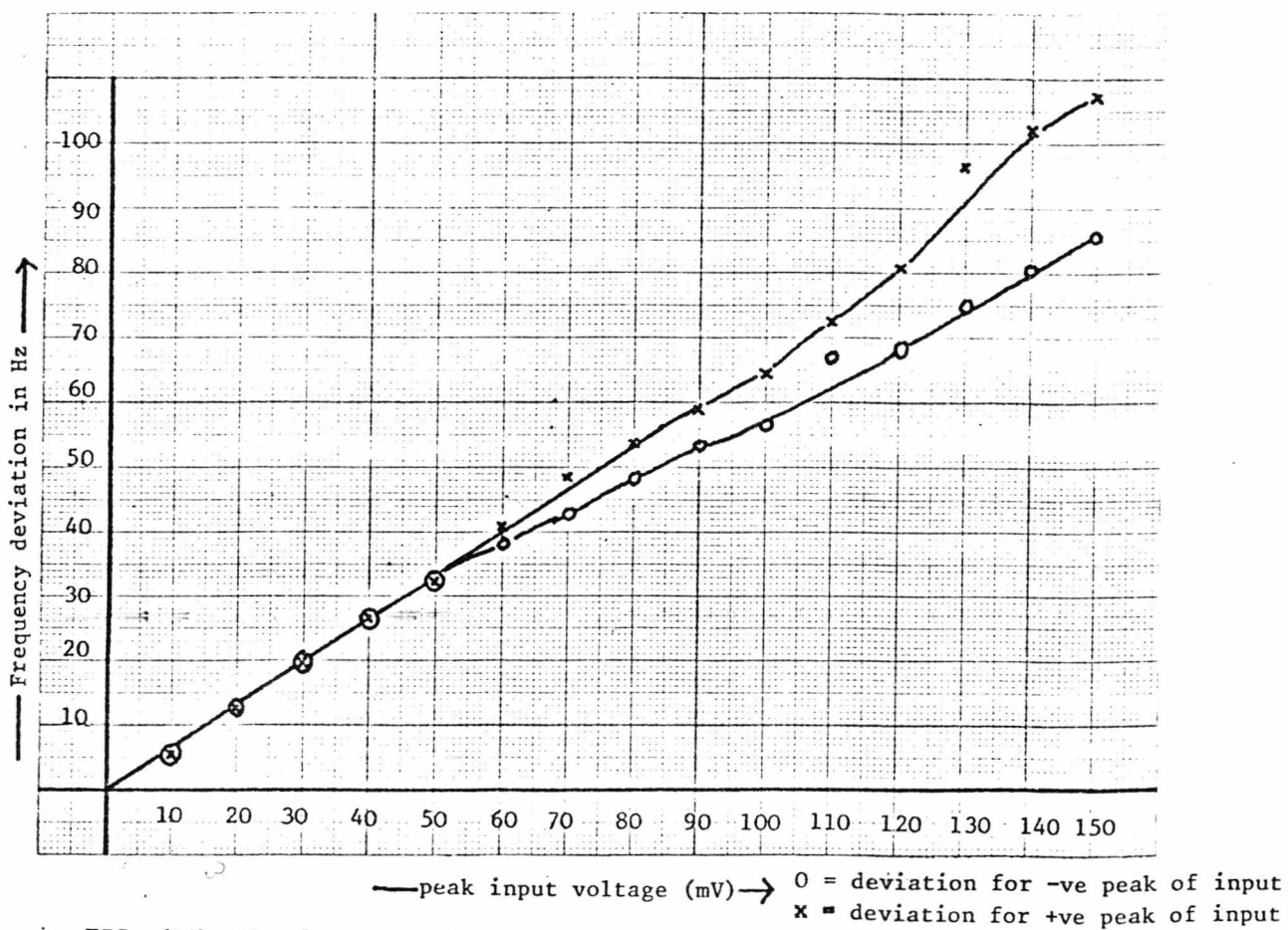


FIG. (16) The frequency deviation of the subcarrier oscillator and input voltage polarity

over a much wider range of bias, and had a modulation sensitivity of several KHz/mV. Due to the small range of bias voltages, and allowing for the battery voltage changes during its lifetime, then only 10mV peak-to-peak modulation could be used. This corresponded to frequency deviations of only 2 KHz, but the receiver was able to detect these and gave an output of 5 to 10mV. The change in bias with battery lifetime is plotted in *Fig. (17)* and since the -ve battery determines the bias value it is the changes in this that cause the greatest deviations. Decreases in the voltage of the +ve battery have little effect at all. The current consumption of the whole circuit was 380μA with the modulator alone consuming 50μA - which is the desired improvement over existing circuitry for tunnel diode oscillators.

5.5 THE RECEIVER

The receiver used was a QUAD F.M.3, a commercial unit for the broadcast bands. Its detector gave sufficient output even though the depth of modulation was quite small. For the initial transmitter that was built, the sub-carrier was demodulated by the same frequency to voltage converter that was used for the ambulance telemetry system (Chapter 4). For four channel operation, phase locked loops will be used for the separation of the signals. The devices can have both an input bandwidth and centre frequency defined[20] so that they are well-suited to the separation of the channels *(Fig. (3))*.

5.6 THE SYSTEM PERFORMANCE

The transmitter sections were assembled on purpose built printed circuit boards and put into a small plastic box measuring

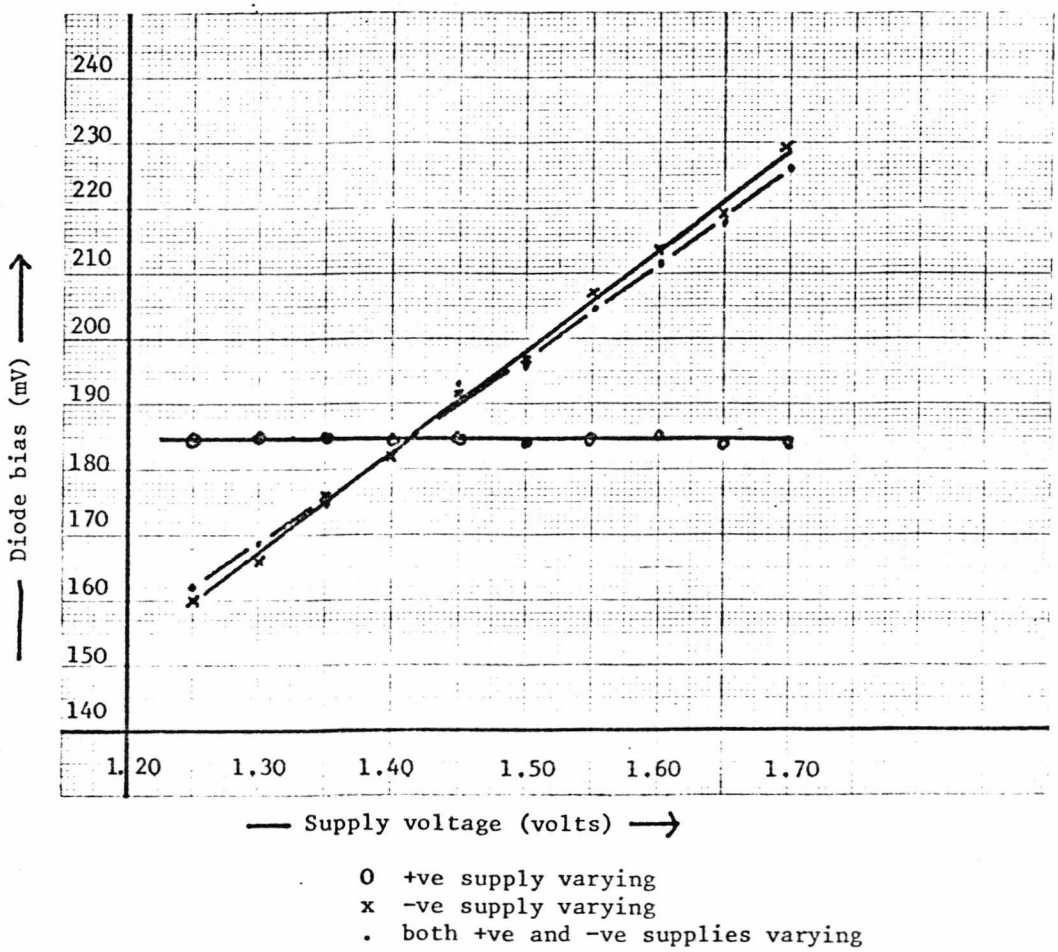


FIG. (17) The variation of the Tunnel Diode Bias against changes in Supply Voltage

7½cms × 6cms × 3cms and the whole transmitter with the batteries weighing only 100gms. The total current consumption was 450μA; and with the batteries used (Mallory type MN2400) this gave a battery life of six to eight weeks continuous running. This was more than adequate for the situation, and could allow for the use of much smallter batteries (i.e. Mallory type RM312) should the overall size have to be reduced any further. Because of the long life of the prototype transmitter the circuit power supply wires were soldered directly to the batteries, although this is obviously an impossible practice for the routine clinical operation of the system.

Examples of transmitted traces can be seen in *Figs. (18) & (19)* (the former from a simulator, the latter from a subject). The top trace in each figure is a direct recording of the signal and the bottom trace shows the signal after being transmitted. It can be seen that the transmitted signals are identical to the directly recorded ones, and thus the preamplifiers, modulators and demodulators are working satisfactorily.

The far field range was tested and found to be approximately 100ft. with a quarter wave length aerial, and 150ft. with a half wave length aerial (using a standard half wave dipole for the receiver). Examples of transmitted traces for different ranges can be seen in *Fig. (20)*. The top recording is the signal taken directly from the simulator to the ECG machine, and the remainder are the transmitted signals from 20, 60 and 100ft.

When the unit was taken inside the building, however, a significantly different set of results ~~were~~ was obtained. Although the range in an open field was 100 to 150ft., the range in the I.T.U. was found to be very variable - and sometimes only a few feet. Often, moving the transmitter through a distance of only 3 or 4ft. could cause the receiver to lose the signal and then

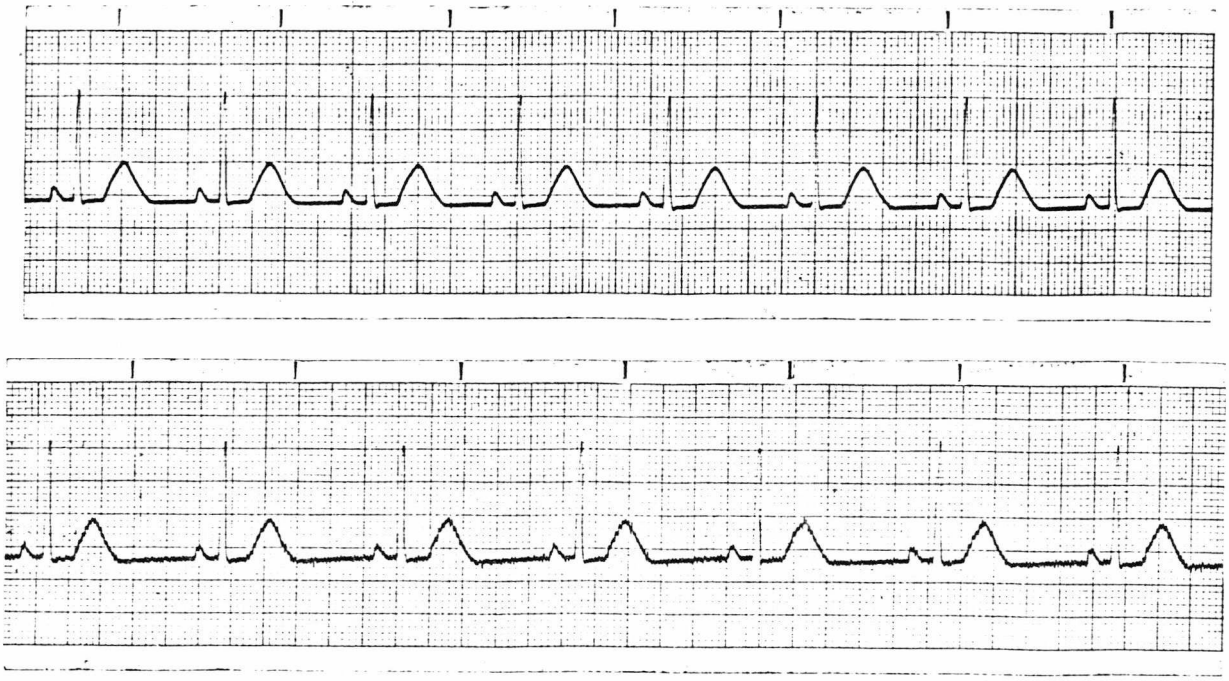


FIG. (18) The electrocardiogram recorded from a simulator.
TOP: is the direct recording from the simulator
BOTTOM: is the transmitted trace

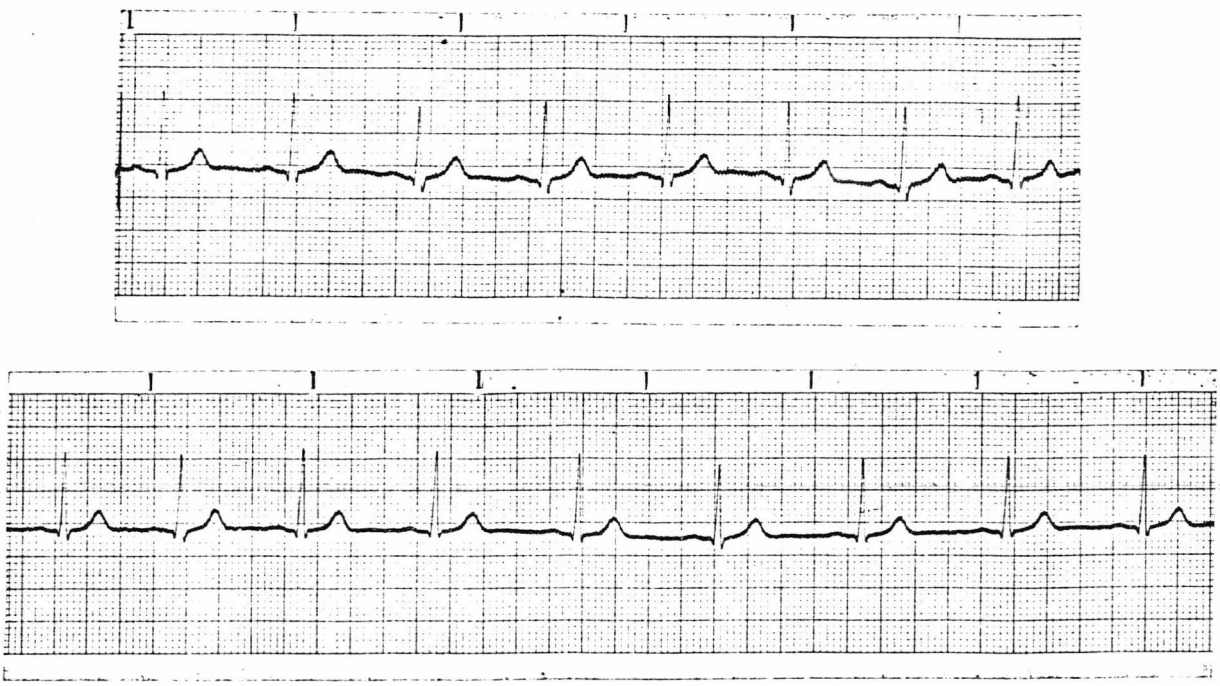


FIG. (19) TOP: a direct recording from a healthy 28 year old male
BOTTOM: the recording of the transmitted ECG from the same person

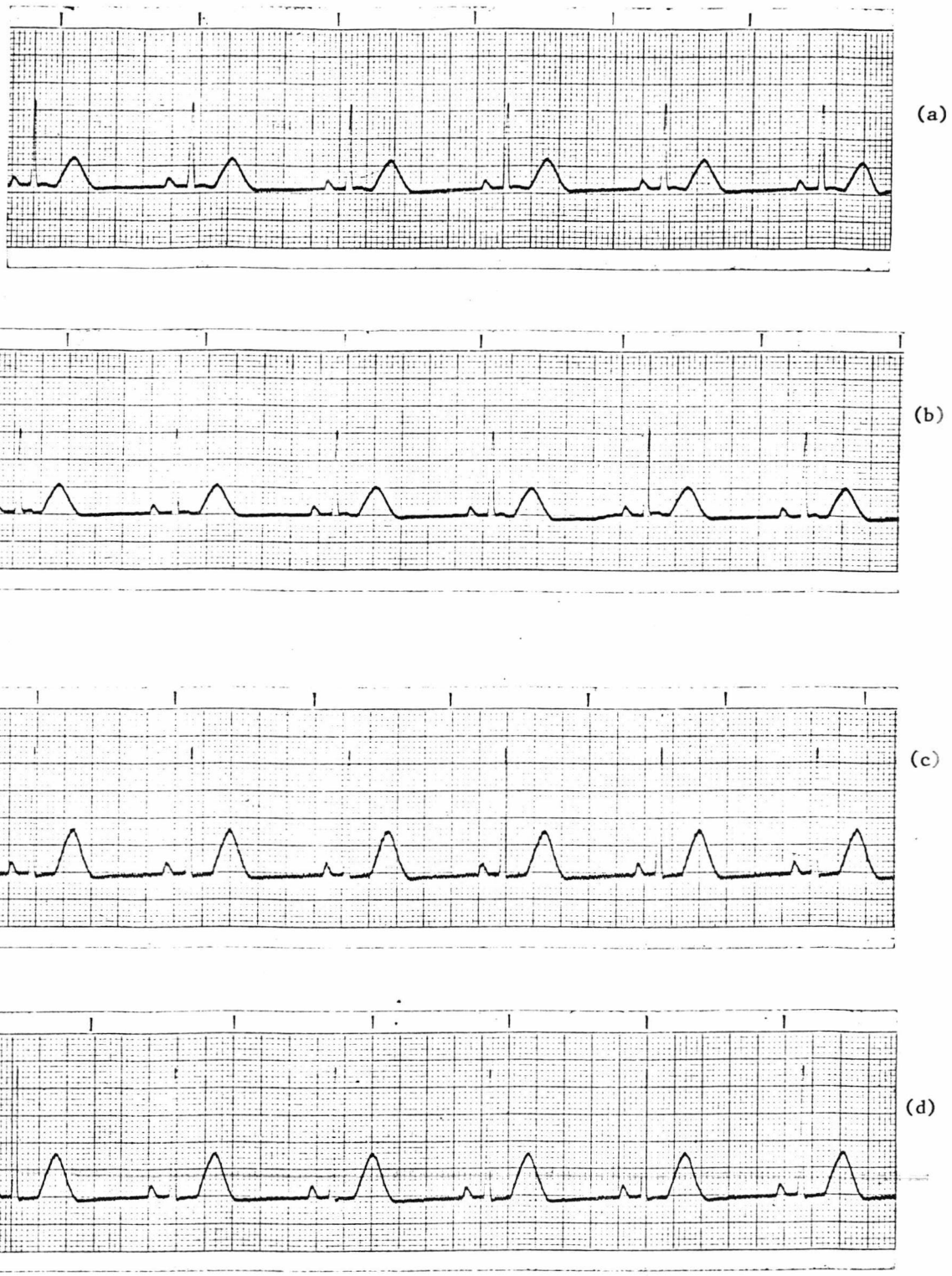


FIG. 20 Recordings as received from the transmitter from different ranges

(a) is a direct recording
(b) 20 feet
(c) 60 feet
(d) 100 feet

catch it again. Also, the moving of objects and people within the I.T.U. were found to affect the reception of the signals. The reason for these anomalies is similar to that experienced with indoor T.V. aerials[21] i.e. the presence of standing waves and multiple reflections in the room.

Due to the large amounts of metal present in reinforced concrete, and the numbers of pre-fabricated panels used in the construction of the Hospital, then any waves propagating in the I.T.U. will be reflected from the walls. The multiple reflections will interfere with the incident waves to form many standing waves and the distribution of these depends upon factors which include the position of the transmitter and the presence of other metal objects (e.g. beds and trolleys) and is unpredictable and changeable. Thus the quality of the reception depends upon the position of the nulls of the standing waves since, if the receiving aerial is in one of these, little or no signal will be received.

With the lower power transmitter it was found that there were considerable problems with these standing wave distributions, and the transmitter as it was constructed was unreliable for regular clinical use. The variation of field strength of the transmitter under different conditions is illustrated by *Figs. (21) & (22)*. The values of the ordinate are not absolute values of the field strength but are a measure of the amplitude of the final i.f. amplifier of the receiver (just before the limiter) and so an indication of the strength of the received signal. The variations that occur when the transmitter is moved from one side of the ward to the other are shown in *Fig. (21)* and *Fig. (22)* shows the effect of people moving about the ward. It is interesting to note that in *Fig. (21)*, as the transmitter approaches the receiver (marked R) and then recedes

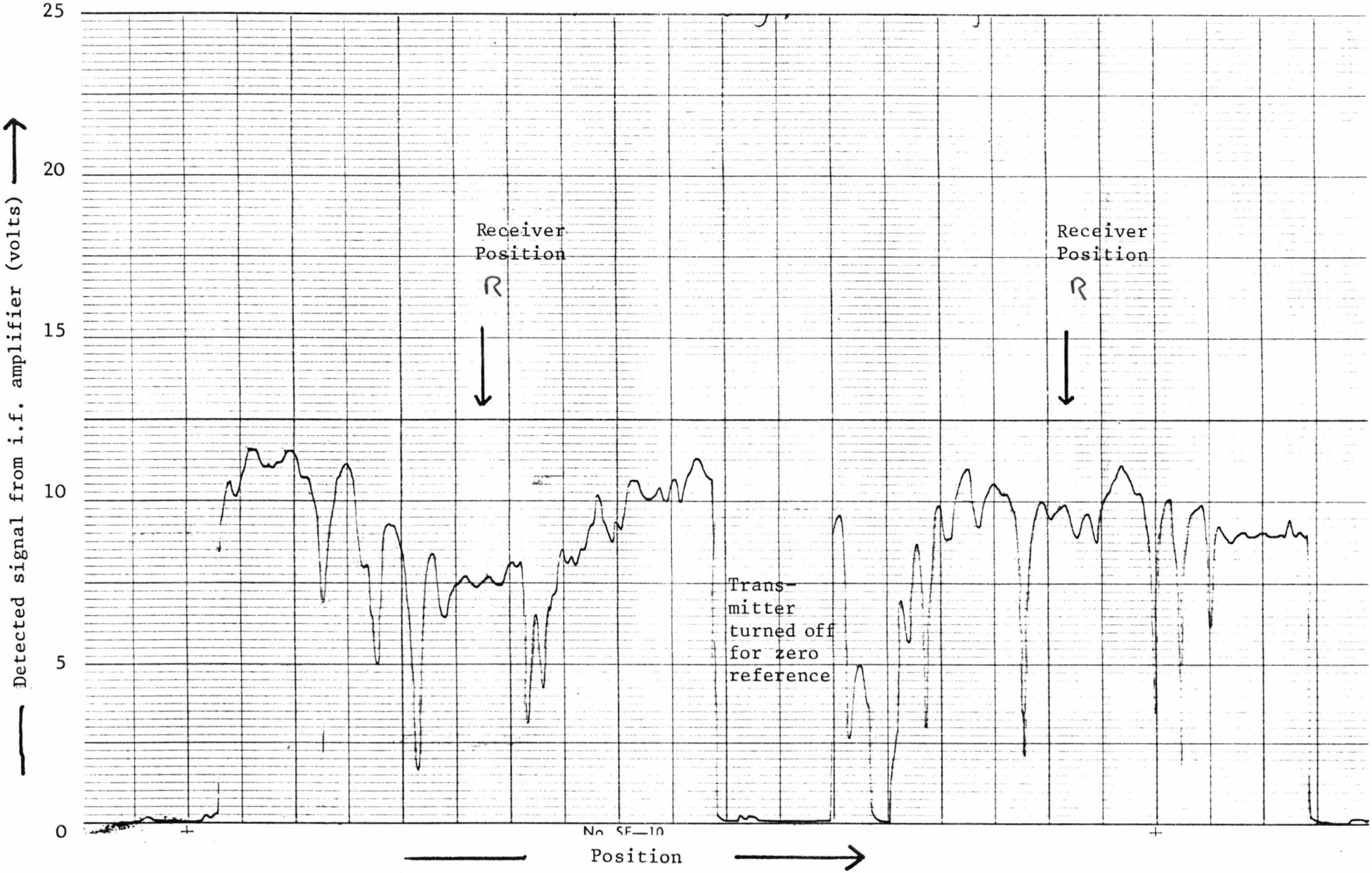


FIG. (21) The change in received signal as the transmitter is moved about the I.T.U.

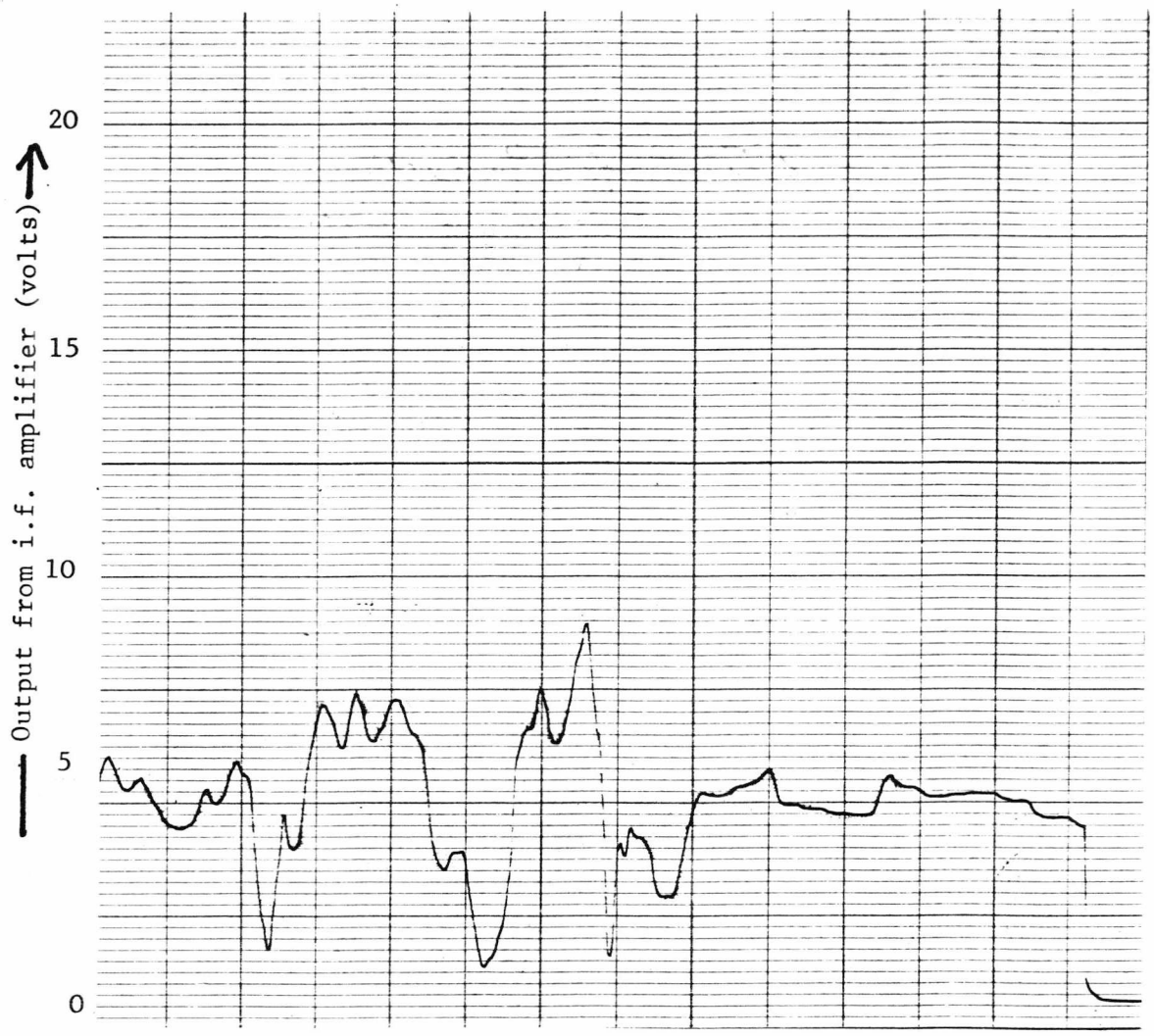


FIG. (22) The transmitter is stationary 20 feet from the receiver, and people are moving about

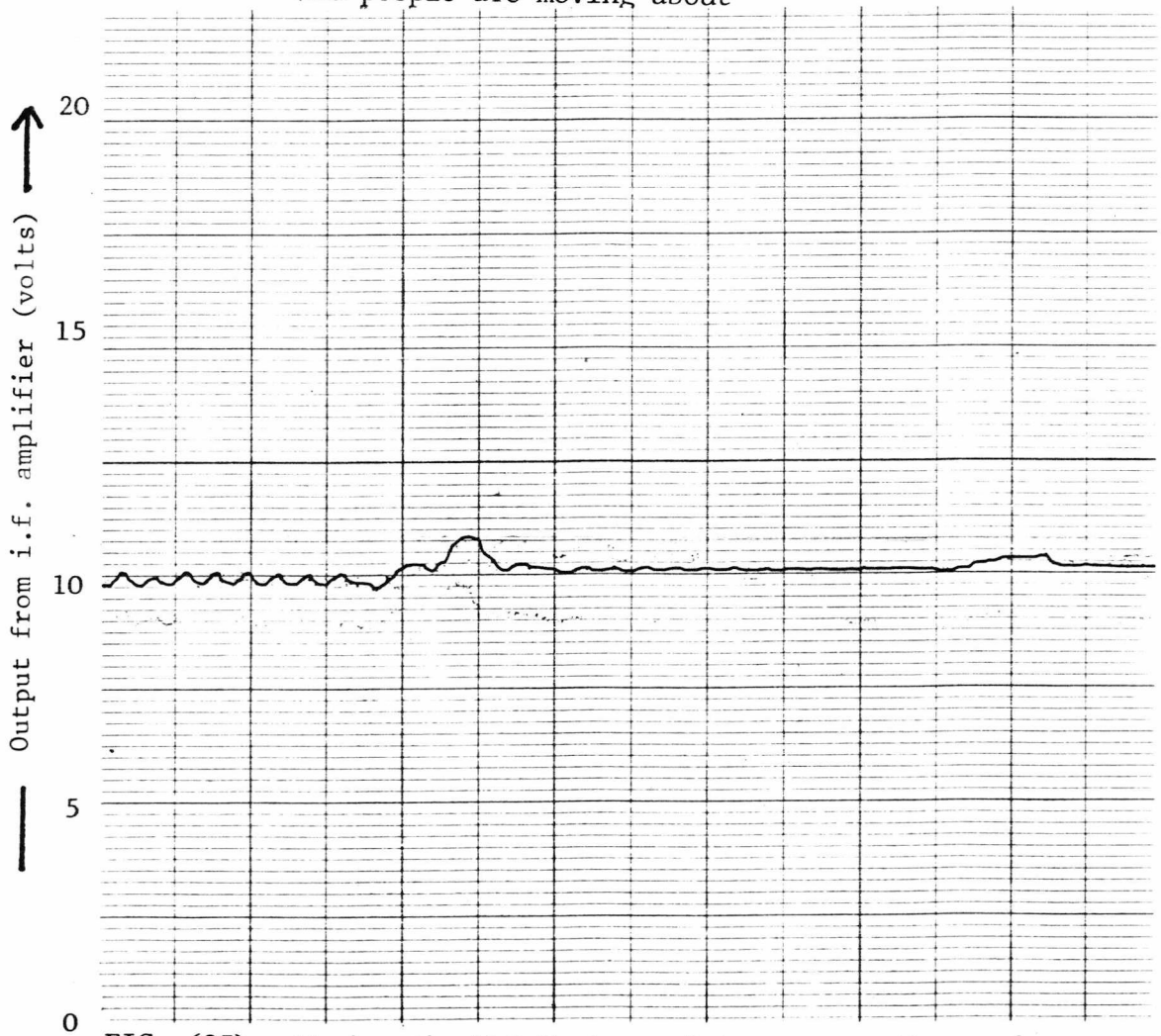


FIG. (23) Moving the M.I.E. transmitter across the ward

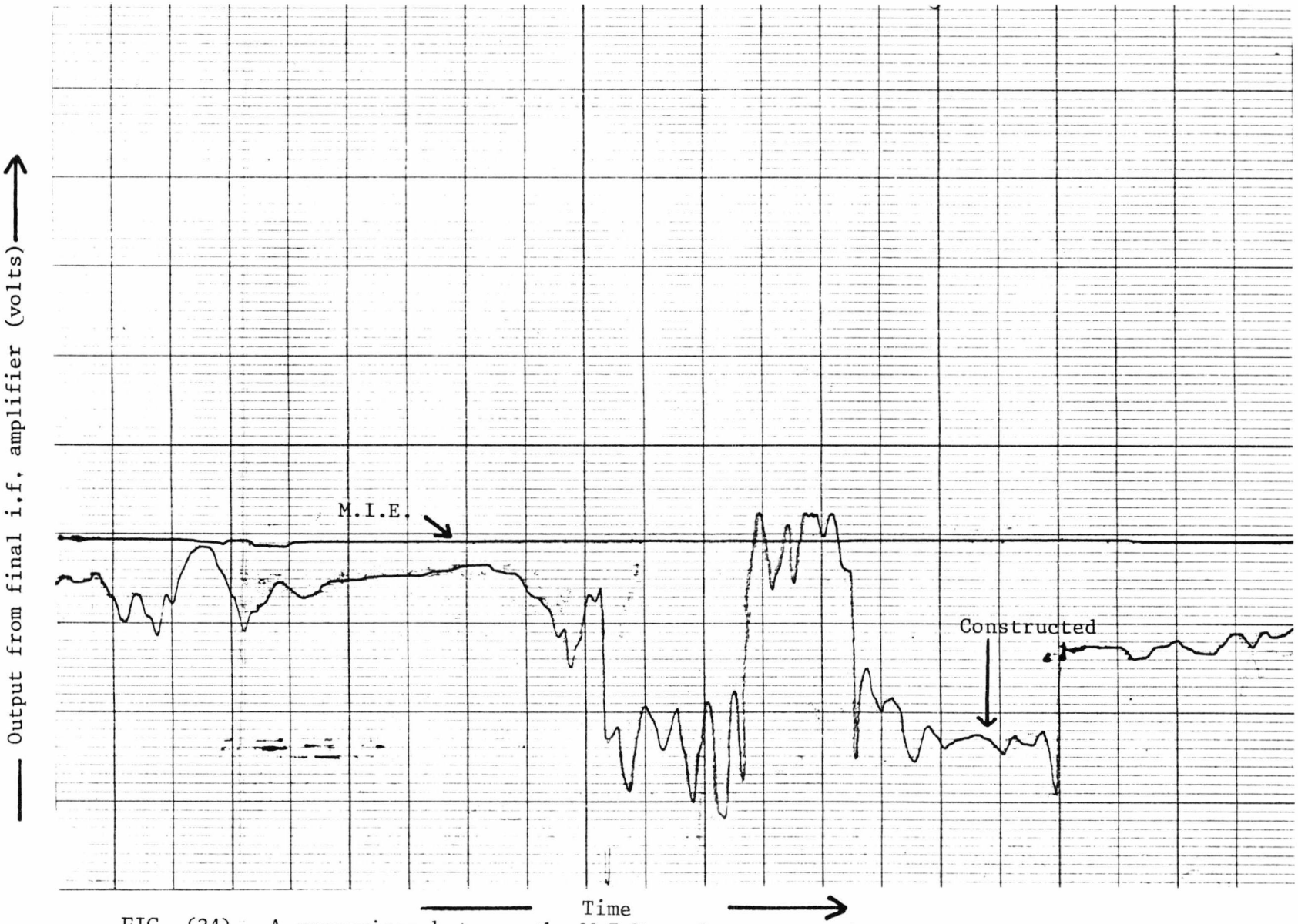


FIG. (24) A comparison between the M.I.E. and constructed transmitters when both placed 20 feet from the receiver, and with people moving about the ward.

from it, that the field strength does not just rise and then fall - it goes through a series of peaks and troughs as the transmitter establishes different sets of standing waves.

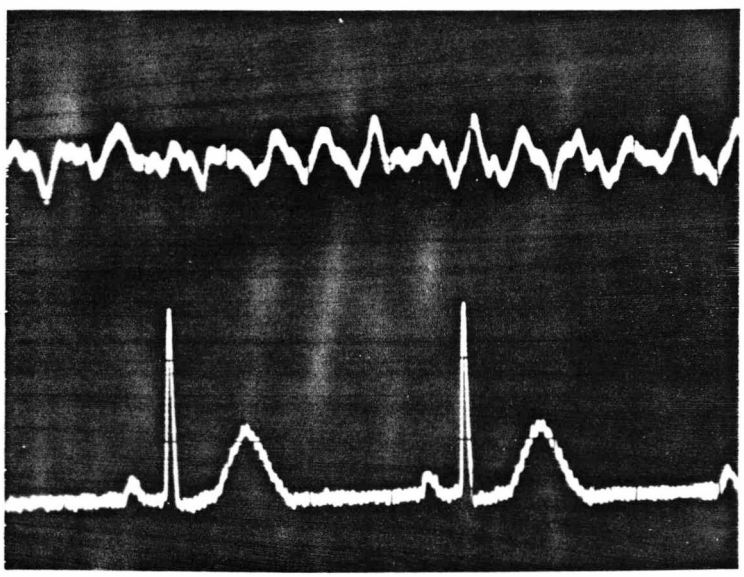
A commercial transmitter was used in the ward as a comparison. This is a much more powerful device, but consequently is very much heavier (270gms) and has a relatively short battery life of 1 to 2 days. As can be seen from the results, however, it does not suffer from the same problems as the constructed transmitter as regards reliability of reception *Figs. (23) & (24).*

Another problem is the noise that is present on the baseline of the output, and hence on the traces *Fig. (25).* Its r.m.s. value is in the order of 10mV and with an ECG signal of peak amplitude 100mV to 120mV, it spoils the trace considerably. Feeding a standard sine wave wave generator into the decoder gives noise levels of 2mV or less and so most of the noise can be attributed to short term instability in the sub-carrier oscillator and noise incurred during transmission. Feeding the tunnel diode modulator with a signal from a standard oscillator instead of the normal sub-carrier generator produced noise outputs of approximately 5mV. Thus 5mV is due to sub-carrier instability and 3mV to the transmission link. With a simple oscillator it would be difficult to decrease the short term instability. The 5mV noise represents an average deviation in frequency of $\frac{1000}{1600} \times 5$ Hz (*Fig. (9)*, Chapter 4) which when compared with the centre frequency of 2.7 KHz, means an error of $<\frac{1}{2}\%$. The best way to overcome the problem is to increase the signal to noise ratio rather than try to improve an oscillator stability. This means increasing the modulation sensitivity of the sub-carrier oscillator, and one way to do this is to have a constant current feedback to capacitor C_6

Vertical

Horizontal

10mV/div



20mS/cm

50mV/div

200 msec/cm

FIG. (25) The noise output from the system

(Fig. (5)) rather than feedback via R_{13} *(Fig. (5))*. This would increase the linearity range, and enable much larger signals to be fed into the sub-carrier generator - which would, in turn, lead to larger frequency deviations and hence a better signal-to-noise ratio at the output.

The results from a subject on the exercise bicycle are shown in *Figs. (26-28)*. The first two of these figures show the results obtained by the present method of doing the test. The patient has two electrodes placed either side of the chest and leads are connected from these to the ECG machine. Movement of these leads causes considerable artefact as can be seen from *Fig. (27)*. Normally, a second technician holds the wires so as to minimise the artefact, whilst the first technician operates the ECG machine.

The transmitter can easily be strapped to the chest of a subject, with short leads to the electrodes, and the results can be seen in *Fig. (28)*. They are a considerable improvement over the standard recordings - especially at the higher pedalling speed of 20 m.p.h. (although patients are requested to keep to 10 m.p.h. only) and can, of course, be obtained with only one technician.

5.7 SUMMARY

A transmitter was designed and built to the specifications determined. When it was completed it transmitted ECGs faithfully and was light and unobtrusive to wear. Although its range was over 100 feet outside when used inside the operation was unreliable due to the generation of standing waves in the ward. The output signal to noise ratio also had room for improvement.

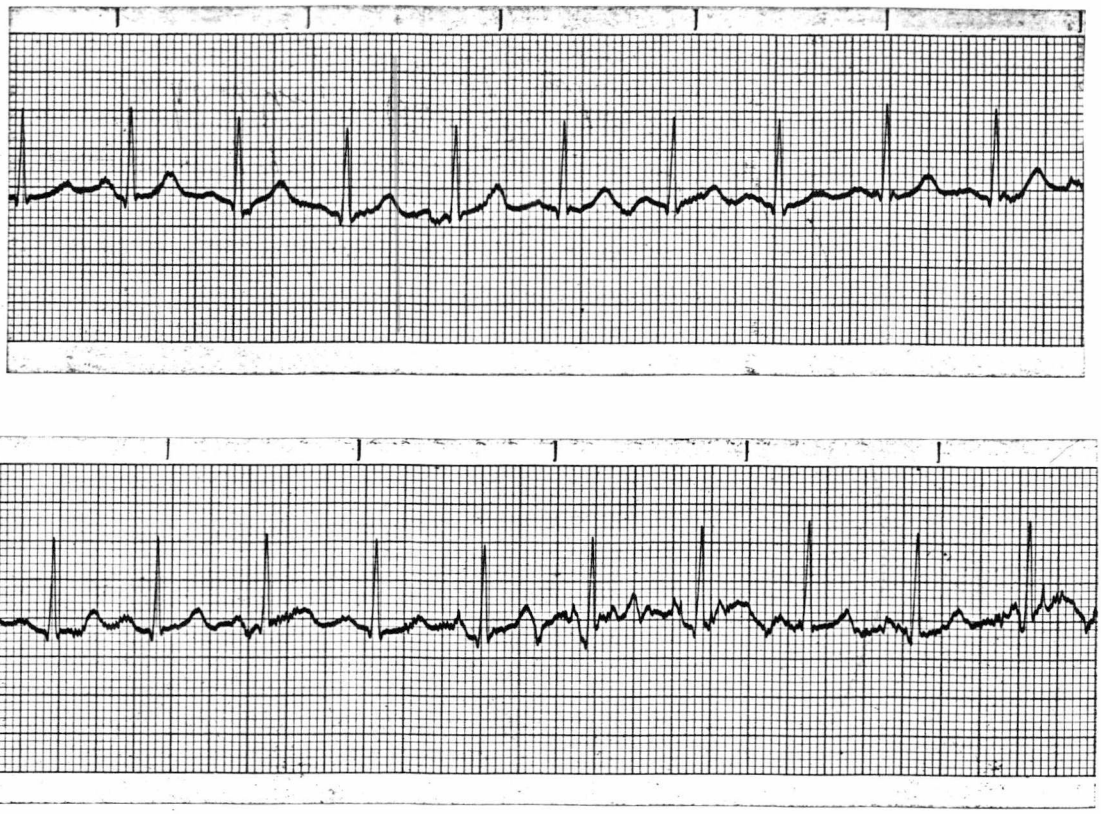


FIG. (26) The direct recordings of the ECG from a subject on the exercise bicycle - the leads are being supported so that as little movement as possible occurs.

TOP: pedalling at 10 mph indicated
BOTTOM: pedalling at 20 mph indicated

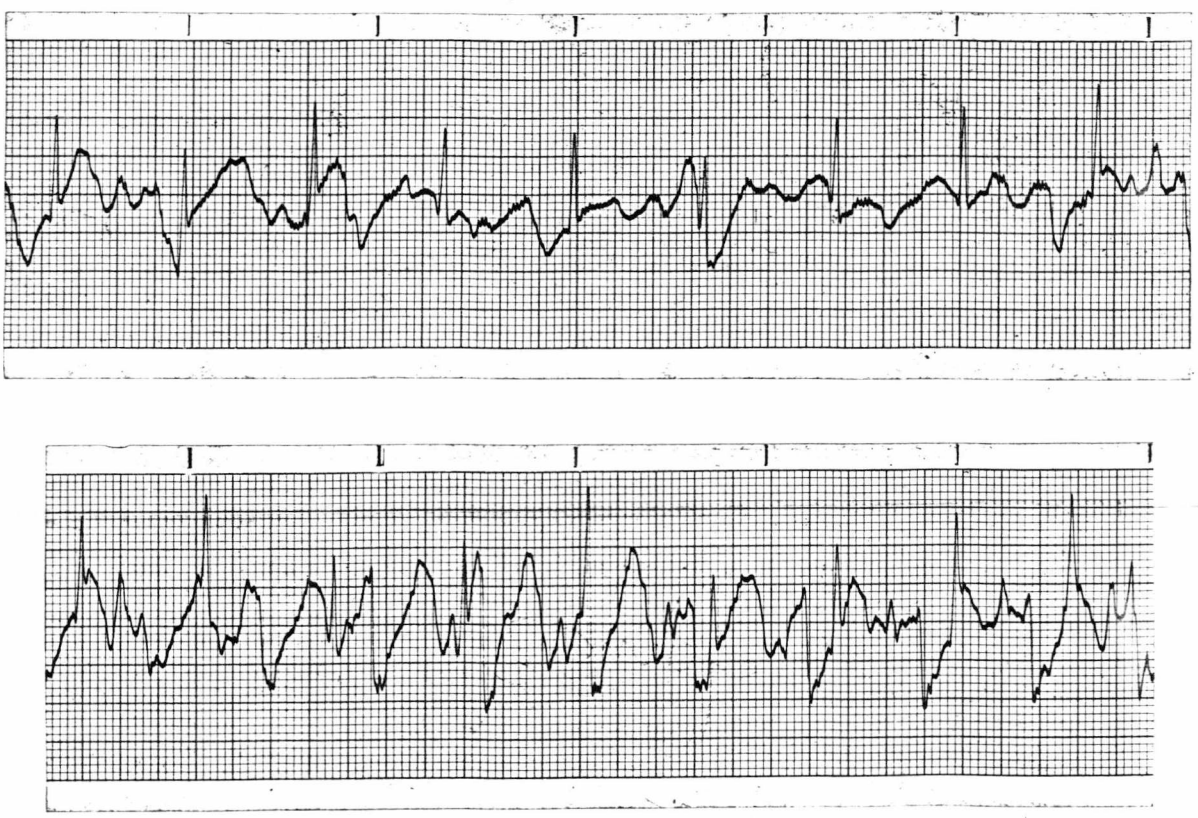


FIG. (27) The direct recordings of the ECG from a subject on the exercise bicycle, with the leads unsupported.

TOP: pedalling at 10 mph indicated
BOTTOM: pedalling at 20 mph indicated

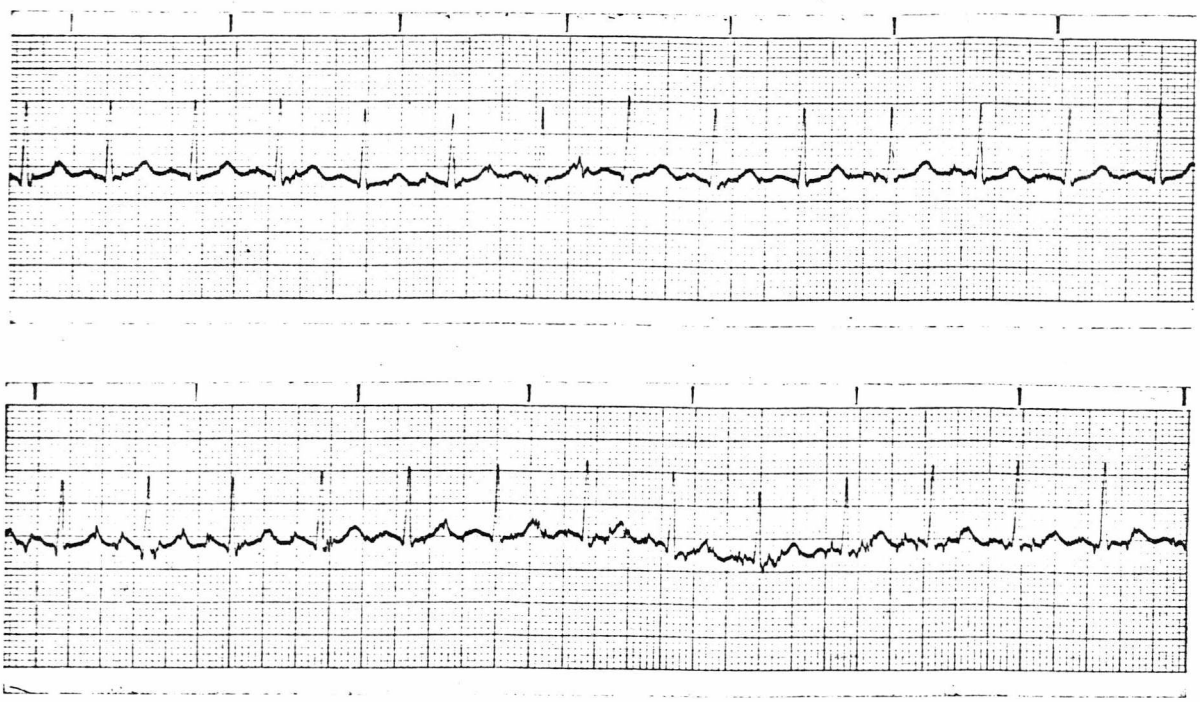


FIG. (28) The transmitted ECG from a subject on the exercise bicycle

TOP: Pedalling at 10 mph indicated

BOTTOM: Pedalling at 20 mph indicated

When used to transmit ECGs from subjects, and in use on the exercise bicycle, the transmitter proved that it could be a very useful tool - well able to satisfy all the criteria and uses which were discussed in the beginning of the chapter.

The unnecessarily long battery life of the transmitter (about 6 weeks) is also a good feature since it allows the possibility of increasing the transmitter power. By reducing the battery life to one week, five times the present current could be supplied to ~~the~~ an output stage, and this would overcome the range problems in the ward. This modification, coupled with the constant current feedback path in the sub-carrier oscillator, will give a versatile and reliable ECG transmitting system, which is easily extendable to four channels - a considerable improvement over commercial equipment.

REFERENCES

(1) J. RAWLES & G. CROKETT "Evaluation of ECG monitoring apparatus on a general medical ward" 1969. Bio-Medical Engineering, Vol. 4, No. 4, p. 154.

(2) D.J. JEWITT et al "Analysis of 150 patients with acute myocardial infarction admitted to Intensive Care Units" 1968. British Medical Journal, Vol. 1, p. 787.

(3) B. PENTECOST & N. MAYNE "Results of general hospital coronary care service" 1968. British Medical Journal, Vol. 1, p. 830.

(4) S.N. POCOCK "Earth Free Patient Monitoring" 1972. Medical and Biological Engineering, Vol. 7, No. 1, p. 21.

(5) HOSPITAL TECHNICAL MEMORANDUM No. 8 "Safety Code for Electro-Medical Apparatus" Aug. 1974. H.M.S.O.

(6) D.S. KORNFIELD "Psychiatric view of the Intensive Care Unit" 1969. British Medical Journal, Vol. 1, p. 108-110.

(7) EDITORIAL COMMENT "Anxiety in Intensive Care" 1971. Bio-Medical Engineering, Vol. 6, No. 2.

(8) P.B. HEWETT "Subjective follow-up of patients from a surgical intensive therapy ward" 1970. British Medical Journal, Vol. 4, p. 669.

(9) J. DOMINIAN & M. DOBSON "Study of patients' psychological attitudes to a Coronary Care Unit" 1969. British Medical Journal, Vol. 4, p. 795.

(10) S. RAMO & J.R. WHINNERY "Fields and waves in communication electronics" 1965. Wiley International Edition, Tokyo, Japan.

(11) R.J. COLLIER "Personal Communication" 1975. Univ. of Kent at Canterbury.

(12) R. JACOB "Aerial design in inductive loop telemetry" 1976. IEEE Trans. on Bio-Medical Engineering, Vol. BME 23, No. 2.

(13) HARRIS SEMICONDUCTOR DIVISION "Wide range programmable operational amplifier HA2730/2735" 1974. Specification sheet.

(14) BURR-BROWN "Operational amplifiers - design and applications" 1971. McGraw-Hill, Ch. 6, Editors: J. Graeme, G. Tobey.

(15) G.B. CLAYTON "Operational amplifiers" 1974. Butterworths, Ch. 7, London.

(16) G.E.C. Transistor Manual, 1964. Ch. 14, G.E.C.

(17) G.E.C. Tunnel Diode Manual, 1961. G.E.C.

(18) L. KAVANAGH "A sub-miniature crystal controlled biological

transmitter" 1967. Digest of the 7th International Conf. on Medical and Biological Engineering, Stockholm.

(19) M. DIPROSE et al "A low power transmitter for electrocardiograms using tunnel diodes" 1976. Medical and Biological Engineering, Vol. 14, No. 3.

(20) SIGNETICS CORPORATION "Phase locked loops SE565/NE505" Nov. 1970. Information sheet.

(21) W.T. COCKING "Television receiving equipment" 1957. Iliffe & Sons Ltd., London, Ch. 23, 4th Edition.

CHAPTER 6

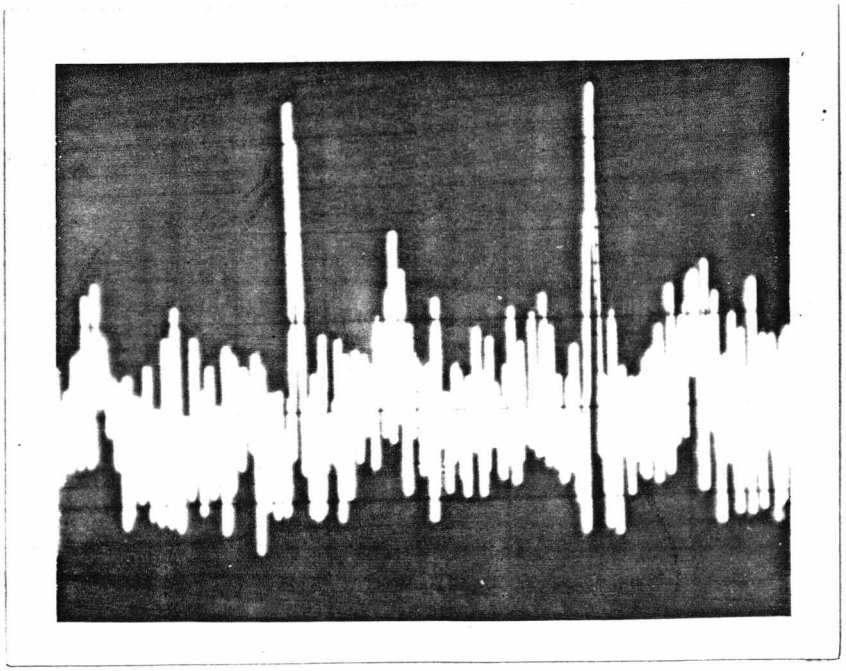
ELECTRODES

6.1 INTRODUCTION

In discussing the advantage of telemetry in Chapter 5, *Fig. (27)* showed the noise and artefact present on an ECG recording whilst the patient was exercising. This type of problem is not confined to exercising patients, but also arises in wards[1] when the interference can trigger off false alarms, and also when trying to monitor the cardiac performance of subjects such as swimmers[2] and astronauts[3]. The artefact interferes considerably with the recordings, and often can make them unreadable even when only trying to obtain a heart rate recording and not a diagnostic ECG.

The interference can be traced to three main sources, muscle artefact, lead movements, and electrode movements. The first of these occurs when the signals generated by the active muscles of the moving subject are detected by the electrodes as in *Fig. (1a)*. It is possible to filter out a lot of this noise, since its frequency content lies in the range 20Hz to 2500Hz[4] *Fig. (1b)* - which is mostly outside the required ECG bandwidth. It is also possible to place the electrodes to minimise the detected muscle noise[5].

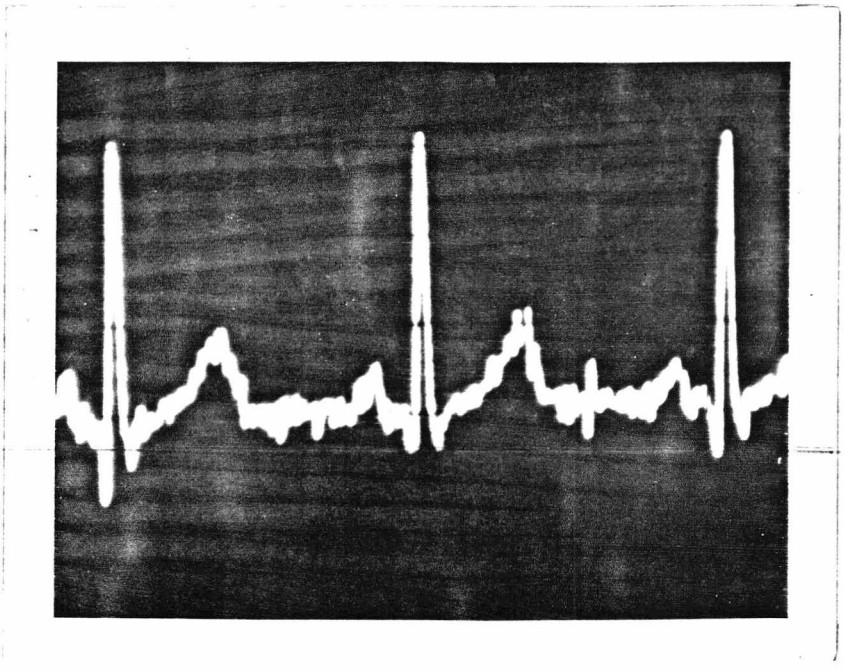
The second source of noise is that generated in the cable itself whenever it is flexed. This arises because the outer conductor in screened cable does not always make proper contact with the dielectric surrounding the inner conductor[6]. This allows small amounts of static electricity to collect due to friction or cable flexing. These are discharged when the outer conductor is pressed against them when the cable



0.2volts/cm

0.2secs/cm

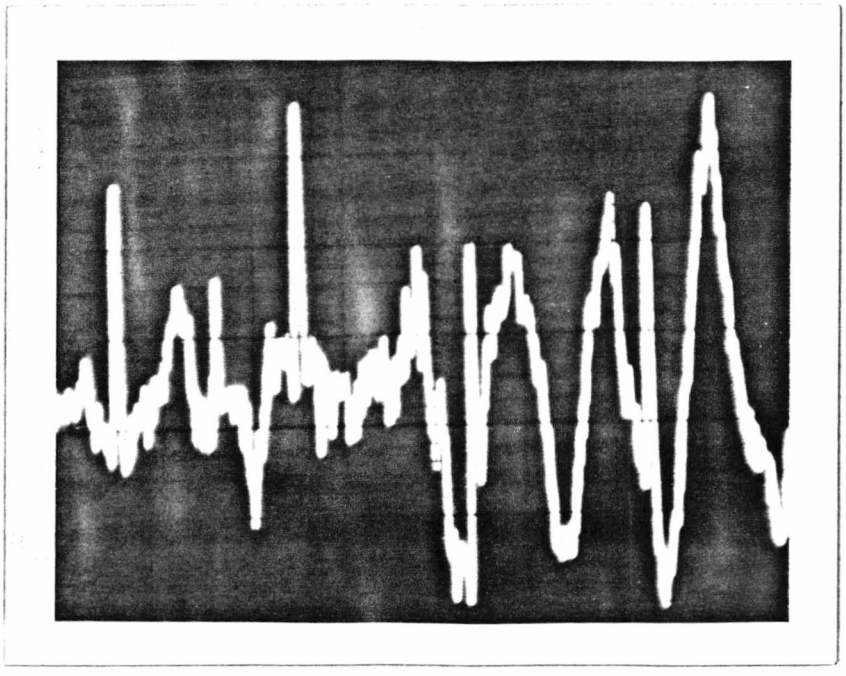
FIG.(1a) E.C.G. with muscle noise B/W 10kHz



0.2volts/cm

0.2secs/cm

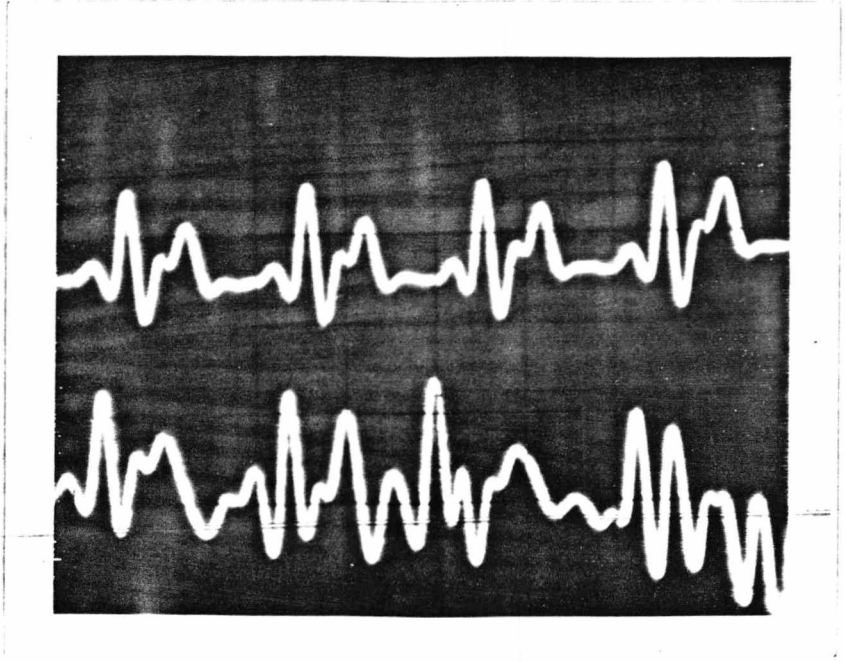
FIG.(1b) The same E.C.G. but with the B/W at 100Hz



0.2volts/cm

0.2secs/cm

FIG.(2a) Tugging at electrode B/W 100Hz



0.2volts/cm

0.2secs/cm

FIG.(2b) Top, E.C.G. with B/W 10Hz bottom. tugging at electrode B/W 10Hz

moves, and this gives rise to interference signals. This is best removed by judicious placing and support of the electrode lead-out wires since the frequencies it generates can often be in the same range as the ECG *(Fig. (2))* and thus are beyond filtering.

The third source - that of the noise produced by electrode movement also cannot be filtered out and is the most difficult to remove of the three[7]. Whenever the patient moves, then the electrode is vibrated or stressed, and this movement causes the artefact. Apart from direct patient stimulus, the electrode can also be moved by its lead wire jerking or pulling on it.

Since the first two modes of artefact production can be adequately dealt with - this leaves the third mode - and a lot of work has been done by trying to formulate new types of interference free electrodes[8-11].

By its nature an electrode on a patient is like an electrochemical cell - or strictly a half cell i.e., it is a metal in contact with an electrolyte, and changes an ionic charge flow to an electronic one[12]. Much effort has gone into understanding the electrode cells and their behaviour for both active and passive types[13-17], and into the stabilisation of the electrode skin interface since it is the disturbance of this that causes the artefact.

This, and the following chapter, investigates a new approach to the problem which is more electronic than electrochemical. It is proposed to use very low input impedance amplifiers for the ECG signals, and although this normally leads to severe distortion of the signal this is not a necessary conclusion if correct compensation networks are used. It is proposed that the low input impedance will load

the half cells of the electrodes so that it will tend to dampen any generated potential differences that may arise through movement of the system. The relationships between electrode and amplifier characteristics are investigated and it can be shown that having a low input impedance (e.g. 10KΩ) has no effect upon the common mode rejection ratio or signal-to-noise ratios, and with a single capacitor for compensation, can have little effect upon the shape of the ECG complex.

6.2 CELLS AND ELECTRODES

6.2a Electrochemical Cells

When a soluble metal such as silver or copper is placed into an electrolytic solution several processes occur. Ions leave the metal and go into the solution and at the same time ions already in the solution tend to be deposited on the metal[18]. When the ions leave the metal they leave behind their electrons and eventually the value of this negative charge is of sufficient magnitude to stop any more leaving, and also to attract back some of those that are in the solution. The net result is that a layer of positive ions lies close to the metal surface *(Fig. (3))*[19]. In its turn, the layer of positive ions attracts negative ions to it, and since they are prevented from touching by hydration[20], a double layer of charge is formed *(Fig. (3))*[21],[22]. This layer is named the Helmholtz or sometimes the Stern or Gouy layer depending upon the exact spatial distribution of the ions[23], and it is this distribution which gives the electrode its properties. The equilibrium that is reached is a dynamic one, with the rate of solution of ions equalling the rate of deposition. As the ions are charged, their movements to and fro

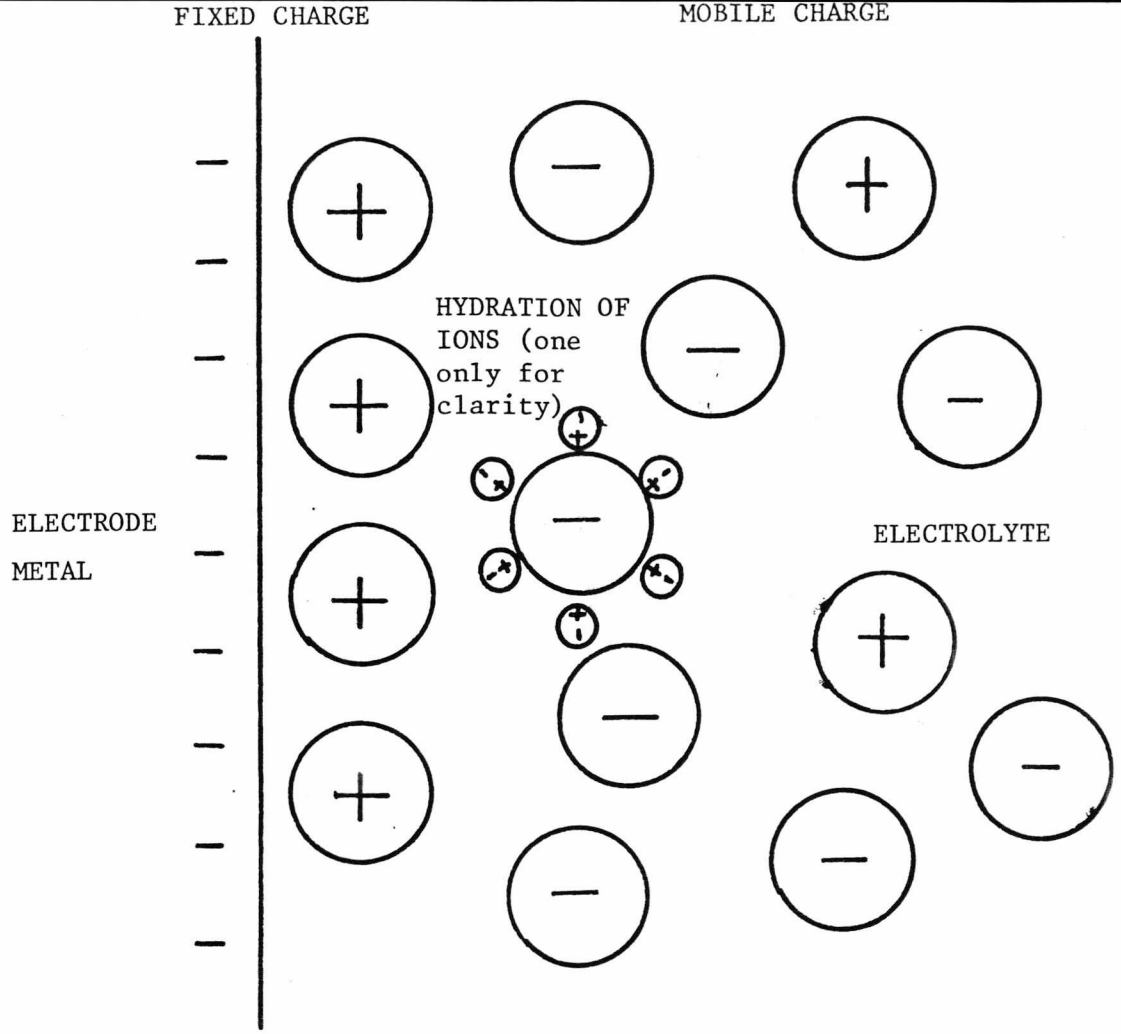


FIG. (3) The formation of the double layer

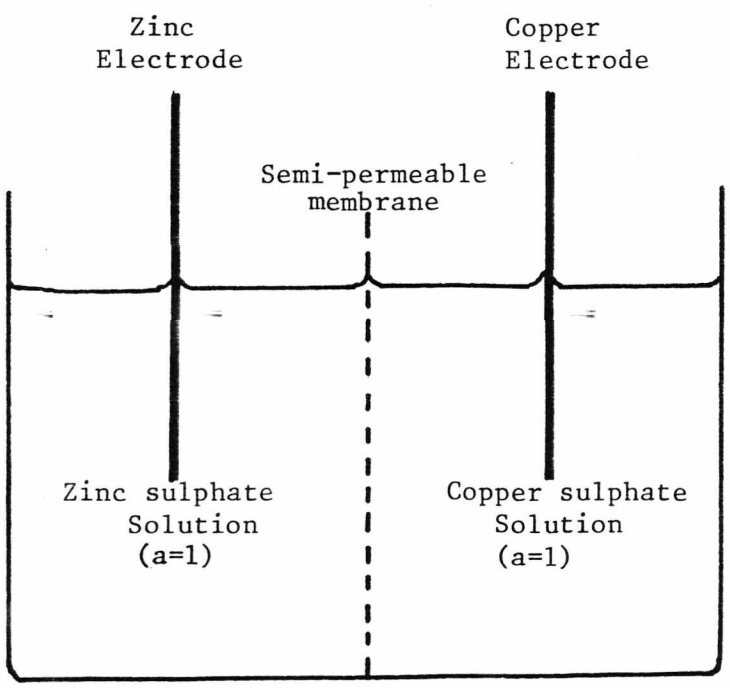


FIG. (4) A "Daniell" cell

constitute electric currents called exchange currents, with the net exchange current density at equilibrium being zero.

6.2b The Electrode Potential

The presence of a discontinuity between any two media means a potential energy difference[24] which in the case of electrodes in solution supports the double layer of ions at the interface. By considering the balance of electrical and diffusive forces, Nernst[25] derived an equation for the e.m.f. of the cell formed by a metal in an electrolyte, e.g. for the reaction

$$M \rightleftarrows m^+ + e^-$$

The e.m.f. $E = E_o + \frac{RT}{ZF} \ln Am^+$ (26)

E = e.m.f. of cell

E_o = a constant potential at 25°C

R = Universal Gas constant

T = temperature ° abs.

Z = valence of the ion

F = Faraday's constant

Am^+ = Activity of the m^+ ions in solution

The definition of E_o is difficult, as it is impossible to measure an isolated e.m.f. from an electrolytic cell. It is, therefore, used in conjunction with a standard hydrogen electrode as a reference. The difference between the two at 25°C is measured, and the hydrogen electrode is defined to be zero. Thus the measured potential is ascribed to the other electrode, and is called its standard electrode potential E_o, and each electrode is called a half cell. Table (1) gives a

list of some of the standard potentials for different half cells.

Half Cell Type	Cell Reaction	Standard Potential E_o (volts)
Al/Al^{3+}	$Al \rightleftharpoons Al^{3+} + 3e^-$	-1.66
Zn/Zn^{2+}	$Zn \rightleftharpoons Zn^{2+} + 2e^-$	-0.763
Fe/Fe^{2+}	$Fe \rightleftharpoons Fe^{2+} + 2e^-$	-0.440
Pb/Pb^{2+}	$Pb \rightleftharpoons Pb^{2+} + 2e^-$	-0.250
Ag/AgCl	$Ag+Cl^- \rightleftharpoons AgCl+e^-$	+0.2224
Cu/Cu^{2+}	$Cu \rightleftharpoons Cu^{2+} + 2e^-$	+0.337
Au/Au^{3+}	$Au \rightleftharpoons Au^{3+} + 3e^-$	+1.50

TABLE (1)

Half Cells and their Standard Potentials.

Thus, for example, a copper electrode in a copper sulphate solution:-

$$Cu \rightleftharpoons Cu^{2+} + 2e$$

and $$E = +0.337 + \frac{RT}{ZF} \ln A_{Cu}^{2+}.$$

In general, if a reaction is

$$\alpha A + \beta B = \gamma C + \delta D + Ze^-$$

then $$E = E_o + \frac{RT}{ZF} \ln \left(\frac{A_c^{\gamma} \cdot A_d^{\delta}}{A_a^{\gamma} \cdot A_b^{\beta}} \right) \qquad (26)$$

6.2c CONCENTRATION CELLS

Whole electrochemical cells called Galvanic cells are made

from two half cells, and are usually formed by using two different electrode materials in solutions of their own ions, and the solutions are separated by a semi-permeable membrane e.g., the Daniell Cell *(Fig. (4))*[27]

$$\text{e.g.} \quad Zn/Zn^{2+}_{a=1} \,||\, Cu^{2+}_{a=1}/Cu$$

the e.m.f. of the cell is by convention the e.m.f. of the righthand side minus the e.m.f. of the lefthand side

$$= \left(E_{o_1} + \frac{RT}{ZF} \ln A^{2+}_{Cu}\right) - \left(E_{o_2} + \frac{RT}{ZF} \ln A^{2+}_{Zn}\right)$$

$$= \left(+0.337 + \frac{RT}{2F} \ln(1)\right) - \left(-0.763 + \frac{RT}{2F} \ln(1)\right)$$

$$= +0.337 + 0.763$$

$$= \underline{+1.100 \text{ volts}}$$

It is possible, however, to make a Galvanic cell from similar electrode metals in similar solutions, but the latter are of different concentrations. Thus, the difference in potential depends upon the different electrolytic concentrations, and the whole is called a concentration cell[28].

Consider a concentration cell of electrode metal M in contact with its own ions in solutions of concentrations Cm_1^{2+} and Cm_2^{2+}, the subscripts 1 and 2 referring to the different cell halves *(Fig. 5)*

$\therefore$ since Activity = constant $\times$ Concentration

$$A = \gamma C$$

$\therefore$ the activities of the two solutions are

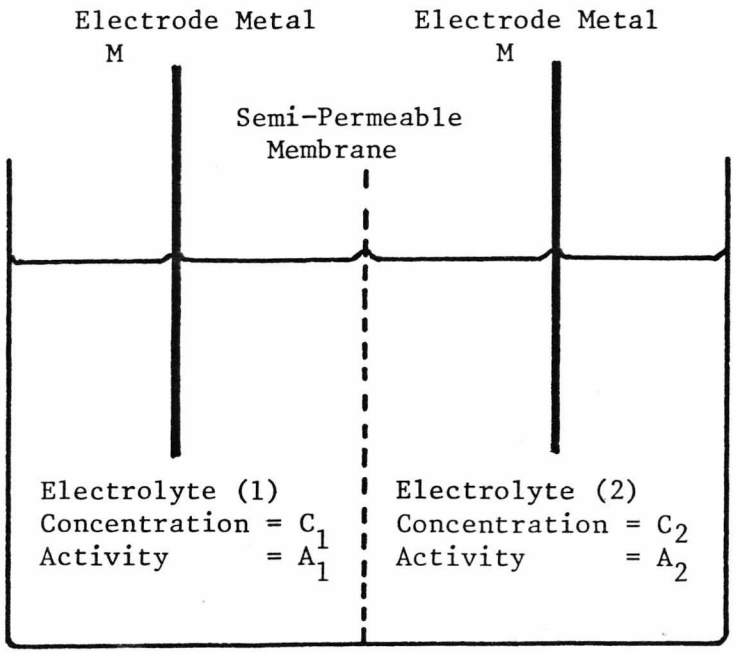


FIG. (5) A concentration cell

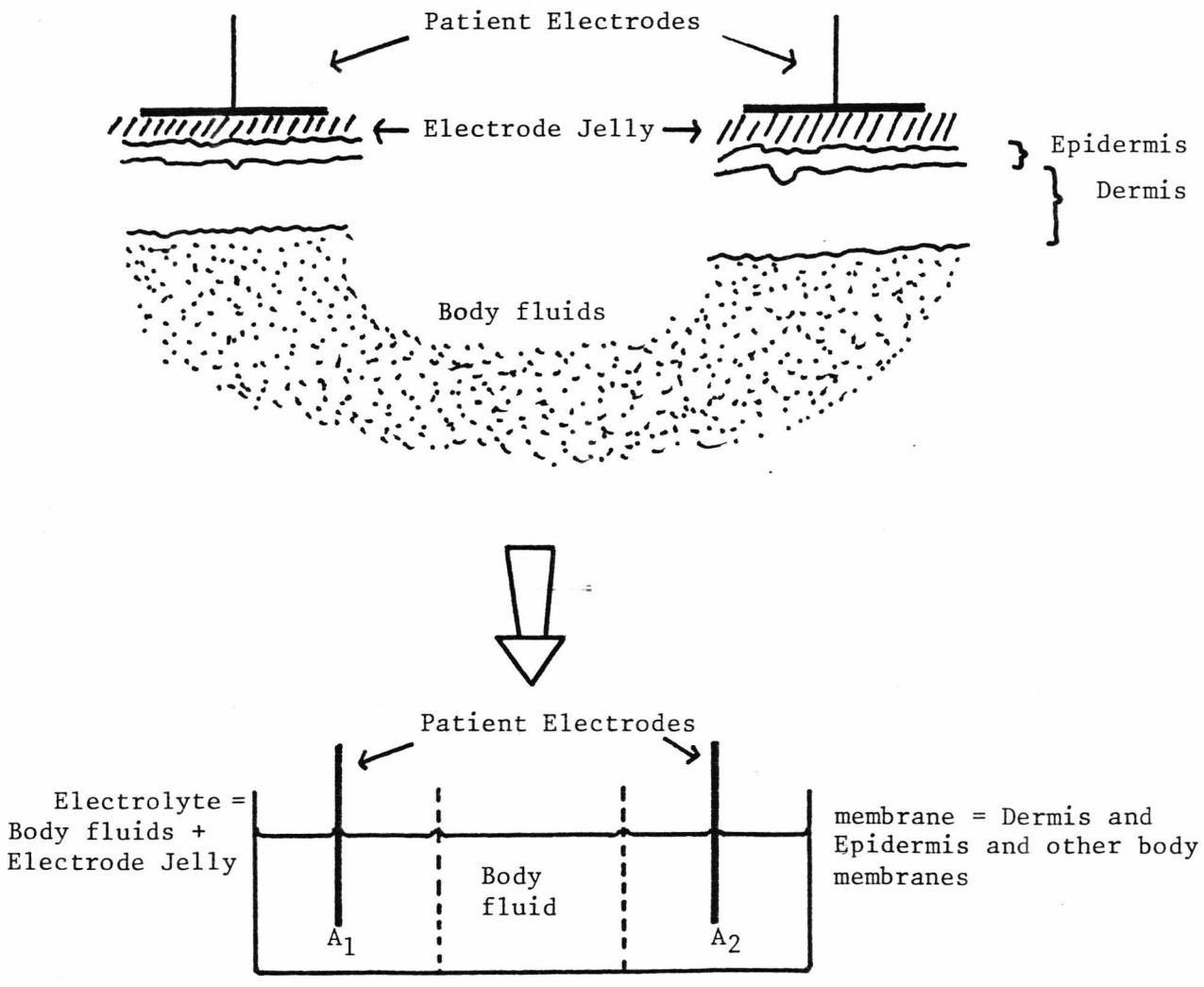


FIG. (6) Two patient electrodes form a concentration cell.

$$Am_1^{z+} = \gamma_1 \, Cm_1^{z+}$$

$$Am_2^{z+} = \gamma_2 \, Cm_2^{z+}$$

The electrode reaction is

$$M \rightleftharpoons m^{z+} + Ze^-$$

$\therefore$ for the first electrode

$$E_1 = E_{o_1} + \frac{RT}{ZF} \ln Am_1^{z+}$$

and for the second electrode

$$E_2 = E_{o_2} + \frac{RT}{ZF} \ln Am_2^{z+}$$

The cell potential $= E_2 - E_1$

$$= E_{o_1} + \frac{RT}{ZF} \ln Am_1^{z+} - E_{o_2} + \frac{RT}{ZF} \ln Am_2^{z+}$$

Since $E_{o_1} = E_{o_2}$

$$= \frac{RT}{ZF} \ln \frac{Am_1^{z+}}{Am_2^{z+}} \quad ,$$

i.e. the cell potential depends upon the concentration difference of the electrolytes[28], due to the previous definition of activity.

6.2d Recording Electrodes

When a bioelectric event such as an ECG is being recorded, electrodes are used to detect the events. Most electrodes in common use consist of flat plates of metal of varying sizes

used with an electrode jelly of some kind[29] and placed onto the skin at an appropriate anatomical position. It can be seen then that biological recording electrodes are really half cells with the metal plate as the half cell electrode and the electrode jelly and consequent saline solutions from perspiration, acting as the half cell electrolyte. An electrical double layer is formed under the recording electrode just the same as in the half cell, and this is what gives rise to the movement artefact. The double layer is measured in ionic dimensions and thus is very thin and easily disturbed. Since the structure of the double layer determines the potential, then mechanical movement of the electrode or of the underlying skin or vibration, will deform the distribution of the ions, and give rise to varying potentials - the movement artefact.

In practice at least two recording electrodes are used and the difference in potential is amplified and displayed. Since the two electrodes are of the same material, then this situation is like that of a concentration cell, *Fig. (6)*. Since it is unlikely that identical physical conditions will exist underneath each cell, then the activities of the electrolytes will be different and a potential difference will exist of the form

$$E = \frac{RT}{ZF} \ln \frac{Am_1^{z+}}{Am_2^{z+}}$$

It is this potential difference that is known as the d.c. offset potential. In practical systems this can be up to ± 100mV and is unpredictable and variable. This is not surprising, since it is impossible to precisely determine the conditions in the electrolytes.

Moving one or both of the electrodes provokes changes in the potential differences. As this is a potential difference between the electrodes then this is amplified as if it were a signal, and the normal ECG becomes lost completely amongst the fluctuating offset potential. The biological signals are detected by a similar mechanism. When the event occurs e.g. ventricular contraction, the generated electrical forces cause changes in ionic strengths to propagate throughout the body. When they reach the electrodes, the activities of the adjacent electrolytes are altered in sympathy with the original cardiac event, and so potential differences are established and amplified.

6.2e Silver/Silver Chloride Cells

Another type of electrode that is finding increasing use in medical recording is the silver/silver chloride electrode. In this system a silver electrode is in contact with a layer of silver chloride which is, in turn, in contact with an electrolytic solution containing chloride ions. This removes the direct silver/electrolyte interface and thus introduces a measure of stability when the electrode is mechanically stimulated. The electrode reaction is:-

$$\underset{\text{solid}}{Ag/AgCl} \rightleftharpoons Ag^{+} + Cl^{-} \qquad (30)$$

The dissociation rate of silver chloride is constant and at equilibrium must equal the rate of association. Now the rate of association is equal to the concentrations of ion species present

$$Ks = A_{Ag^{+}} \cdot A_{Cl^{-}}$$

where Ks = solubility constant

and A_{Ag^+}, A_{Cl^-} are the activities of the ions in the electrolyte.

Now, the dissociation rate does not change with the fact that chlorine ions are present in the electrolyte, and since the association product is a constant, then the activity of the silver ions must decrease accordingly.

The reaction $Ag \leftrightharpoons Ag^+ + e^-$ at the silver surface

and $$E = E_o + \frac{RT}{ZF} \ln A_{Ag^+}$$

and since $$A_{Ag^+} = \frac{Ks}{A_{Cl^-}} \quad \text{and} \quad Z = 1$$

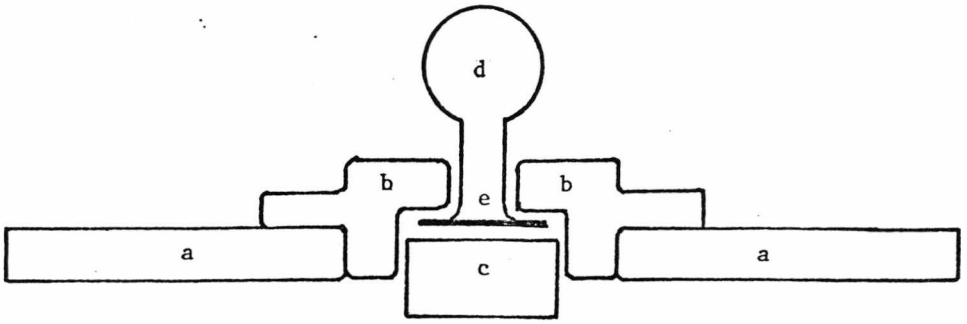
$$E = E_o + \frac{RT}{F} \ln Ks - \frac{RT}{F} \ln A_{Cl^-} .$$

Since $Ks = 1.78 \times 10^{-10}$ at 25^oC

$$= 0.2224 - \frac{RT}{F} \ln A_{Cl^-}$$

i.e., the electrode potential is solely a function of the chlorine ion activity. This type of electrode is one of the most stable, reproducible and convenient there is, next to the hydrogen electrode[30].

In its use as an ECG electrode, it is made from a small disc that is silver plated and then chlorided or else from a sintered disc of silver and silver chloride powders[8]. This is in contact with a small piece of sponge containing the electrolytic jelly, *(Fig. 7)*. The whole is held to the body by an adhesive pad. These electrodes are reliable, and have typical d.c. offset voltages of ± 5mV. Since the electrodes are mass produced it is likely that the difference in potential arises, not so much from the differences in the electrodes as before, but from the effects of the liquid junction formed by the electrolytic jelly and the body fluids. This latter



a = adhesive ring for contact to patient

b = plastic body of electrode

c = foam pad, soaked in an electrolytic jelly containing silver chloride

d = silver plated contact stud

e = silver plated electrode disc

FIG. (7) Section through a silver/silver chloride patient electrode

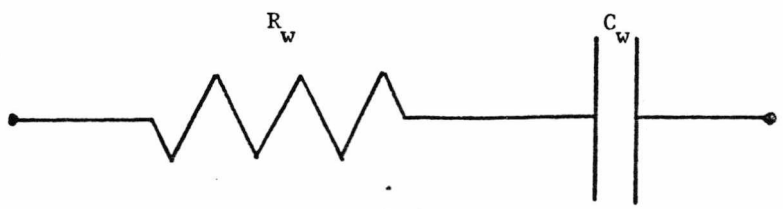


FIG. (8) Warburg's series equivalent circuit of an electrode

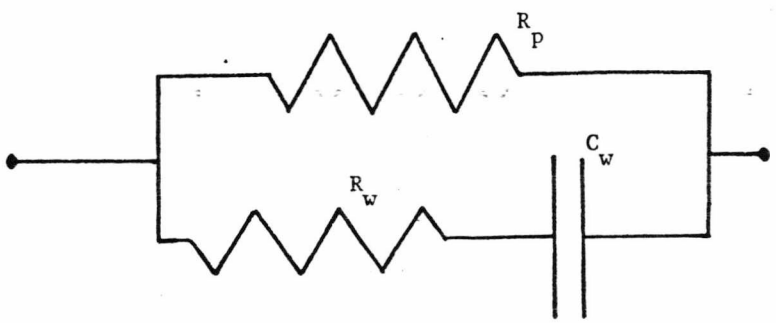


FIG. (9) A parallel resistor added to the circuit of Fig. 8 to account for conduction at D.C.

junction is unpredictable, but liquid junctions in general only have low potential differences, and this reflected as a small d.c. offset voltage. Since the electrode potentials are a function of chlorine ion activity, then any imbalance in this, caused by cardiac or other bioelectric events, causes a signal to be recorded from between the electrodes.

6.3 ELECTRODE IMPEDANCE AND EQUIVALENT CIRCUITS

6.3a The Equivalent Circuit

In investigating the characteristics of electrodes when recording bioelectric events, it is useful to use an equivalent, electrical circuit. Warburg suggested a simple series circuit of a resistor and capacitor *(Fig. 8)* after experimental and theoretical investigations of the diffusion properties of the ions at the metal/electrolyte interface[31]. This was subsequently modified by the addition of a parallel resistor *(Fig. 9)* to allow for the passage of current at d.c., and an even more comprehensive circuit is shown in *Fig. 10*[32]. R_E is the resistance of the bulk of the electrolyte and R_{CT} is the charge transfer resistance. The latter is the effective resistance shown when the metal ions actually leave the metal and go into solution. R_W and C_W are the Warburg resistance and capacitance respectively (transformed from the series equivalent to the parallel equivalent). These arise because of the concentration gradients that exists (of metal ions) just around the electrode[31]. The flow of current is limited by the diffusion rates of these areas, and this gives rise to an impedance - the Warburg impedance. C_H is called the incremental capacitance and is generated by the presence of the two layers of charge separated by a small distance.

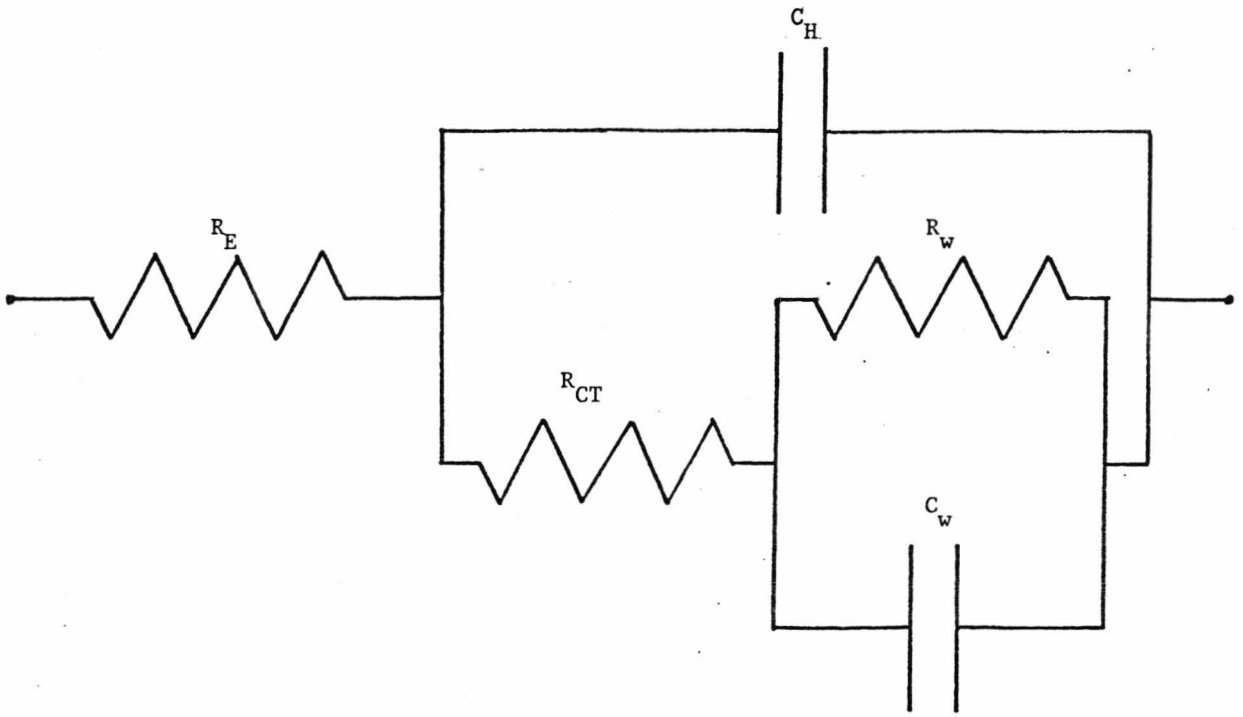


FIG. (10) Complete equivalent circuit of an electrode

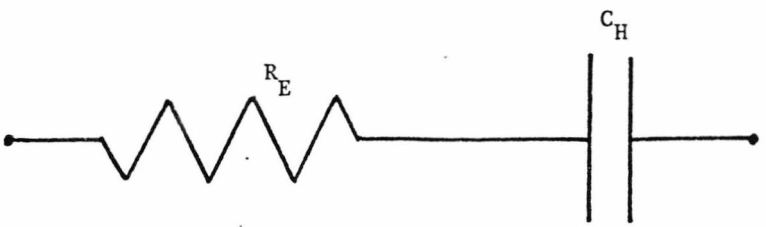


FIG. (11) High frequency approximation to the circuit

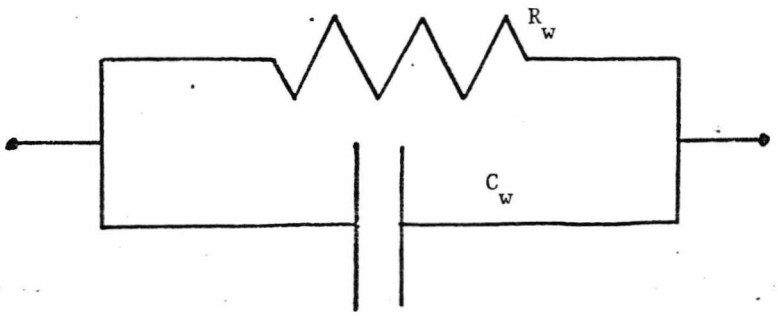


FIG. (12) Low frequency approximation to the circuit

Fig. (11) and Fig. (12) show the circuits at high and low (<1 KHz) frequencies respectively[32], and since the ECGs frequency content extends to only 100 Hz, then the circuit of *Fig. (12)* is that most commonly used. Typical values for different types of electrodes are given in Table (2)[33].

Type of Electrode	R_W(kΩ)	C_W(μF)
Metal Plate Limb Electrodes	3.35	0.58
Multipoint Limb Electrodes	7.33	0.128
Plastic Cup Electrodes	2.59	0.74
Spray-on Chest Electrodes	52.6	0.124

(measured at 1 KHz)

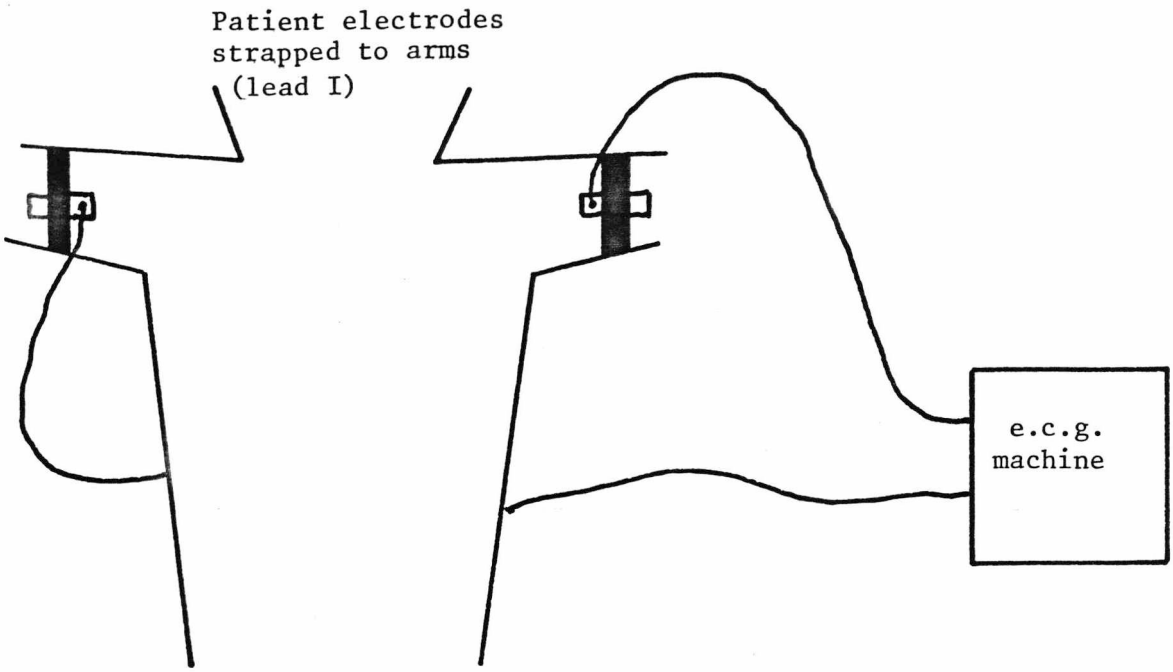
TABLE (2)

The diagram of *Fig. (13)* shows the complete equivalent circuit representing the two recording electrodes connected to the limbs, including the body resistance R_B, and the biological generator.

6.3b The Variation of Parameters with Frequency

It has been shown that the values assigned to the components of the equivalent circuits are not constant when measured, but are dependent upon frequency[34],[35]. Measurements made upon many types of electrode have shown a general dependance on the frequency of the impedance components of the form:-

$$R \alpha f^{-\alpha} \quad \text{and} \quad C \alpha f^{-\alpha}$$



This gives the following equivalent circuit

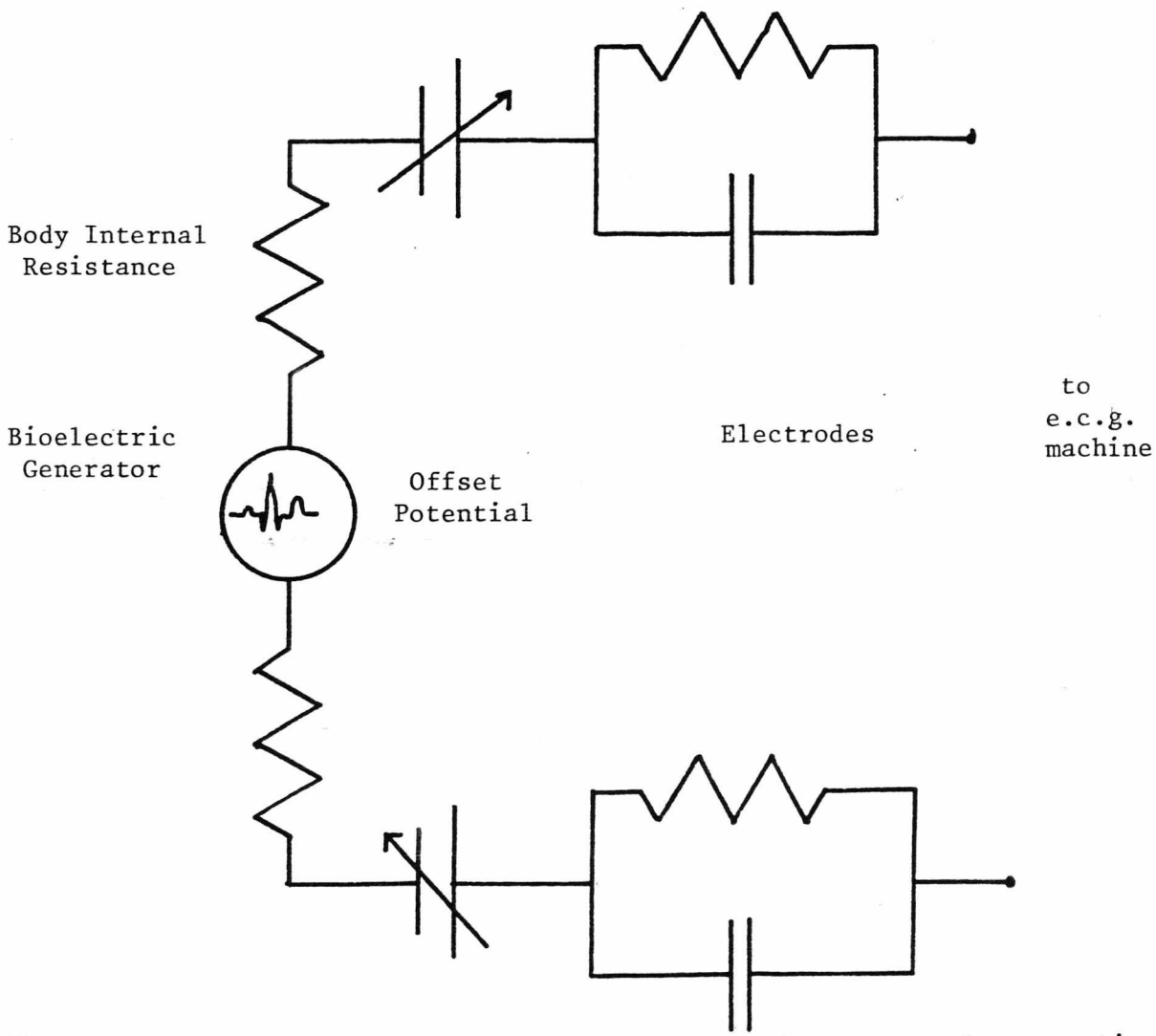


FIG. (13) The equivalent circuit of two electrodes connected to a patient

The constant α has a value which is nominally 0.5[34], but values have been found from 0.3 to 0.8 depending upon the particular type of electrode system used. Table (3) shows some examples of the constant α for the capacitance of several electrode types.

Electrode Type	Co.($\mu F/cm^2$)	α
Pt.Ir/Black 0.9% Saline	8,619	0.299
Pt.Black/0.9% Saline	4,950	0.366
Ag/0.9% Saline	359	0.328
Pt.Ir/0.9% Saline	2,696	0.79
Stainless steel/0.9% Saline	160.8	0.525
Pt/0.025NHCl	322	0.495
Pt/1.46NH_2SO_4	2,642	0.58
Au/1.1% H_2SO_4	2,873	0.541

TABLE (3)

Warburg showed experimentally that the value of the constant was 0.5, and also that the value of the resistance was equal to the value of the capacitative reactance, and this has since been confirmed by other authors. Since these values are equal, then the phase difference between voltage and current should be constant over a wide frequency range and this also is found to be so[36]. Fricke showed that

$\delta = \frac{(m+1)\pi}{2}$ where m is an integer

δ = the phase difference

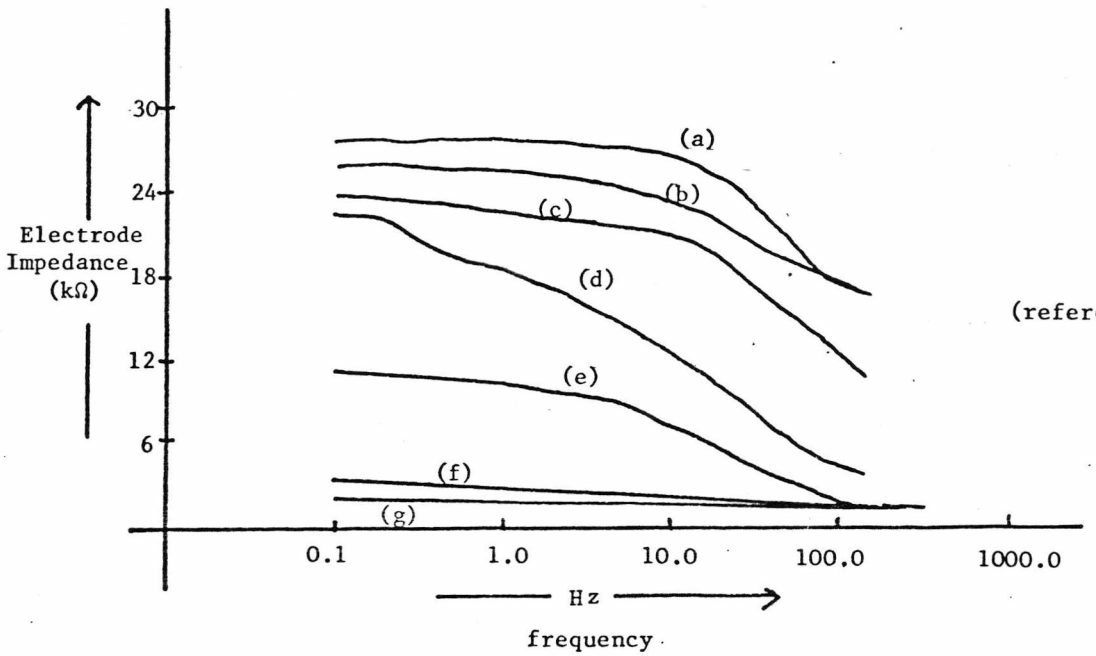
and this gives a phase value of 45^{o}.

Most of these measurements have been performed at 10 Hz and above, mostly because of the measuring bridges being used reaching their limits of linearity at the low frequencies. Hill & Khandphur, however, obtained measurements of impedance down to 0.1 Hz, and they found that it was constant up to 10 Hz and then dropped away *(Fig. 14)*[37].

Another parameter which can cause the components to vary is the current density passing through the electrodes. As this is increased beyond a certain limit (usually about $1ma/cm^2$)[38],[39], then the capacitance rises, and the resistance decreases. This can be a considerable problem when using electrodes for stimulating tissue[40], but when recording signals, the resulting current densities can be measured in $nanoamps/cm^2$ or $microamps/cm^2$, and so this consideration does not, then, apply.

6.3c The Magnitude of the Electrode Impedance

Beyond saying that the impedance of electrodes decreases with increasing frequency, it is difficult to quantitatively describe them any further. It cannot be said that electrode type A will have RΩ impedance when used for patient recording. Impedances for metal plate electrodes have been found to vary from 400Ω to 100KΩ at times on the same person at different sites[41]. Benson & Pipberger[42] published their results on skin electrode impedances and from these the extent of the range of values can be seen. Table (4) shows some of the results they obtained from square shaped silver alloy plates



(reference 37)

FIG. (14a) The variation of electrode impedance over low frequencies, for 7 electrode types

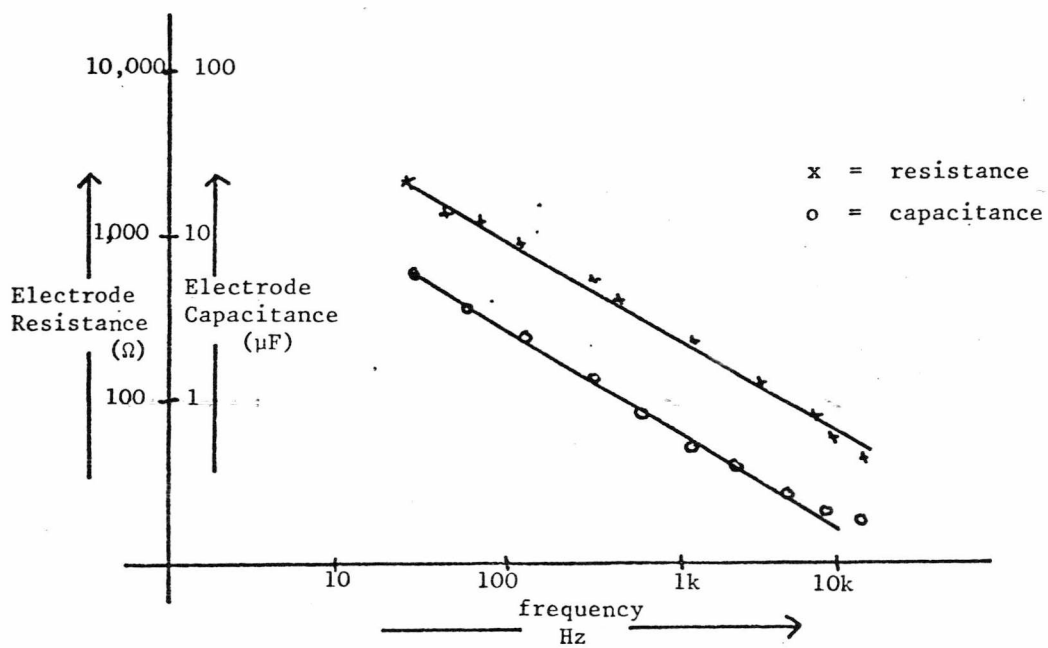


FIG. (14b) The variation of Impedance Components with frequency

(2.5 cms per side) measured 15 minutes after application at a frequency of 10 Hz. It can be seen that little or no correlation exists between the values of the impedances at the same sites on different patients or by comparing the sets of sites on any two or more patients.

Patient Number	Electrode Position			
	E	H	F	I
1	5	0	7	3
2	0	0	33	33
3	47	2	24	30
4	11	3	101	21
5	47	16	3	19
6	16	3	16	36
7	17	1	43	1
8	9	3	9	2
9	4	4	54	45
10	32	0	8	12

Electrode Positions E, F, H and I are for the Standard FRANK leads.

All impedance values are in KΩ and were measured at 10 Hz.

TABLE (4)

The Variation of Electrode Impedance with Site and Patients.

Similar work by Almasi & Schmitt showed the same type of results[41] - that the magnitude of the impedances measured on different parts of the body with identical electrodes is very variable. They also concluded that between 1 and 10 Hz the impedances measured would exceed 100KΩ for 2% of the population. This result has considerable influence on the

design of the amplifiers used since their input impedances are normally required to be considerably in excess of those of the electrodes.

6.4 AMPLIFIER INPUT IMPEDANCE

6.4a Signal Distortion and Amplifier Impedance

At the close of the previous section (6.3c) it was stated that the electrode impedance must be much less than the input impedance of the following amplifier. This is due to two reasons; the first is that the signal becomes distorted if too low an input impedance is used, and secondly the common mode rejection ratio (CMRR) (the ability of a differential amplifier to reject a signal which is common to both inputs) becomes adversely affected.

Geddes & Baker have demonstrated[43],[44] the deterioration in the ECG which occurs when the input impedance of an amplifier is progressively reduced by placing successively lower values of resistor across the input *(Fig. 15)*.

The control amplifier impedance that was used was 4.4MΩ, and the ECG (canine in this case) was observed. Then different values of resistance were placed across the input, and the resulting ECGs recorded and compared with the control. As the resistances became smaller, not only did the amplitude of the R wave decrease, but also the QRS complex changed from being monophasic to biphasic, *Fig. (15)*. The P and T waves gradually reduced as well, until at 10KΩ they, too, were biphasic and one half of their original size. It was also found that the size of the electrode had an important influence on the result. The smaller the electrode, then the sooner these abnormalities occur. For needle electrodes of 1 sq.mm. area the distortion

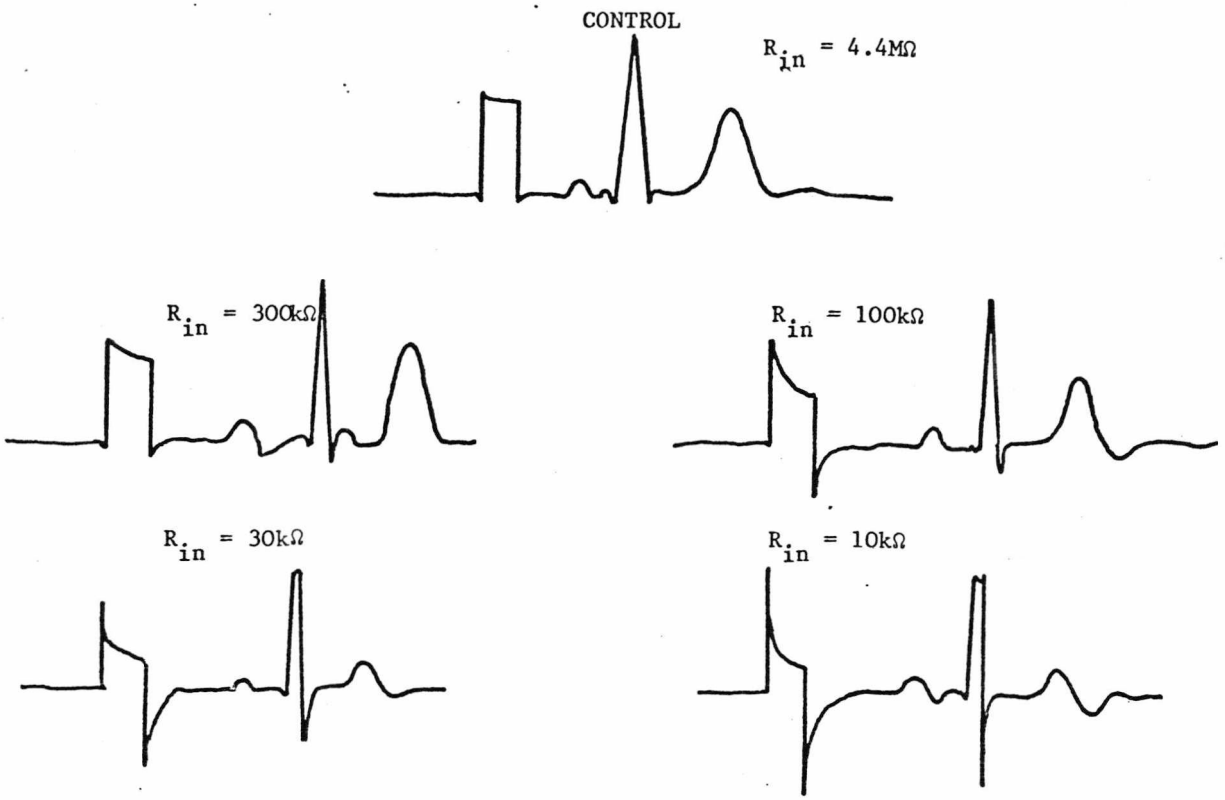


FIG. (15) The distortion of an e.c.g. signal as amplifier input impedance is lowered

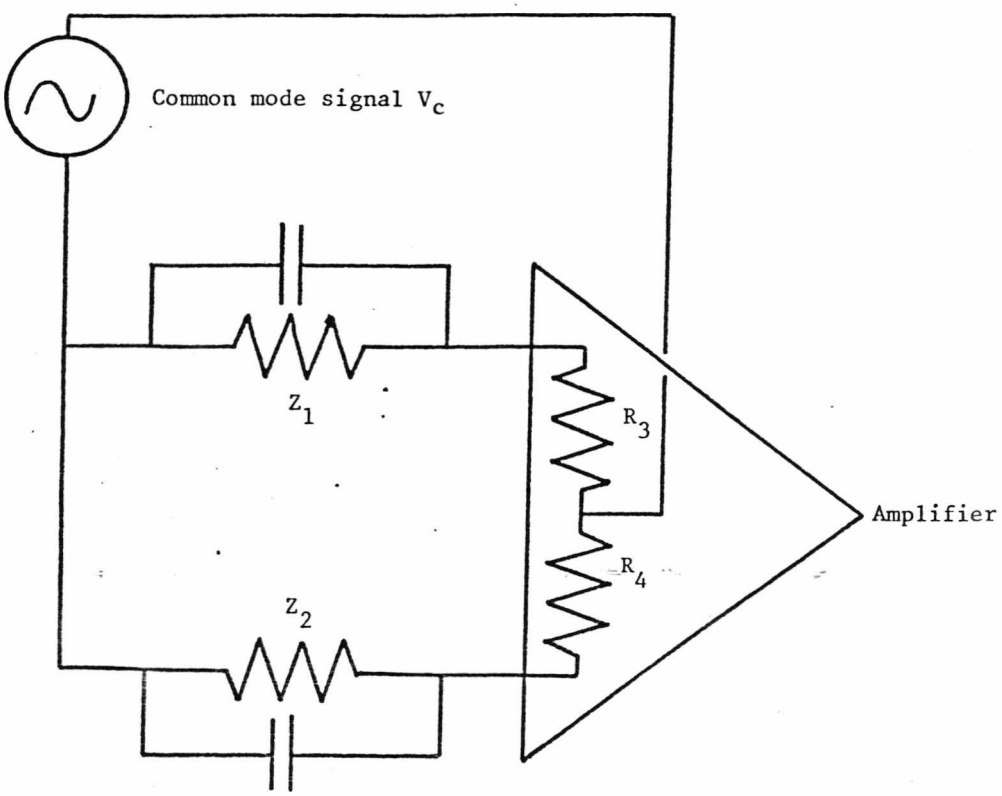


FIG. (16) The common mode circuit of an amplifier

evident at 10KΩ, was not equalled by the 76 sq.mm. electrodes until a resistance of 100Ω was placed across the input[43].

Geddes et al[45] had performed a similar series of experiments in 1968, in which the diminution of R, P and T waves of different electrodes and sizes with successively lower input resistances had been plotted.

From all the work it is concluded that, since not only a change of amplitude but also significant changes in shape (consistent with differentiation[43]) occurred with low input resistances, then, for clinical use, the input impedance of an ECG amplifier must be very high. The American Heart Association stipulate that it must be in excess of 500KΩ(ref. (1), Ch.4) and in practice values of 2 to 10 times this are usually encountered. Thus, there exists a wide margin of safety so that amplifiers can cope with any electrode type and size, placed at any position, without distortion of the bioelectric signal.

6.4b CMRR and Amplifier Impedance

If, in measuring the ECG, the two patient electrodes were identical, and the input stages of the differential amplifier were also identical to each other, then any common signal that appears would not be amplified. This is illustrated in *Fig. (16)*. When $Z_1 = Z_2$ and $R_3 = R_4$, then V_{out} of the amplifier is zero for signals of any frequency appearing at the input. In practice, however, although the amplifier inputs can be built to match extremely well, there is usually an impedance mismatch between the patient electrodes to a greater or lesser extent. This mismatch means that unequal signals appear across each amplifier input when the system is driven by a common

voltage, and so an output appears. Cobbold has illustrated this[46], and he shows that the CMRR depends upon the factor $\frac{Z_1-Z_2}{R_3}$. With $R_3 = R_4 = 10M\Omega$, and $Z_1 = Z_2 = 10K\Omega$, then a practical CMRR is 100 dBs. If there is a mismatch of 20% between the electrodes then the factor drops to 74 dBs. If R_3 is only $1M\Omega$, however, then the same mismatch leads to a CMRR of 54 dBs, and so from these examples it can be seen that the input impedance must be kept as high as possible to ensure that good rejection of common signals is obtained and as little interference as possible is detected[47].

6.5 LOW INPUT IMPEDANCE AMPLIFIERS

6.5a Proposition of Low Input Impedances

In the introduction to this chapter, the problems due to the offset potential from electrode pairs were outlined, and the ensuing sections showed the origins of the offset, and then investigated the a.c. characteristics of the electrodes themselves, and the necessary input conditions of the following amplifier. Although it is possible to minimise the artefact produced when recording ECGs by inhibiting mechanical disturbance of the system, it cannot be eliminated altogether and interference can still be generated. Once the offset potential is disturbed it may take some considerable time to recover once[48] again - a complication which may lead to serious consequences as in the case when a manufactured brand of ECG electrodes used on a patient, took several minutes to stabilise after defibrillation[49].

It is proposed to place a very low resistance of $10K\Omega$ to $47K\Omega$ across the input of the amplifier to overcome the problem by inhibiting the formation of offset potentials. The

low resistance will allow larger currents than normal to flow through the concentration cell formed by the electrodes to equalise the electrolyte conditions as soon as possible and reduce any offset to zero. Should a mechanical disturbance occur thereby altering the electrolyte concentrations then the low resistances will allow rapid transfer of charge, and equalisation of potentials once more.

Strong[50] suggested that this approach may be used, but indicated values in the region of 2MΩ to discharge the offset potential but Huhta & Webster[47] examined this and found little value in it, as 2MΩ is still a large value of resistance, and for the reasons previously stated in 6.4, they did not want to use any lower values of resistance. They concluded that no real advantage could be gained by using 2MΩ, and it would be better to remain at the 10MΩ level if possible.

Further, if indirect, support for the proposal is given by Geddes[51] who points out that it is a practice of Physiologists to short together new electrodes in electrolyte before using them. This is found to have a good stabilising effect, both on the h.f. noise and electrode drift and much of the artefact.

It is impossible, of course, to operate an ECG recording with the electrodes completely shorted together, but using the values of Rin of 10KΩ-47KΩ it is hoped this may approximate the shorted condition for the offset, but allow a.c. signals still to be detected.

Even though 10KΩ is, perhaps, 1000 times smaller than normal, the currents that flow are still quite small. For the ECG signal, the peak R wave of 1mV across 10KΩ means a current of 10^{-7} amps, and since most ECG electrodes are greater than 1 sq.mm. in area, the current density is con-

siderably below the 1 ma/sq.cm. which has been found to be the limit of linearity, and so no distortion due to current density should be experienced.

6.5b The Effect of a Low Resistance on a Concentration Cell

It was shown in section 6.2d that when a pair of metal plate electrodes are placed on a patient they form a concentration cell. It has been proposed that a method of reducing the offset potential is to discharge the cell as quickly as possible with a low resistance. By considering the current flow through an external resistor across a concentration cell, it will be shown that the current falls away exponentially, and that the decay time constant is proportional to the external resistance - thus the smaller the resistance, the quicker the offset potential will decay.

Consider the concentration cell as shown in *Fig. (17)*. The e.m.f. E of a concentration cell is given by:

$$E = \frac{RT}{ZF} \ln \frac{a_1}{a_2}$$

and if the concentrations vary with time

$$E(t) = \frac{RT}{ZF} \ln \frac{a_1(t)}{a_2(t)} .$$

At time t = 0, switch S_1 is closed, and current I_o flows in the resistance Res. When the current flows, simultaneous reactions occur at the electrodes, equivalent to solute being transferred from one to the other. Thus the reactions mean that the concentrations are changed, and so the potential changes.

Now $a_1 = \gamma_1 C_1$

$a_2 = \gamma_2 C_2$

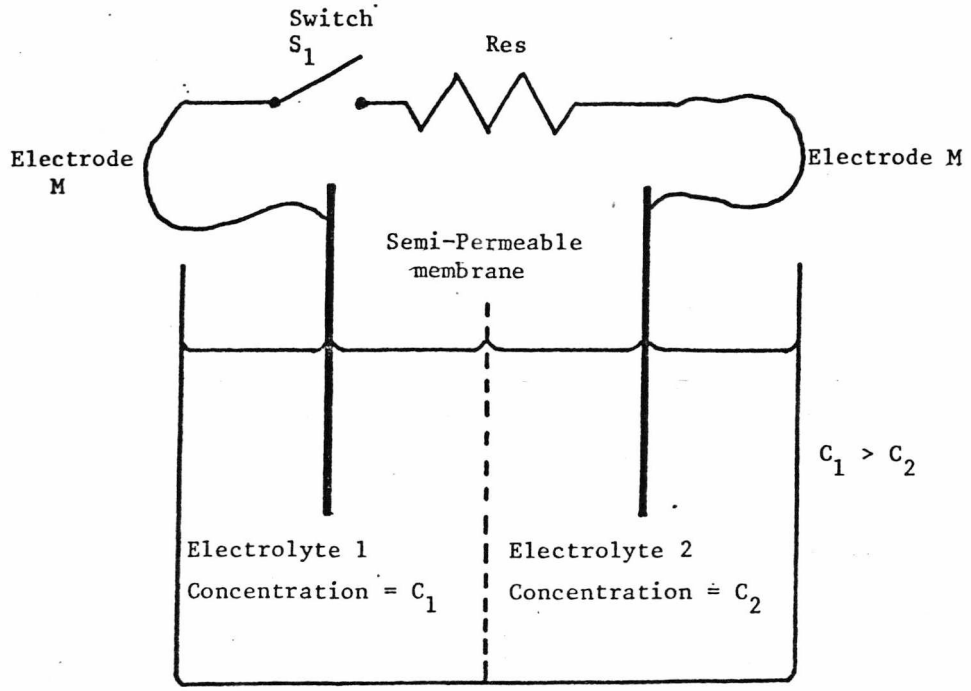


FIG. (17) The discharge of a concentration cell

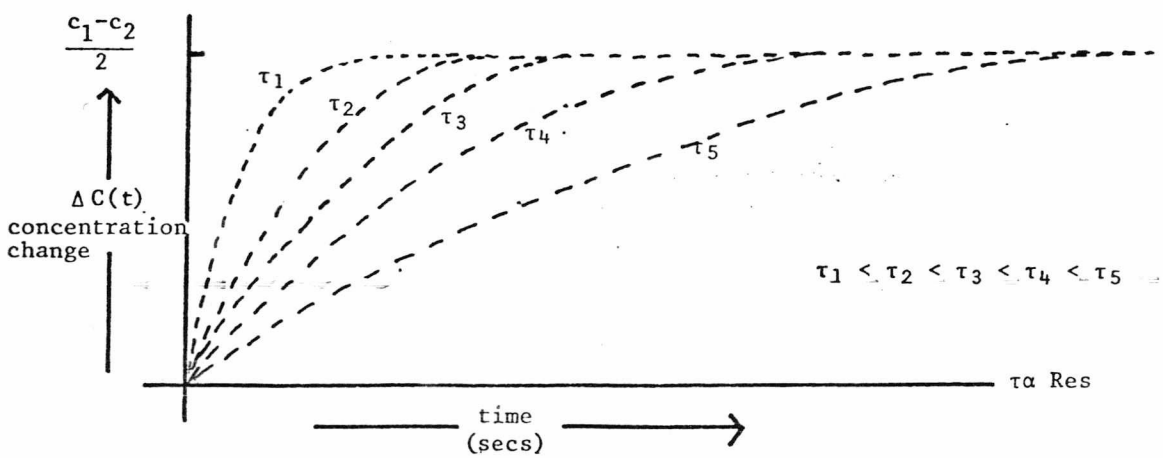


FIG. (18) The effect of different values of Res. upon the discharge of a concentration cell

and since solutions are almost the same $\gamma_1 = \gamma_2$

$$\therefore \frac{a_1}{a_2} = \frac{c_1}{c_2}$$

$$\therefore E = \frac{RT}{ZF} \ln \frac{c_1(t)}{c_2(t)} .$$

$\therefore$ For a change in current dI

$$dI = \frac{dE}{Res} \quad \text{(Ohm's Law for the resistor)}$$

and during a time dt

$$\frac{dI}{dt} = \frac{dE}{dt} \cdot \frac{1}{Res} .$$

Now $\frac{dE}{dt} = \frac{d}{dt} \left[\frac{RT}{ZF} \ln \frac{c_1(t)}{c_2(t)} \right]$.

Now if at t=0 $c_1(t) = c_1(o)$

$c_2(t) = c_2(o)$

and also that the concentration change $\Delta c \propto$ charge transferred

$$\therefore \Delta c(t) = K \times \text{charge transferred}$$

$$= K \int_o^t I(t).dt$$

These conditions are true if the current is carried across the membrane by charge carriers other than those around the electrode, as in the case of electrodes on a patient where the charge is carried by saline solution in the body.

$$\therefore c_1(t) = c_1(o) - \Delta c(t)$$

$$c_2(t) = c_2(o) + \Delta c(t)$$

$$\therefore \frac{dE}{dt} = \frac{RT}{ZF} \frac{d}{dt} \left[\ln \frac{c_1(o) - \Delta c(t)}{c_2(o) + \Delta c(t)} \right]$$

Assuming that $\frac{d}{dx} \ln \left(\frac{a(x)}{b(x)} \right) = \frac{b(x)}{a(x)} \left[\frac{b(x)a'(x) - a(x)b'(x)}{b(x)^2} \right]$

and also $\frac{d}{dt} \Delta c(t) = KI(t)$.

Then $$\frac{dE}{dt} = -\frac{RT}{ZF}\frac{c_2(o) + \Delta c(t)}{c_1(o) - \Delta c(t)} \times \frac{d}{dt}\Delta c(t) \cdot \frac{(c_1(o) + c_2(o))}{(c_2(o) + \Delta c(t))^2}$$

$$\frac{dE}{dt} = -\frac{RT}{ZF} K.I(t) \cdot \frac{[c_1(o) + c_2(o)]}{[c_1(o) - \Delta c(t)][c_2(o) + \Delta c(t)]}$$

Expanding the denominator and ignoring second order terms

$$\therefore \frac{dE}{dt} = -\frac{RT}{ZF} \cdot K.I(t) \frac{c_1(o) + c_2(o)}{c_1(o) \cdot c_2(o)}$$

$$\therefore \frac{dI}{dt} = -\frac{RT}{ZF\ Res} \cdot K.I(t) \cdot \frac{c_1(o) + c_2(o)}{c_1(o) \cdot c_2(o)} \cdot$$

$$\therefore \frac{dI}{I(t)} = -\underbrace{\frac{RT}{ZF\ Res} \cdot K \cdot \frac{c_1(o) + c_2(o)}{c_1(o) \cdot c_2(o)}}_{K_1} \cdot dt = -K_1 dt$$

$\therefore$ let a solution of this equation be

$$I(t) = I_o \exp^{-K_1(t)}$$

where $$I(o) = I\ t=0 = \frac{RT}{ZF\ Res} ln \frac{c_1(o)}{c_2(o)} \cdot$$

$$\therefore I(t) = \frac{RT}{ZF\ Res} ln \frac{c_1(o)}{c_2(o)} \cdot \exp\left(-\frac{RT}{ZF\ Res} K \cdot \frac{c_1(o) + c_2(o)}{c_1(o) \cdot c_2(o)} \cdot t\right)$$

in the form $I = I_o \exp -\frac{t}{\tau}$ where $K_1 = \frac{1}{\tau}$

then the time constant τ

$$\tau = \frac{ZF\ Res}{RT} \cdot \frac{1}{K} \cdot \frac{c_1(o) \cdot c_2(o)}{c_1(o) + c_2(o)} \cdot$$

i.e. the time constant is proportional to the external resistance, so the lower this is the smaller τ is.

Now charge transferred in period t = Q(t)

$$Q(t) = \int_o^t I(t).dt$$

Since $\int_o^t e^{-K_1 x} = -\frac{1}{K_1}\left[e^{-K_1 x}\right]_o^t$

$$Q(t) = -\frac{RT}{ZF\ R\phi s} \ln \frac{c_1(o)}{c_2(o)} \times \frac{ZF\ R\phi s}{R.T.K.} \frac{c_1(o).c_2(o)}{c_1(o)+c_2(o)} \left[e^{-K_1(t)}\right]_o^t$$

$$= -\frac{1}{K} \ln \frac{c_1(o)}{c_2(o)} \cdot \frac{c_1(o).c_2(o)}{c_1(o)+c_2(o)} \left[1 - e^{-t/\tau}\right]$$

$\therefore$ when $t=0$, $Q(o) = 0$

$t=\infty$ $\quad Q(\infty) = -\frac{1}{K} \ln \frac{c_1(o)}{c_2(o)} \cdot \frac{c_1(o).c_2(o)}{c_1(o)+c_2(o)}$

$\therefore$ Since $K.Q(t) = \Delta c(t)$

$$\therefore \Delta c(t) = \ln \frac{c_1(o)}{c_2(o)} \cdot \frac{c_1(o).c_2(o)}{c_1(o)+c_2(o)} \exp^{-t/\tau}$$

$\therefore$ The smaller τ, then the faster the reaction occurs initially, although the final result is always the same ($t=\infty$) irrespective of τ. Thus, the smaller the external resistance Res, then the smaller τ, and the sooner the electrode offset potential settles down after a disturbance *(Fig. (18))*. The resistance cannot be too small, however, otherwise the signal voltages which appear as electrolyte concentration changes under the electrodes will be balanced out too, and lost.

6.6 AN ANALYSIS OF THE EFFECT OF LOW INPUT IMPEDANCES ON AMPLIFIER CHARACTERISTICS

6.6a The Distortion of the ECG and Low Input Resistance

It has been shown that a low input resistance will reduce the effects of offset potential by discharging the concentra-

tion cell. The values of resistance used, however, are directly contrary to the conclusions reached by Geddes et al i.e., that high value resistances must be used to avoid distortion and poor common mode rejection. It will be shown in this section, however, that a low input impedance can be used, whilst avoiding the problems mentioned above. The equivalent circuit of Fig.(19a) is that of the measurement system used when determining how much distortion occurred. The electrodes are represented by a parallel RC networks and the added input resistance by R_i. To find the effect of R_i upon the output, the transfer function of the network must be found.

$$\frac{V_{out}}{V_{in}} = \frac{Z_i}{Z_a + Z_b + Z_i}$$

$$Z_a = R_1/1 + j\omega R_1 C_1 \qquad Z_b = R_2/1 + j\omega R_2 C_2 \ , \ Z_i = R_i$$

$$\therefore \frac{V_{out}}{V_{in}} = \frac{\Omega i}{R_i/1 + j\omega R_1 C_1 + R_2/1 + j\omega R_2 C_2 + R_i}$$

This equation can be expanded algebraically to give a quotient of two complex numbers. From this, the following can be found :-

when $\omega \to 0$

$$\left|\frac{V_{out}}{V_{in}}\right| = \frac{R_i}{R_1 + R_2 + R_1}$$

and $\phi = 0$

when $\omega \to \infty$

$$\frac{V_{out}}{V_{in}} = 1$$

$\phi = 0$

Fig.(20) shows the result in graphical form.

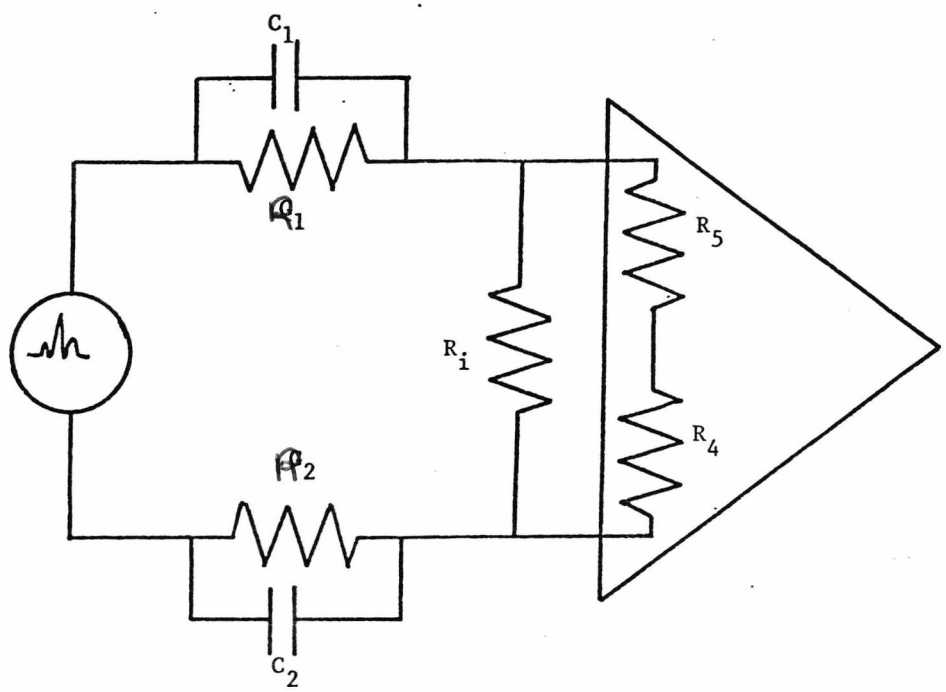


FIG. (19a) The circuit as used to determine the effect of low input resistance upon a signal

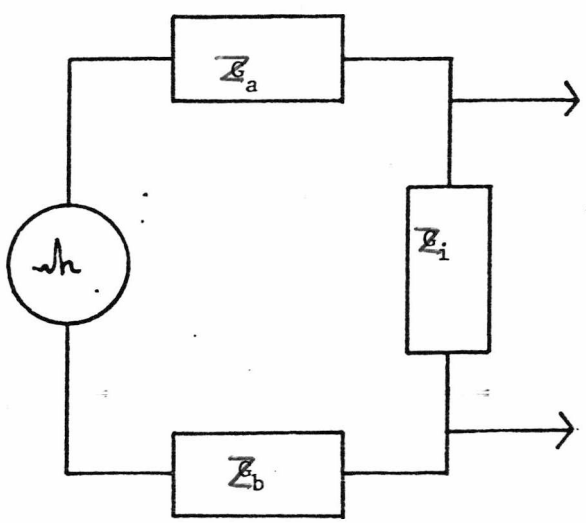


FIG. (19b)

Thus it can be seen that if R_i is very large and $R_i >> R_1$ or R_2 then the amplitude of the frequency response is unity, and there should be no phase shift. This is the condition recommended by Geddes et al. If, however, $R_i \lesssim R_1$ or R_2, then the response is modified, and shown in Fig.(20) i.e., the network behaves as a high pass filter. This explains the type of ECG distortion that was obtained in the experiments with low input resistances.

It was possible to linearise the response by the addition of an input capacitor C_i in parallel with R_i (Fig. (21a)).

Now from Fig. (21b)

$$\frac{V_{out}}{V_{in}} = \frac{Z_i}{Z_1 + Z_2 + Z_i}$$

but now $Z_i = \dfrac{R_i}{1 + j\omega R_i C_i}$

$$\therefore \frac{V_{out}}{V_{in}} = \frac{R_i / 1 + j\omega R_i C_i}{R_1/1 + j\omega R_1 C_1 + R_2/1 + j\omega R_2 C_2 + R_i/1 + j\omega R_i C_i}$$

This can be expanded algebraically into a quotient of two complex numbers, and using the concept of time constants.

$\tau_1 = R_1 C_1$, $\tau_2 = R_2 C_2$, $\tau_i = R_i C_i$ gives

$$\frac{V_{out}}{V_{in}} = \frac{R_i (1+j\omega\tau_1)(1+j\omega\tau_2)}{R_1 (1+j\omega\tau_2)(1+j\omega\tau_i) + R_2 (1+j\omega\tau_1)(1+j\omega\tau_i) + R_i (1+j\omega\tau_1)(1+j\omega\tau_2)}$$

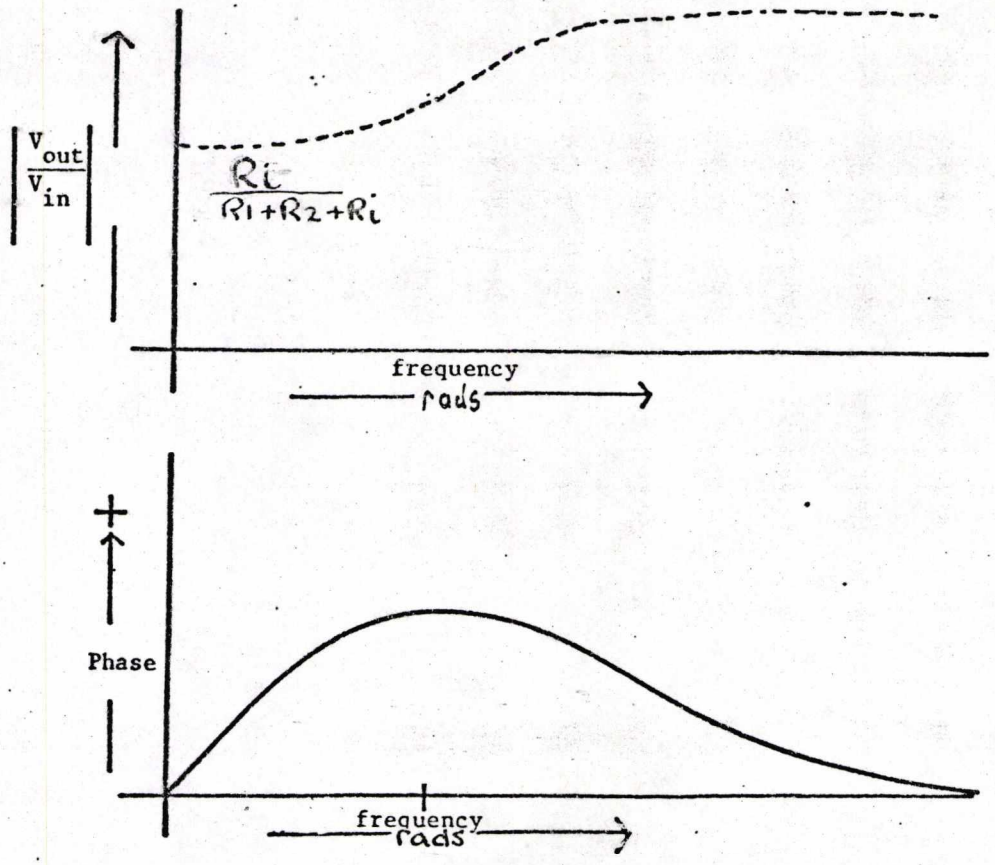


FIG. (20) The response of the circuit of Fig. 19 when $R_i \ll R_1, R_2$

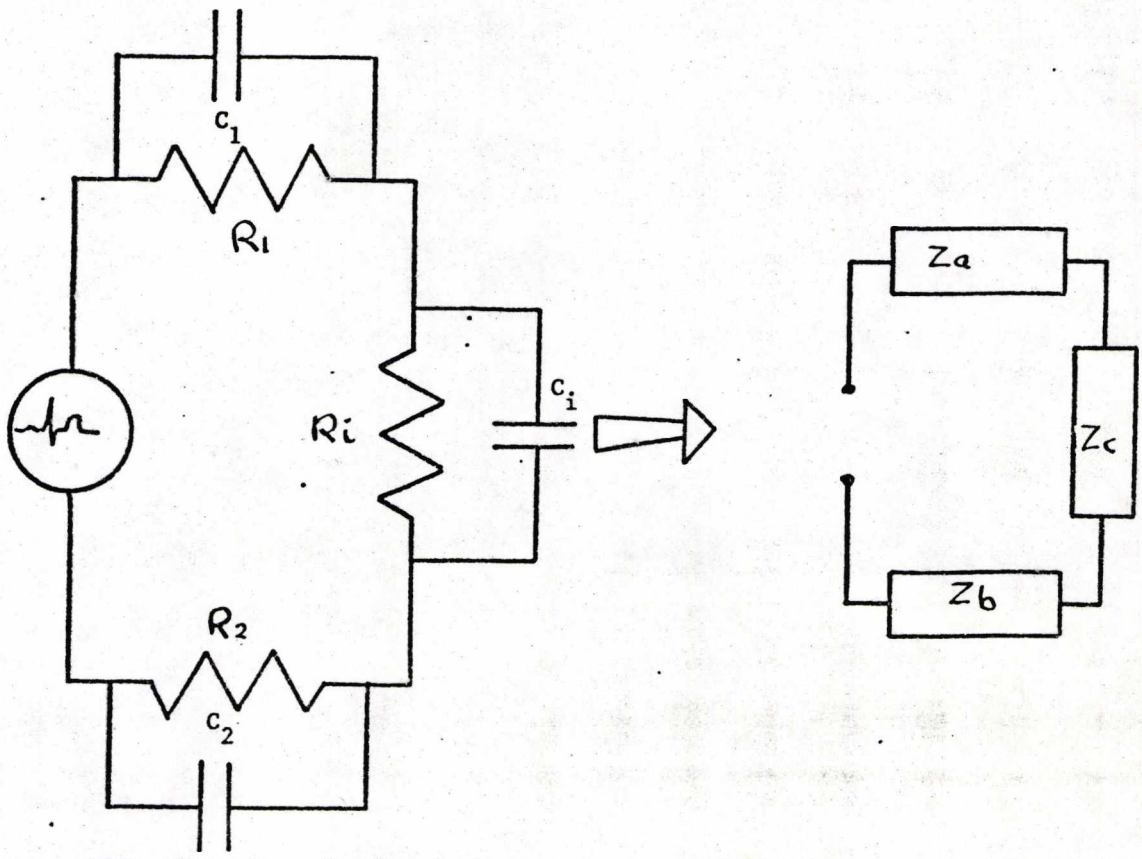


FIG. (21) The circuit of the input, when modified to include a capacitor C_i.

If we arrange all the time constant to be equal we have $\tau_1 = \tau_2 = \tau_i$, and substituting into the previous equation gives

$$\left[\frac{V_{out}}{V_{in}}\right] = \frac{R_i}{R_1 + R_2 + R_i}$$

$$\phi = 0$$

i.e. the response is independent of frequency.

This is illustrated in Fig.(22). If $\tau_i \ll \tau_1$ or τ_2 then the response is as in Fig.(23a), and if $\tau_i \gg \tau_1$ or τ_2 then the response is as in Fig.(23b).

In practice, due to stray capacitances that are present in the lead wires and input circuitry, it is the circuit of Fig.(21a) which represents the situation rather than that of Fig.(19a), but as Fig.(23a) shows, the same results can still be obtained when τ_i is very small i.e., the network has a high pass characteristic.

It can be seen from Fig.(22), however, that should the input capacitance be suitably chosen then the network response is flat, and thus, any signal applied via the network would not be distorted in shape though it would be diminished in amplitude. This latter fact does not really matter, since the gain of the following amplifier can easily be increased to compensate for this. The significant result of the analysis is that a low resistance can be used without distorting the input signal - providing a suitable value of input capacitor is chosen.

6.6b The CMRR and Low Input Resistance

The equivalent circuit used for the analysis is shown in Fig.(24), and initially the capacitors have been left out. Now, using Thevinins Theorem, for the common mode part of the

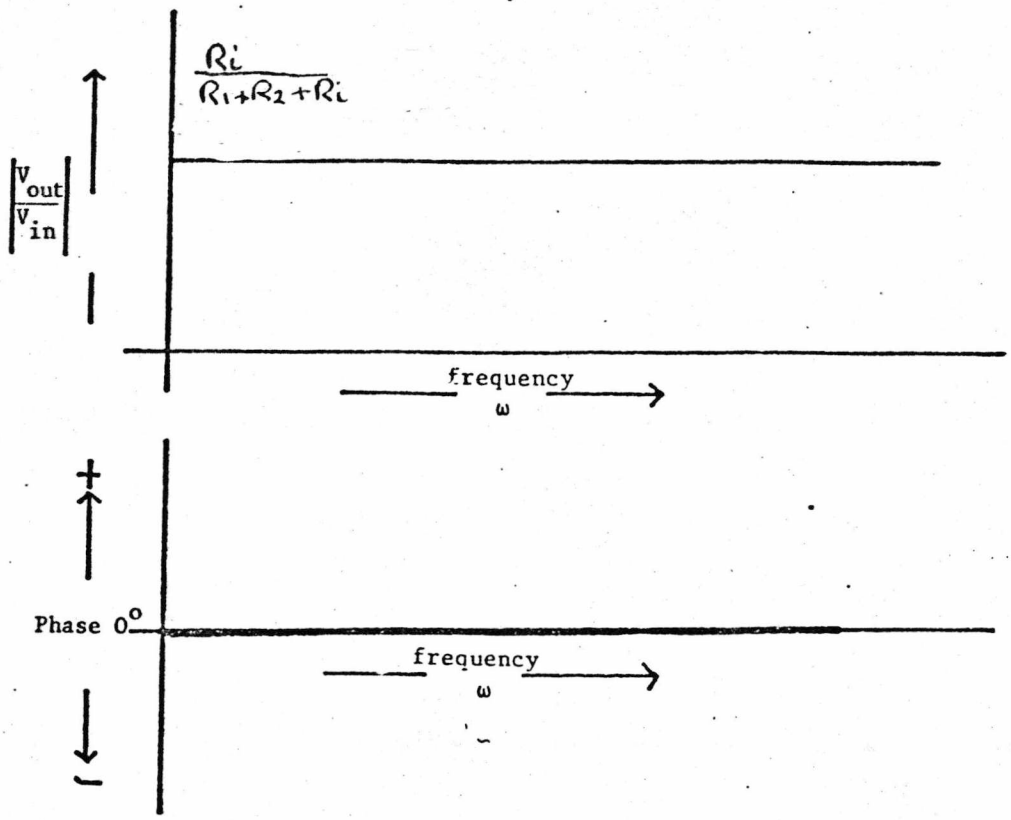


FIG. (22)

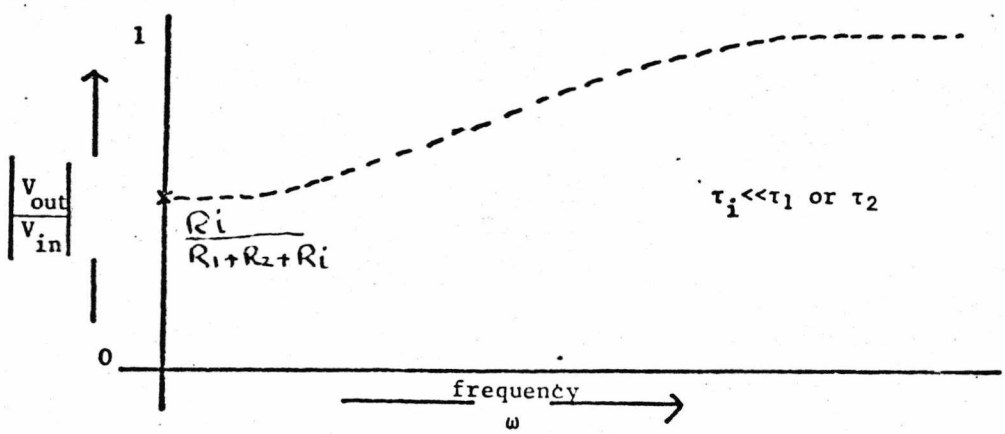


FIG. (23a)

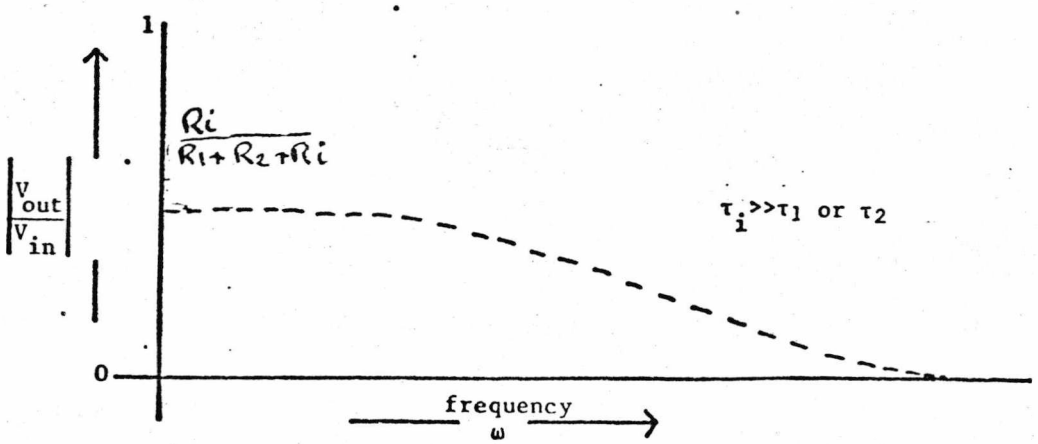


FIG. (23b)

calculation gives the circuit of *Fig. (25)*, let the actual input voltage to the amplifier = V_{cm} and the current in R_3

$$= \frac{V_c \times \left\{ \frac{R_5}{R_5+R_1} - \frac{R_4}{R_4+R_2} \right\}}{R_3 + \frac{R_1R_5}{R_1+R_5} + \frac{R_2R_4}{R_2+R_4}}$$

$\therefore$ The input voltage to the amplifier V_{cm}

$$= I_{R_3} \times R_3$$

$$V_{cm} = \frac{V_c \times R_3 \left\{ \frac{R_5}{R_5+R_1} - \frac{R_4}{R_4+R_2} \right\}}{R_3 + \frac{R_1R_5}{R_1+R_5} + \frac{R_2R_4}{R_2+R_4}}$$

For the differential signal the circuit of *Fig. (26)* will be used.

Let the actual input voltage to the amplifier

$$= V_{DM}$$

= voltage across R_3.

$$V_{DM} = \frac{\frac{R_3(R_4+R_5)}{R_3+R_4+R_5} \times V_D}{R_1+R_2 + \frac{R_3(R_4+R_5)}{R_3+R_4+R_5}}$$

$$= \frac{R_3(R_4+R_5) \times V_D}{(R_1+R_2)(R_3+R_4+R_5)+R_3(R_4+R_5)} .$$

Now the common mode rejection ratio is defined as the ratio $\frac{V_c}{V_D}$ when $V_{cm} = V_{DM}$.

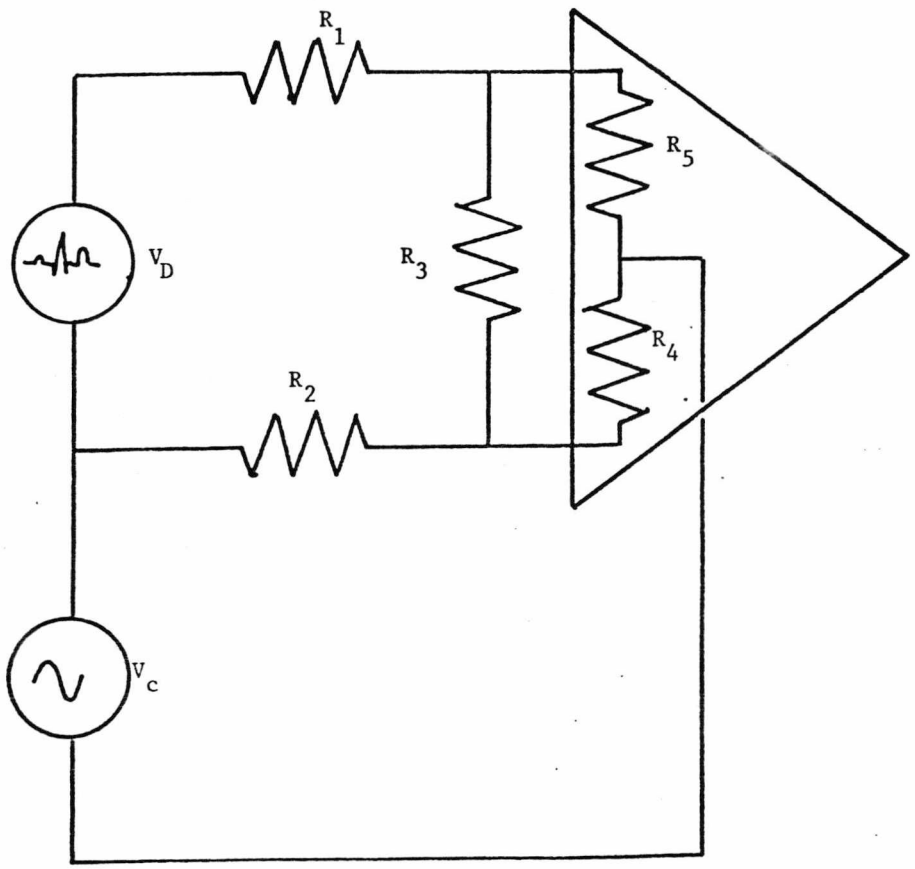


FIG. (24) Complete circuit for CMRR analysis

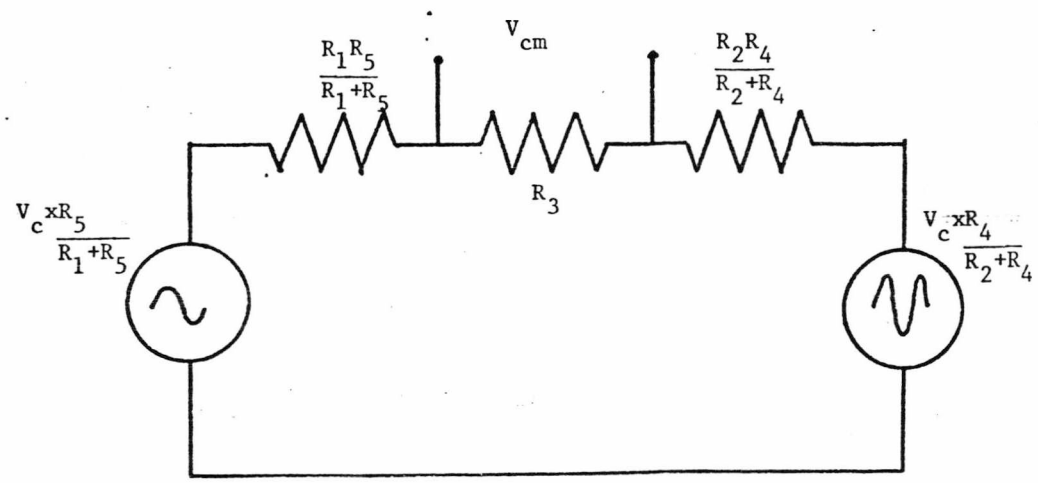


FIG. (25) The equivalent circuit for the common mode signal

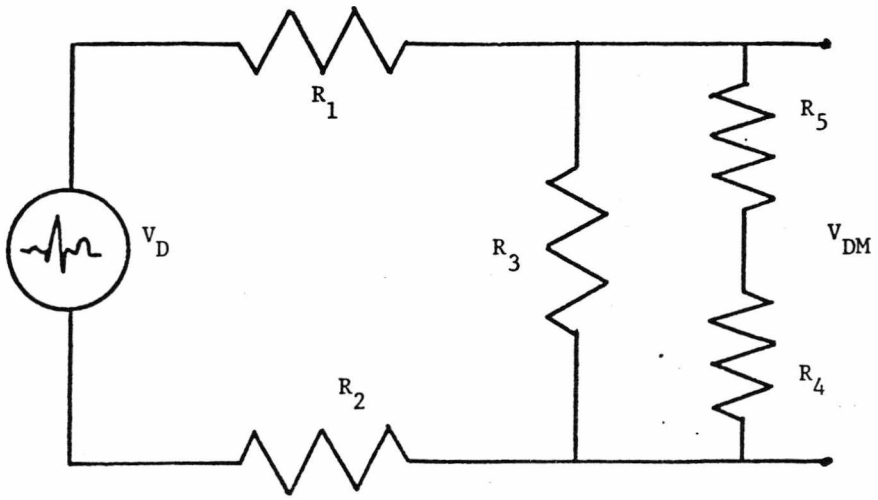


FIG. (26) The equivalent circuit for difference signal

V_{in} = signal in, N_{in} = Noise from electrodes in, N_a = amplifier noise

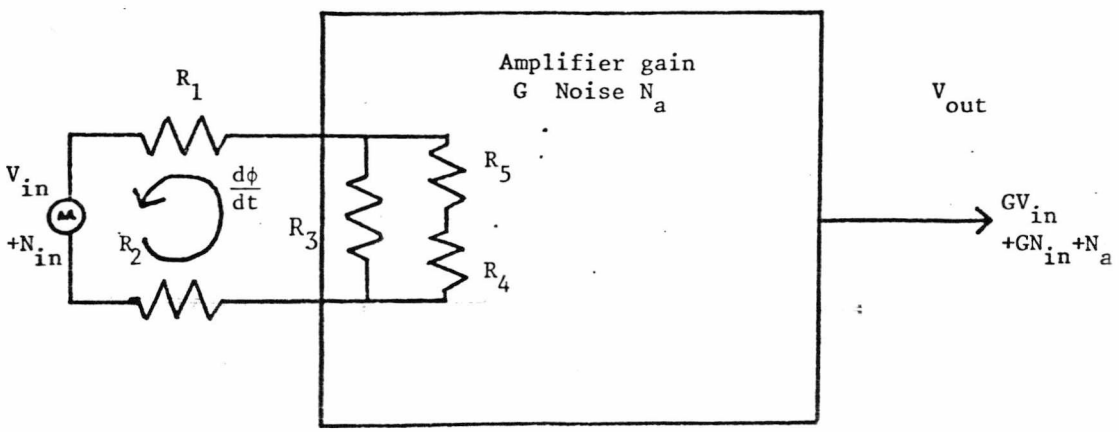


FIG. (27) The circuit for signal to noise calculations

$$\therefore \text{CMRR} = \frac{V_C}{V_D} = \frac{(R_4+R_5)\left(R_3 + \frac{R_1R_5}{R_1+R_5} + \frac{R_2R_4}{R_2+R_4}\right)}{\left\{(R_1+R_2)(R_3+R_4+R_5)+R_3(R_4+R_5)\right\}\left\{\frac{R_5}{R_1+R_5} - \frac{R_4}{R_2+R_4}\right\}}$$

Allow $R_3 \to \infty$, and substituting gives:-

$$\frac{V_C}{V_D} = \frac{(R_4+R_5)}{(R_1+R_2+R_4+R_5)\left\{\frac{R_5}{R_1+R_5} - \frac{R_4}{R_4+R_2}\right\}}$$

Allow $R_3 \to 0$, and substituting in the CMRR equation gives:-

$$\frac{V_C}{V_D} = \frac{\left\{\frac{R_1R_5}{R_1+R_5} + \frac{R_2R_4}{R_2+R_4}\right\}}{(R_1+R_2)(R_4+R_5)\left\{\frac{R_5}{R_1+R_5} - \frac{R_4}{R_4+R_2}\right\}}$$

If, as in practice, $R_5 >> R_1$ and $R_4 >> R_2$,

then $$\frac{V_C}{V_D} = \frac{R_4R_5}{R_5R_2-R_4R_1}$$

$R_3 \to \infty$

and $$\frac{V_C}{V_D} = \frac{R_4R_5}{R_5R_2-R_4R_1}$$

$R_3 \to 0$

It can be seen that the common mode rejection ratio is, therefore, completely unchanged no matter what the value of

R_3, and, in fact, it is the same as that defined by Cobbold and depends solely upon the matching of the electrodes as stated before, and not upon the value of the resistance placed across the input. If capacitances were included in the circuit, then the R's could be changed to Z's, and the same results would still be found.

6.6c Signal-to-Noise Ratios, and Low Input Impedance

Consider the diagram in *Fig. 27*. The signal V_{in} and noise input N_{in} are from the electrodes, and the noise from the input resistor network is considered in with the amplifier noise N_a, and the gain of the amplifier is G.

Then for $R_3 \to \infty$

$$\text{The Noise Out} = G \times N_{in} + N_a$$

$$\text{The Signal Out} = G \times V_{in}$$

$$\therefore \frac{S}{N} \text{ ratio} = \frac{G\,V_{in}}{G.N_{in} + N_a}$$

With $R_3 \to 0$, both the signal and noise going to the amplifier are attenuated by

$$\frac{R_3}{R_1+R_2+R_3}$$

$$\text{therefore the Noise Out} = G \times N_{in} \times \frac{R_3}{R_1+R_2+R_3} + N_a$$

$$\text{the Signal Out} = G \times V_{in} \times \frac{R_3}{R_1+R_2+R_3}$$

$$\therefore \frac{S}{N} \text{ ratio} = \frac{G \times V_{in}}{G \times N_{in} + \frac{R_1+R_2+R_3}{R_3} \times N_a}$$

Thus the S/N ratio is affected only slightly by a small input resistance. It must also be considered, however, that since the amplifier noise includes the input resistor network, then as R_3 is reduced, so the total amplifier noise N_a is reduced as well. In practice, with chlorided silver electrodes detecting an ECG we have typical figures of G = 1000, V_{in} = 1mV, $N_{in} \simeq 10\mu V$[52] and $N_a = 100\mu V$. Then the change in S/N ratio is not apparent until $R_3 << R_1+R_2$. Since it is proposed that R_3 should be 10KΩ→47KΩ, which is approximately equal to the values of R_1 and R_2, then it can be concluded that the S/N ratio is unaffected by having a low input resistance.

Another source of interference are the currents that may be induced in the wires in the leads and input circuits due to the loops formed by these, by stray electromagnetic radiation[47] e.g., the fields propagating from mains wiring installations, *Fig. (27)*.

The induced voltage = $K \cdot \frac{d\phi}{dt}$ in the loop

and the resultant current

$$= \frac{K \cdot \frac{d\phi}{dt}}{\text{Total resistance}}$$

For $R_3 \to \infty$ the induced voltage is that appearing across R_3 and (R_4+R_5) in parallel.

Since $R_3 \to \infty$ Induced Voltage $= R_4+R_5 \times \frac{K}{R_1+R_2+R_4+R_5} \times \frac{d\phi}{dt}$

For $R_3 \to 0$ Induced Voltage $= R_3 \times \frac{K}{R_1+R_2+R_3} \times \frac{d\phi}{dt}$

$\therefore$ The $\frac{S}{N}$ ratio for the induced noise

$$\text{For } R_3 \to \infty = \frac{G\, V_{in}}{G \cdot \frac{(R_4+R_5)\times K}{R_1+R_2+R_4+R_5} \times \frac{d\phi}{dt}}$$

$$\text{For } R_3 \to 0 = \frac{G.V_{in}}{G.\frac{R_3 \times K}{R_1+R_2+R_3} \times \frac{d\phi}{dt}}$$

$$\therefore \text{ the } \frac{S/N\ R_3 \to \infty}{S/N\ R_3 \to 0} = \frac{(R_1+R_2+R_4+R_5)}{(R_4+R_5)}$$

and if $R_4 >> R_2$ and $R_5 >> R_1$

$= 1$

Therefore, the S/N ratio for induced voltage interference does not significantly change when low resistances are placed across the input.

REFERENCES

(1) J. RAWLES & G. CROCKETT "Evaluation of ECG Monitoring Apparatus on a General Medical Ward" 1969. Bio-Medical Engineering, Vol. 4, No. 4, p. 154.

(2) C. FRAMPTON et al "An ECG telemetry system for physiological studies on swimmers" 1976. Bio-Medical Engineering, Vol. 11, No. 3, March.

(3) L.A. GEDDES "Electrodes and the measurement of bioelectric events" 1972. Wiley Inter-Science, p. 57.

(4) P. STRONG "BioPhysical Measurement" 1971. Tetronix Inc. Beaverton, Oregon, p. 216.

(5) J. BOTER et al "Disturbance free skin electrodes for persons during exercise" 1966. Medical and Biological Engineering, Vol. 4, p. 91.

(6) J.A. KLYN & M.J.G.M. KLOPROGGE "Movement artefact suppressor during ECG monitoring" 1976. Cardiovascular Research, Jan., Vol. 8, p. 149.

(7) L.A. GEDDES & L.E. BAKER "Principles of applied biomedical instrumentation" 1975. Wiley Inter-Science, p. 204.

(8) G.C. LUCCHINA & C.C. PHIPPS "An improved electrode for physiological recording" 1963. Aerospace Medicine, March, p. 230-31.

(9) A. KAHN "Electrodes" 1966. Digest of the 6th International Conference on Medical and Biological Engineering, Tokyo.

(10) L.A. GEDDES 1972. Op.Cit., p. 78-90.

(11) D. BENDERSKY "A muscle accelerator and spray-on electrocardiogram electrodes" 1967. Digest 7th International Conference on Medical and Biological Engineering, Stockholm.

(12) R.S.C. COBBOLD "Transducers for Bio-Medical measurements. Principles and Applications" 1974. Wiley Inter-Science, p. 412.

(13) P.B. MANSFIELD "Myocardial stimulation: the electrochemistry of electrode-tissue coupling" 1967. American Journal of Physiology, Vol. 212, No. 6, p. 1475-1488.

(14) V. POLLARK "An equivalent diagram for the interface impedance of metal needle electrodes" 1974. Medical and Biological Engineering, Vol. 12, No. 4, July. p. 454-459.

(15) R.C. GESTELAND et al "Comments on Microelectrodes" 1959. Proceedings of the IRE on Bio-Medical Engineering, Vol. 10, p. 26-28.

(16) R. COOPER "Electrodes" 1963. American Journal of EEG Technology, Vol. 3, No. 4, p. 91-101.

(17) P. STRONG 1971. Op.Cit., p. 219.

(18) A.R. DENARO "Elementary Electrochemistry" 1971. Butterworths, London, p. 88.

(19) A.R. DENARO 1971. Op.Cit., p. 190.

(20) C.W. DAVIES "Electrochemistry" 1967. Newnes. London, Ch. 9.

(21) R.S.C. COBBOLD 1974. Op.Cit., p. 328.

(22) L.A. GEDDES 1972. Op.Cit., Ch. 1.

(23) L.A. GEDDES 1972. Op.Cit., p. 2.

(24) S. LYLE, Dr. Personal Communication, 1975. Univ. of Kent, Chemical Laboratories.

(25) A.R. DENARO 1971. Op.Cit., p. 93.

(26) R.S.C. COBBOLD 1974. Op.Cit., p. 329.

(27) R.S.C. COBBOLD 1974. Op.Cit., p. 331.

(28) A.R. DENARO 1971. Op.Cit., p. 115.

(29) D. LEWES "Electrode jelly in electrocardiography" 1965. British Heart Journal, Vol. 27, No. 1, p. 105-115.

(30) A.R. DENARO 1971. Op.Cit., p. 105.

(31) R.S.C. COBBOLD 1974. Op.Cit., p. 416.

(32) R.S.C. COBBOLD 1974. Op.Cit., p. 418.

(33) D.W. HILL "Electronic techniques in anaesthesia and surgery" 1973. Butterworths, London, p. 191.

(34) L.A. GEDDES 1972. Op.Cit., p. 14.

(35) L.A. GEDDES & L.E. BAKER 1975. Op.Cit., p. 210.

(36) H. FRICKE "The theory of electrolyte polarisation" 1932. Philosophical Magazine, Vol. 14, p. 310-318.

(37) D.W. HILL & R.S. KHANDPHUR "The performance of ECG pre-amplifiers" 1969. World Medical Instrumentation, Vol. 7, p. 12-22, May.

(38) L.A. GEDDES 1972. Op.Cit., p. 21.

(39) R.S.C. COBBOLD 1974. Op.Cit., p. 424.

(40) J. WEINMAN & J. MAHLER "An analysis of electrical properties of metal electrodes" 1964. Medical Electronic and Biological Engineering, Vol. 2, p. 299-310.

(41) J.J. ALMASI & O.H. SCHMITT "Systemic and Random variations of ECG electrode system impedance" 1970. Annals of the New York Academy Science, Vol. 170, p. 509-519.

(42) A.S. BERSON & H.V. PIPBERGER "Skin electrode impedance problems in electrocardiography" 1968. American Heart Journal, Vol. 76, No. 4, Oct., p. 514-525.

(43) L.A. GEDDES & L.E. BAKER "The relationship between input impedance and electrode area in recording the ECG" 1966. Medical and Biological Engineering, Vol. 4, p. 439-450.

(44) L.A. GEDDES "Interface design for bioelectrode systems" 1972. IEEE Spectrum, October 1972.

(45) L.A. GEDDES "The use of liquid junction electrodes in recording the human electrocardiogram" 1968. Journal Electrocardiology, Vol. 1, p. 51-56.

(46) R.S.C. COBBOLD 1974. Op.Cit., p. 430.

(47) J.C. HUHTA & J.G. WEBSTER "60 Hz interference in electrocardiography" 1973. IEEE Trans. on Bio-Medical Engr., Vol. BME20, No. 2, March.

(48) P. STRONG 1971. Op.Cit., p. 226.

(49) DEPT. HEALTH AND SOCIAL SECURITY "3m red dot electrodes for ECG monitoring - withdrawal from use" 1976. Health Service Management Bulletin, No. H.N.(76).64, April.

(50) P. STRONG 1971. Op.Cit., p. 249-267.

(51) L.A. GEDDES 1972. Op. Cit., p. 8.

(52) L.A. GEDDES 1972. Op. Cit., p. 12.

CHAPTER 7

THE PRACTICAL ASPECTS OF LOW INPUT IMPEDANCE RECORDING

7.1 INTRODUCTION

Chapter 6 has examined the possibilities of using a low input resistance for an ECG amplifier, and has shown that the distortion normally encountered can be avoided. It has also been shown that the common mode rejection ratio (CMRR) of an amplifier is not adversely affected by the input conditions, and it has been suggested that a low input impedance could prevent artefact from interfering with ECG recording by removing the offset potential between the electrodes.

It was also stated in Chapter 6 that several workers have verified that the components of the equivalent circuit of an electrode were frequency dependent, and also that the impedance of one type of electrode varied from place to place on one person. It may prove, therefore, that it is impossible to satisfactorily compensate the amplifier input since the condition of equal time constants cannot be met, due to the changing parameters of the electrode throughout one ECG complex. It is the purpose of this chapter to test, experimentally, the theory proposed in Chapter 6 and to find out if it is a practical proposition to use low input impedance amplifiers.

At first ideal circuits will be tested by constructing equivalent model circuits from electronic components and measuring their characteristics. Then experiments with proper electrodes and subjects will be carried out, to test the validity of the comments of the previous paragraph. The frequency response, the CMRR and the offset potential behaviour

will all be investigated.

7.2 THE EFFECT OF R_{in} UPON THE ELECTROCARDIOGRAM

7.2a The Frequency Response of the System

A model was constructed of an electrode recording system and is shown in *Fig. (1)*. The two electrodes are represented by parallel combinations of resistors and capacitors and the input resistance was variable in value to represent different values of input resistance to the amplifier. This was represented by a differential pre-amplifier plug in unit of a storage oscilloscope which was also used to monitor the outputs of the network. The values of components chosen for the model were considered to be reasonably typical values (reference 33, Chapter 6). The frequency of the source was varied over the range 1Hz to 1KHz with the amplitude being kept constant; and the amplitude and phase of the signals appearing across the resistance R_{in} were measured for each frequency. Frequency and phase response graphs were plotted and the results can be seen in *Figs. (2)* and *(3)*. They are similar to those predicted in the theoretical analysis of section 6.6(i) i.e., they have a high pass filter characteristic when R_{in} is low. It can be seen from *Fig. (2)* that as the value of the input resistance R_{in} is increased, ~~this~~ the characteristic shape changes until ~~when~~ R_{in} is 1MΩ when it is almost a flat response. The phase response shows a zero phase shift for all values of R_{in} except the lowest two - 10KΩ and 47KΩ *(Fig. (3))*.

When the experiment is repeated with R_{in} compensated by switching in a suitable capacitor C_{in} so that the time constant $R_{in}C_{in} = R_eC_e$, and the frequency and phase responses are plotted, then, once again, the results are as predicted in

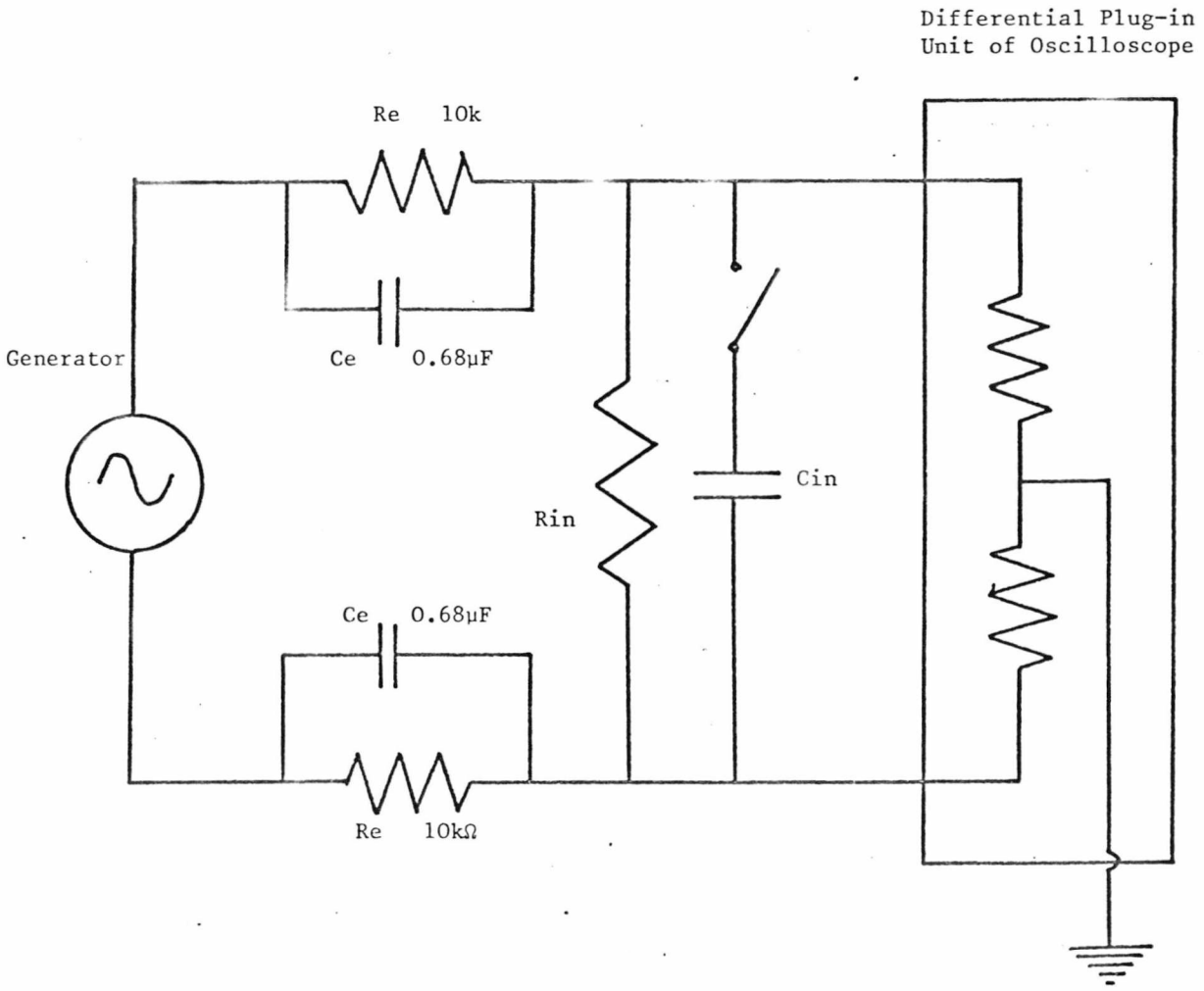


FIG. 1 Circuit Model of an ecg recording system.

FIG. 2 The frequency response of the circuit model of Fig. 1.

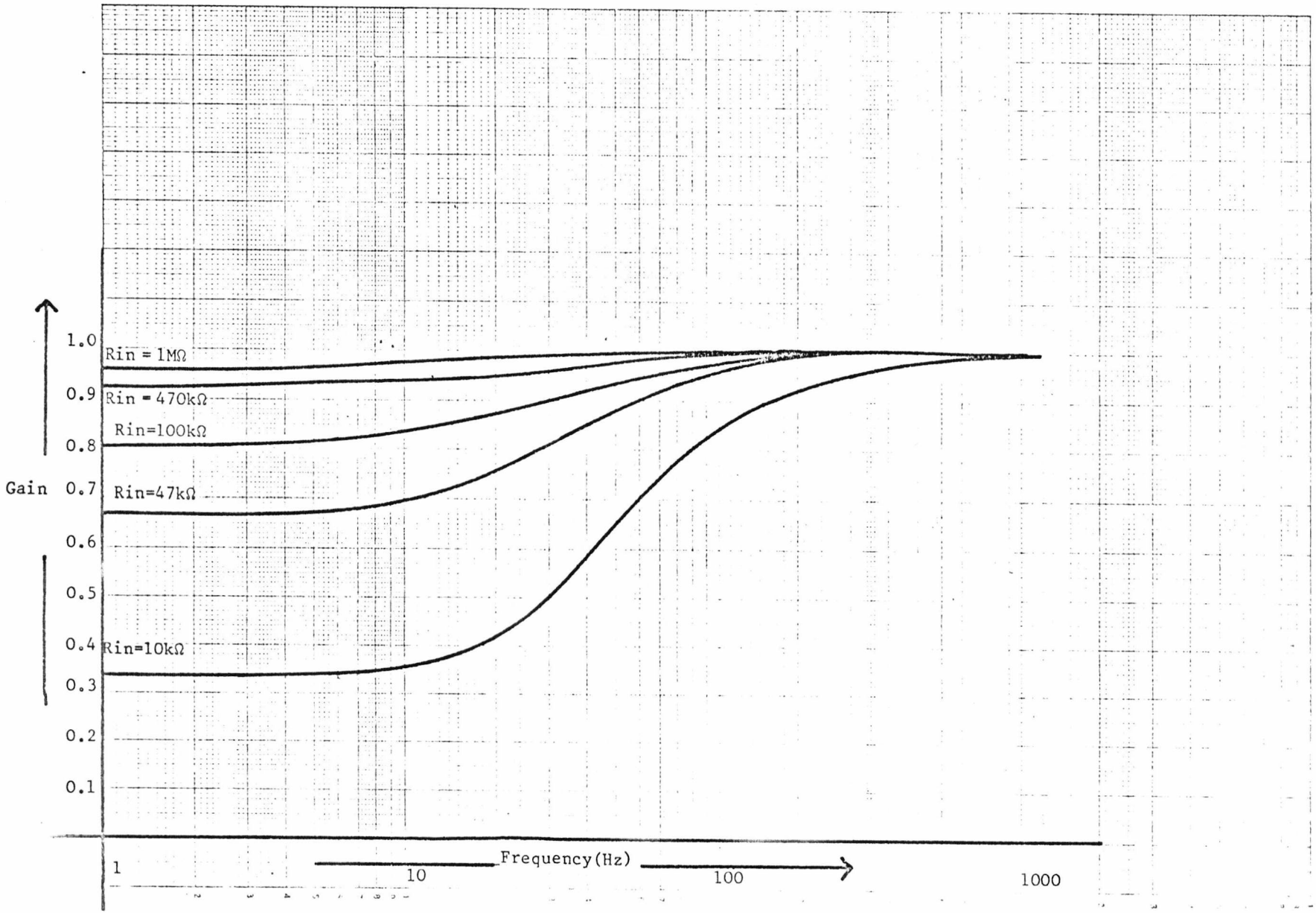


FIG. 3 The phase response of the circuit model of Fig. 1

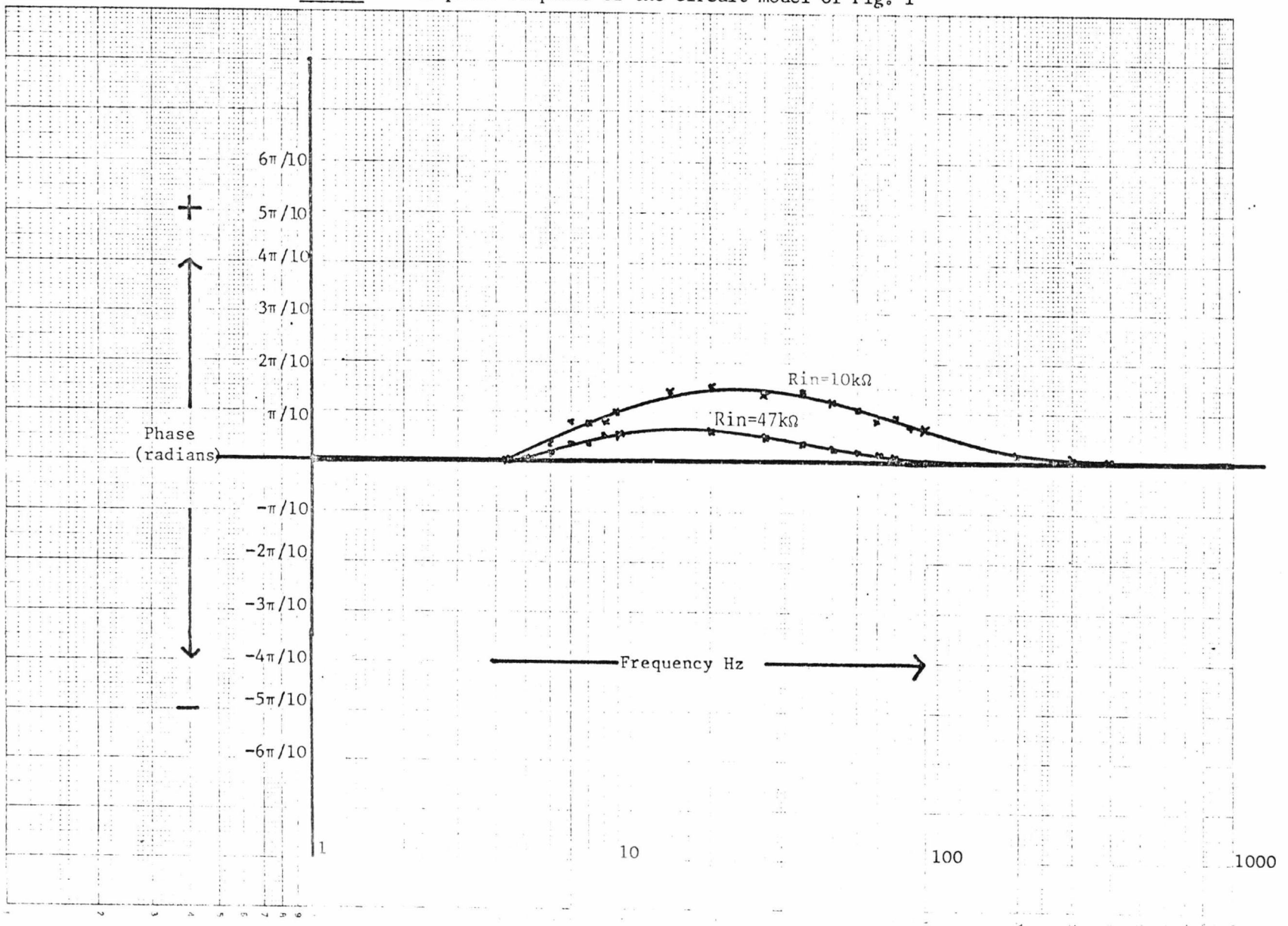
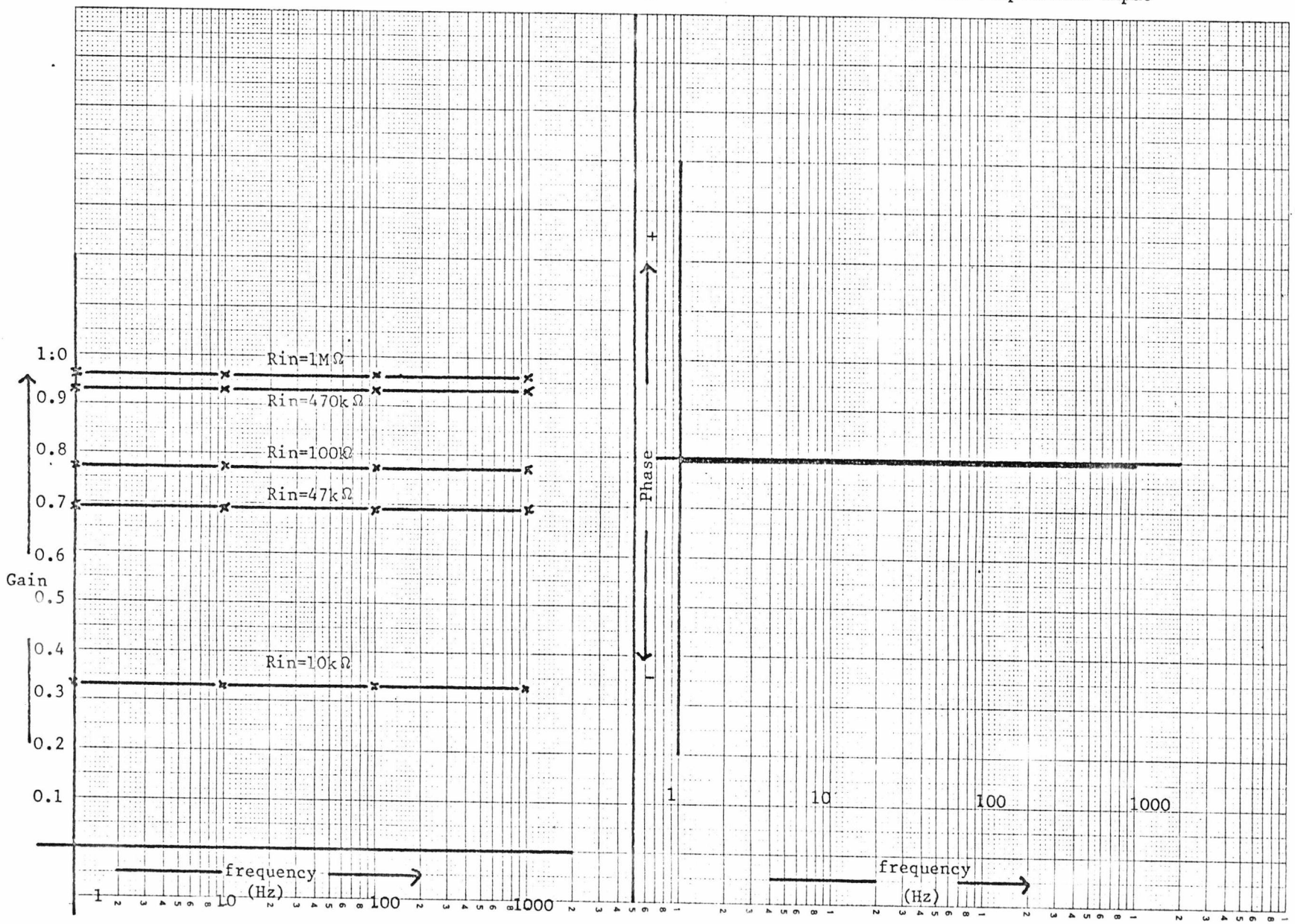


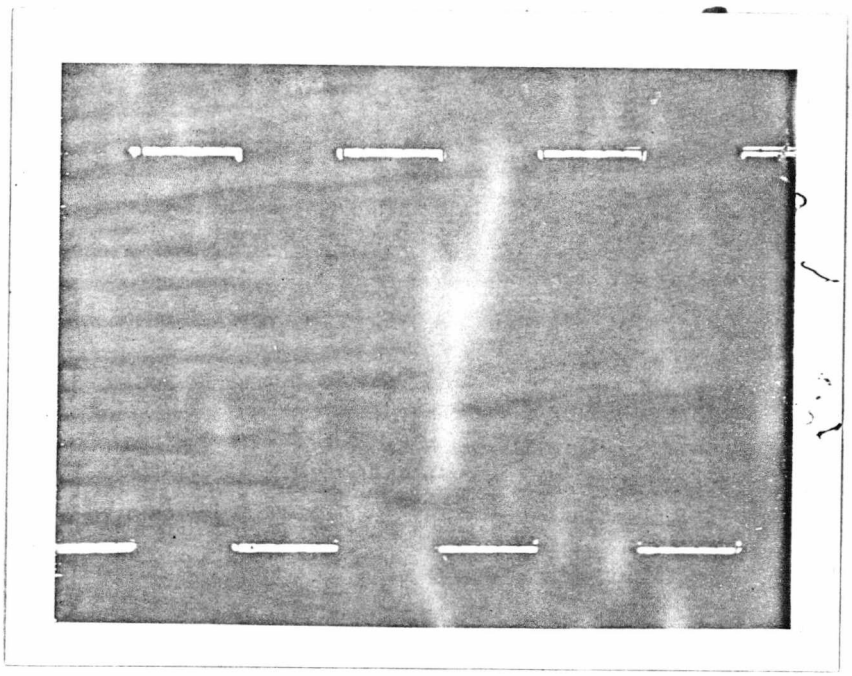
FIG.4 The frequency and phase response of the circuit model with compensated input



6.6(i) and can be seen in *Fig. (4)*. There is no phase shift for any value of R_{in} and the shape of the amplitude responses are quite flat, with just smaller amplitudes for the lower values of R_{in}.

The photographs of *Figs. (5)* and *(6)* show the dynamic response of the system. A 10Hz square wave was used as the source *Fig. (5a)* and the outputs recorded when R_{in} = 10KΩ and 1MΩ are shown in *Fig. (5b)*. The top trace is that of R_{in} = 10KΩ and the overshoot distortion can be clearly seen - there is even a slight overshoot and sloping horizontal sections of the wave when R_{in} = 1MΩ. *Fig. (6a)* shows the results when R_{in} is suitably compensated (10KΩ/0.68μF : 1MΩ/6800pf). Now the signals are both square shaped, and apart from amplitude changes closely resemble the original. *Fig. (6b)* shows the effect of both under and over compensating R_{in} by a factor of 10. The top trace shows the bad high frequency response when $R_{in}C_{in} = 10 \times R_eC_e$, resulting in a triangular shaped waveform whilst the bottom trace again shows overshoot on the edges of the wave when $R_{in}C_{in} = \frac{1}{10} \times R_eC_e$.

After the satisfactory results from the model, the same experiments were repeated, but this time the model electrodes, were placed by real ones on a subject *(Fig. (7))*. The oscilloscope was replaced by an ECG amplifier whose bandwidth satisfied the A.H.A. requirements *(Fig. (8))*. It had connected across its input a box containing a range of resistances (∞, 2.2MΩ, 1.0MΩ, 470KΩ, 220KΩ, 100KΩ, 47KΩ, 22KΩ, and 10KΩ) any one of which could be switched across the input. There was also a box of capacitors which could be connected in parallel with the input resistors, and this was variable from 0.1μF to 4.0μF in steps of 0.01μF. Thus a wide range of input conditions can be provided. Provision is made to introduce a



0.1volts/div ↑

50msecs/div →

FIG. 5a Input signal to the model - a 10Hz aquare wave

Top Trace

Rin=10kΩ

0.1volts/div ↑

50msecs/div →

Bottom Trace

Rin=1MΩ

0.1volts/div ↑

50msecs/div →

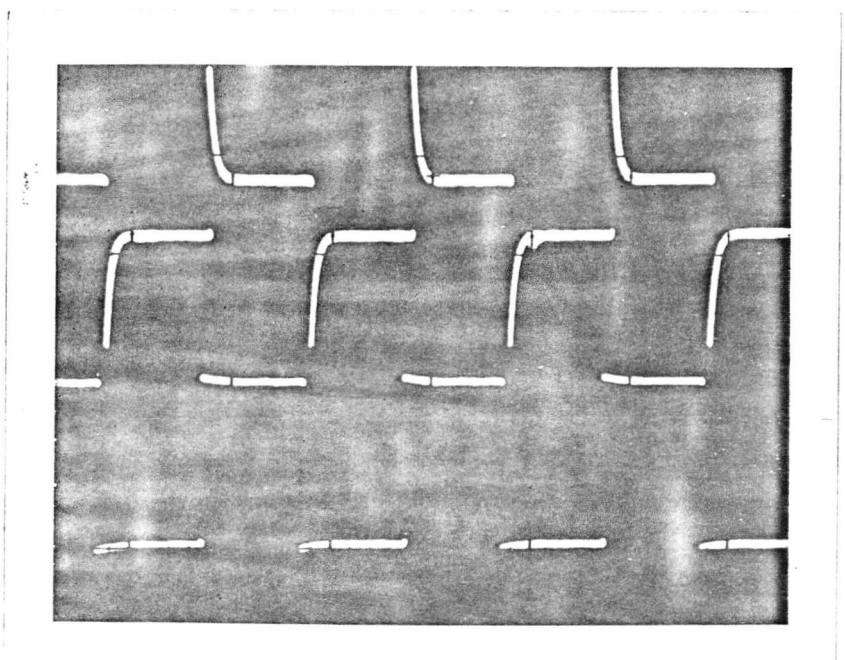


FIG. 5b The output from two values of uncompensated Rin

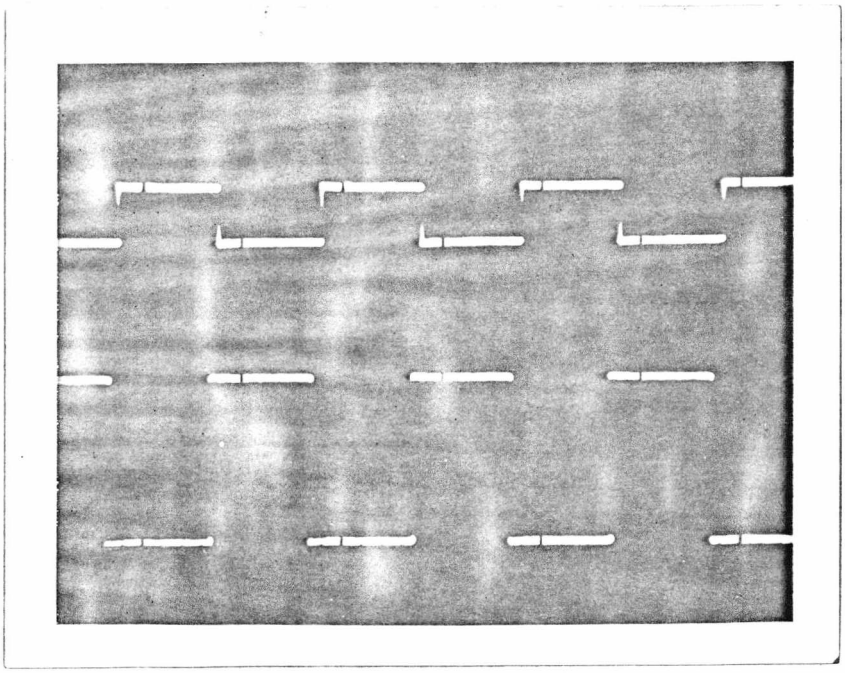
Top Trace

Rin=10kΩ

Cin=0.68μF

0.2volts/div ↑

50msecs/div →



Bottom Trace

Rin=1MΩ

Cin=6,800pf

0.2volts/div ↑

50msecs/div →

FIG. 6a The output with two values of compensated Rin

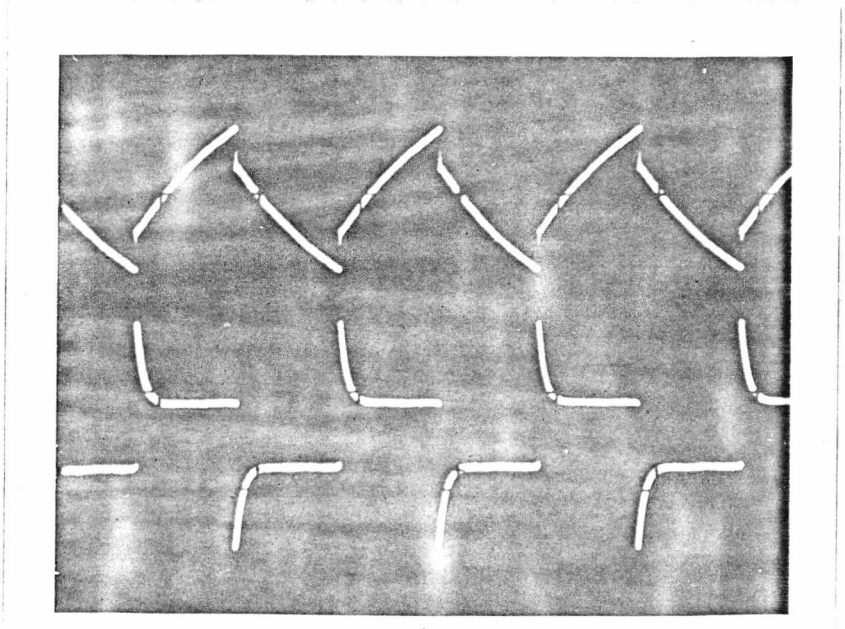
Top Trace

Rin=10kΩ

Cin=6.8μF

0.2volts/div ↑

50msecs/div →



Bottom Trace

Rin=10kΩ

Cin=0.068μF

0.2volts/div ↑

50msecs/div →

FIG. 6b The outputs when Rin is overcompensated and undercompensated

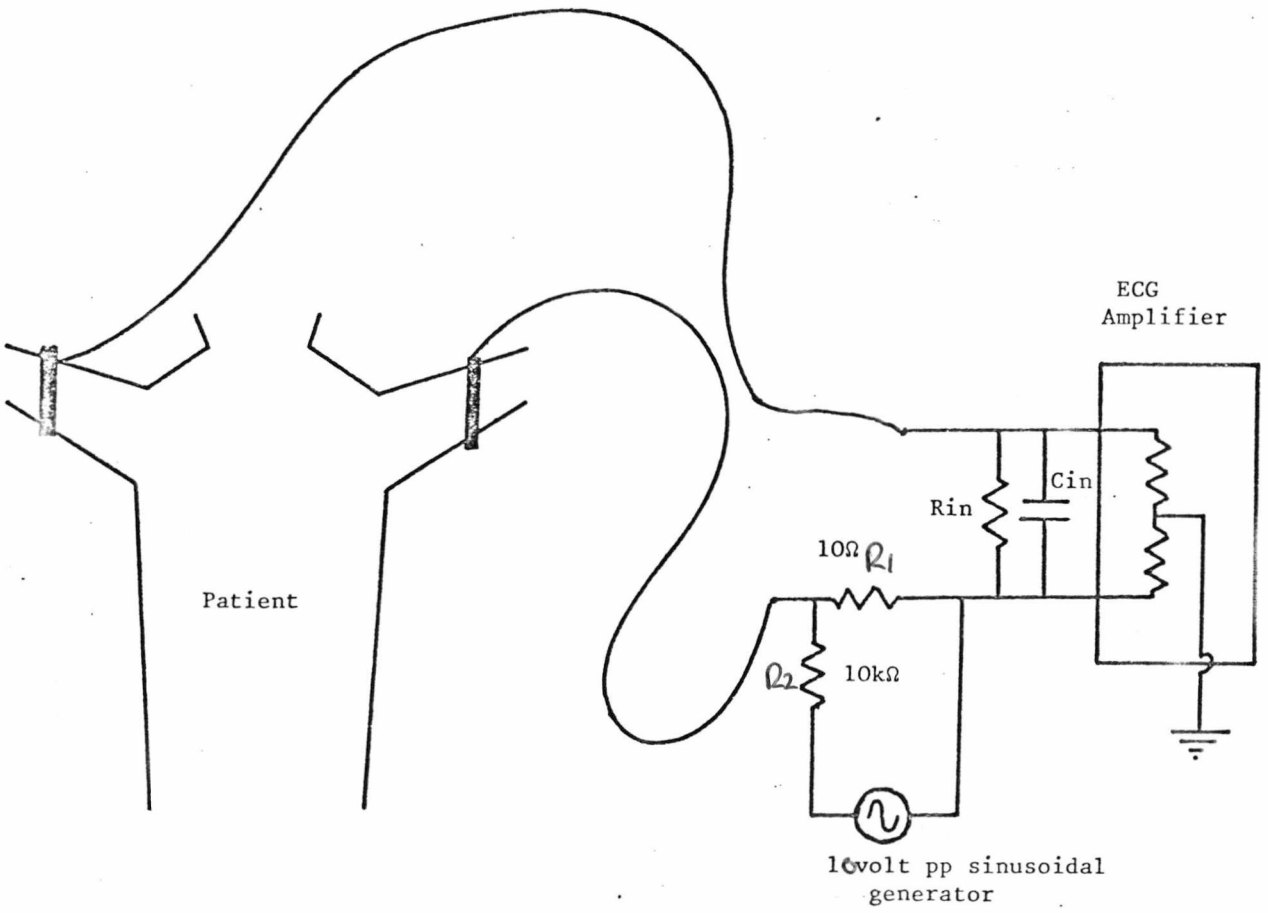
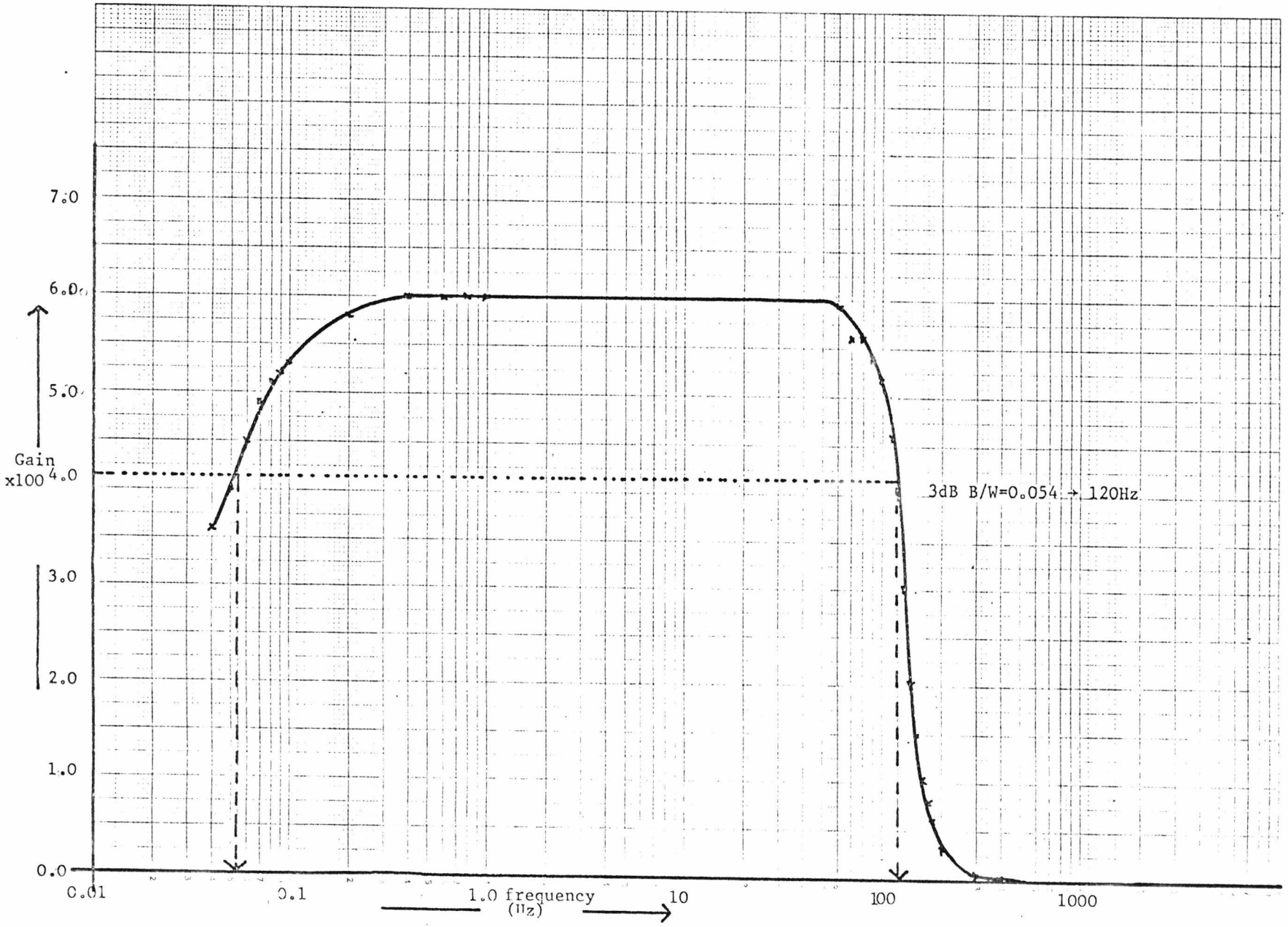


FIG. 7 Testing the frequency response of real electrodes

FIG. 8 The frequency response of the ECG amplifier



small (10mV) sinusoidal signal into the system from a variable frequency source (0.01Hz to 100Hz) via resistors R_1 and R_2. The injected signal was kept as small as possible to avoid large currents flowing through the electrodes and changing their characteristics.

The results are plotted in *Figs. (9)* and *(10)* for disposable silver/silver chloride electrodes and *Figs. (11)* and *(12)* for metal plate electrodes, both types being in common use at the hospital. The results shown in *Figs. (9)* and *(11)* agree with the theory and the model experiments and show the expected changes in frequency response as the input resistance changed from 1MΩ to 10KΩ. *Figs. (10)* and *(12)* show the effects of compensating the input resistance. The capacitor values were chosen by obtaining an ECG from a normal recorder, and adjusting the capacitor value until the ECG was the same as the control. Then the frequency response experiments were carried out.

It can be seen that the results were not quite those predicted by the theory and model, since the responses are not completely flat - even after making allowances for amplifier roll-off. The amplitude of the responses fall at the lower and higher ends (<1Hz and >50Hz) of the frequency scale. The reason for this is the change in components of the electrode impedance with frequency. Most of the energy in the ECG is contained in the 1Hz to 15Hz region (references 2,3,4, Chapter 4) and so choosing the capacitor by compensating the low input ECG until it is the same as the high input impedance one; it is mainly this frequency band which is compensated by equalising time constants. Thus the electrode time constant is greater than the input one for low frequencies, and smaller than the input one for high frequencies. This circumstance

FIG. 9 The frequency response of silver/silver chloride electrodes for various values of uncompensated Rin

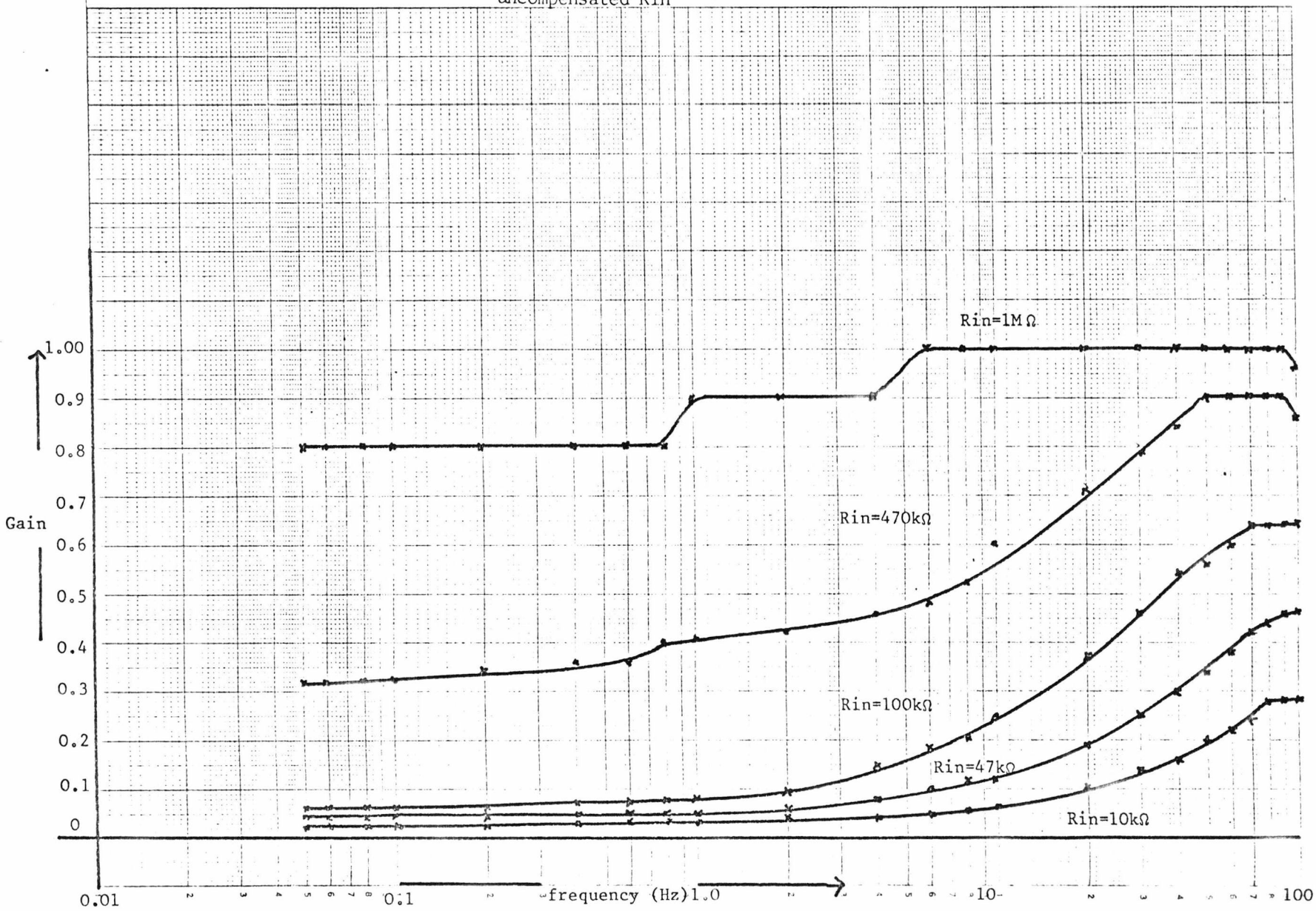


FIG. 10 The frequency response of silver/silver chloride electrodes for various values of compensated Rin.

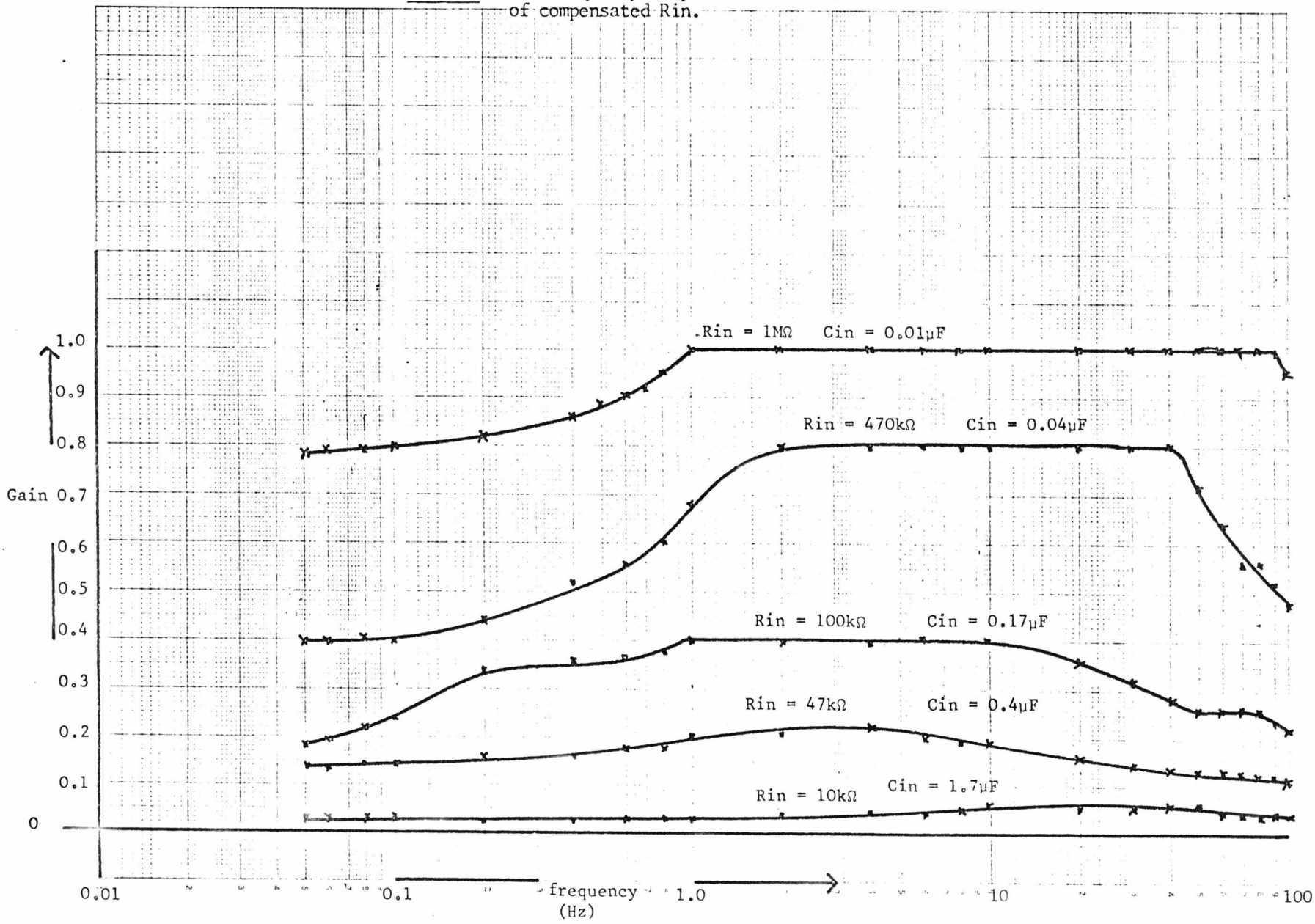


FIG. 11 Frequency response of small metal plate electrodes for various values of uncompensated Rin

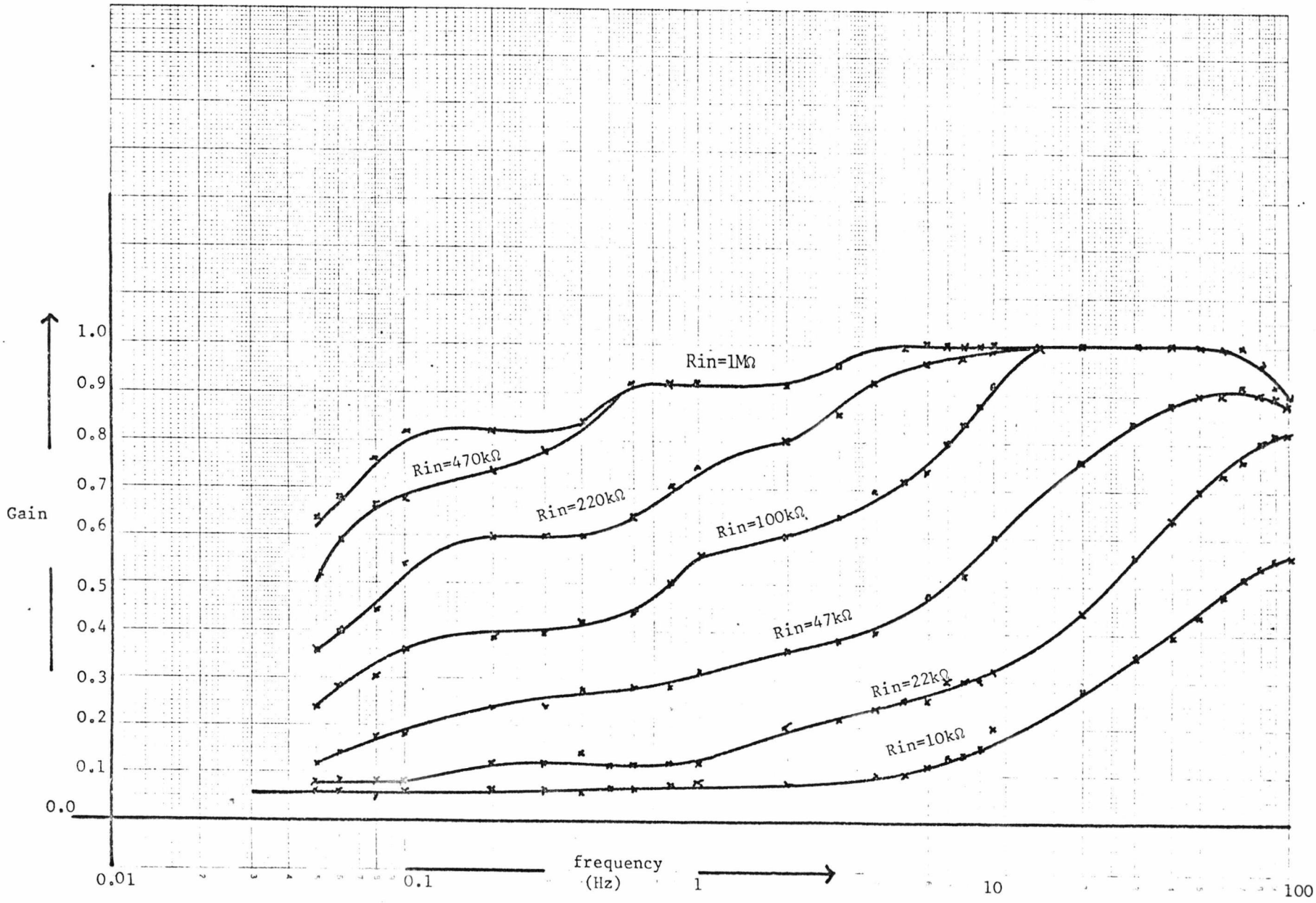
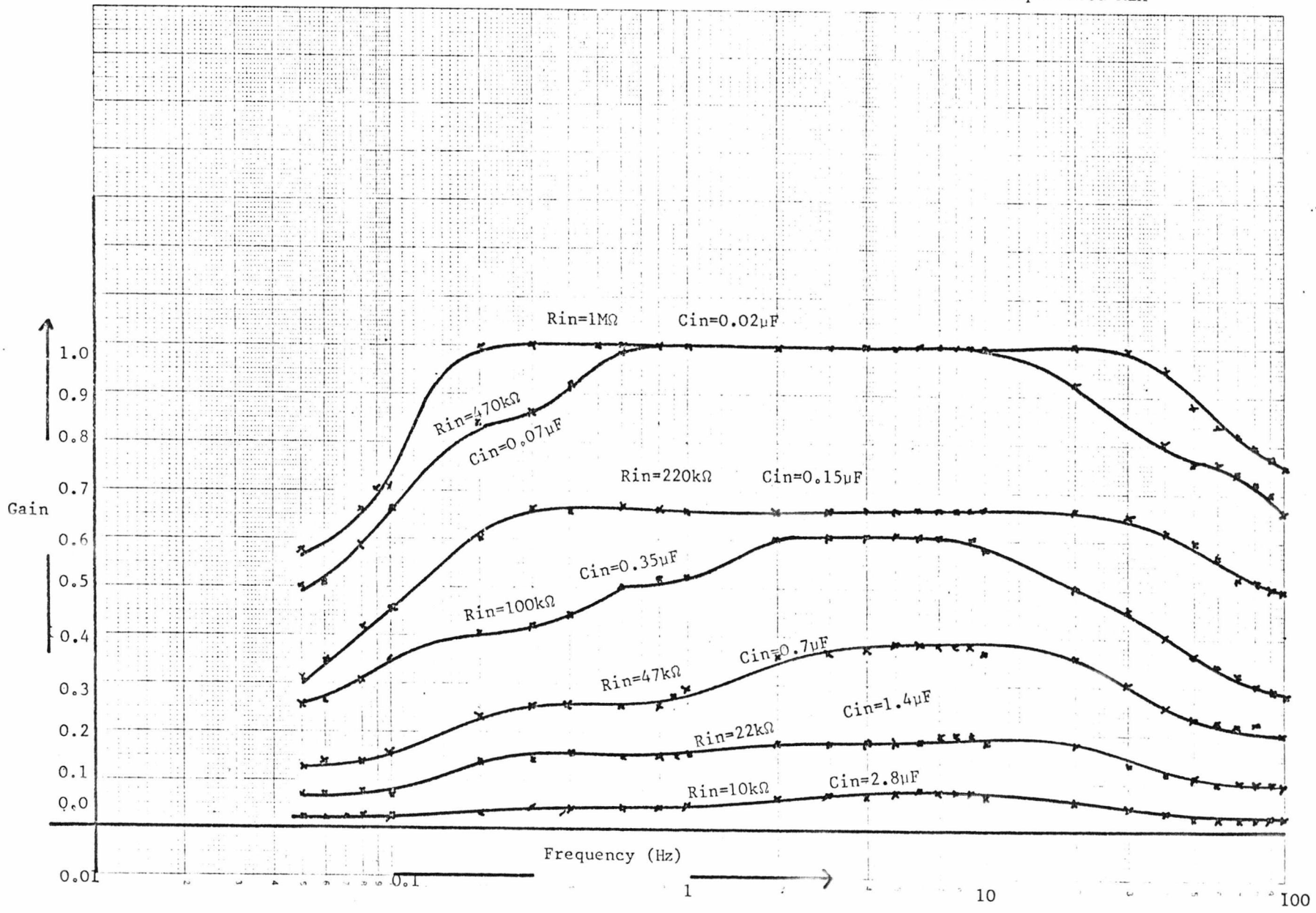


FIG. 12 Frequency response of small metal plate electrodes for various values of compensated Rin



would lead to the observed shape of the results. (*Figs. (23a), (23b)*, Chapter 6).

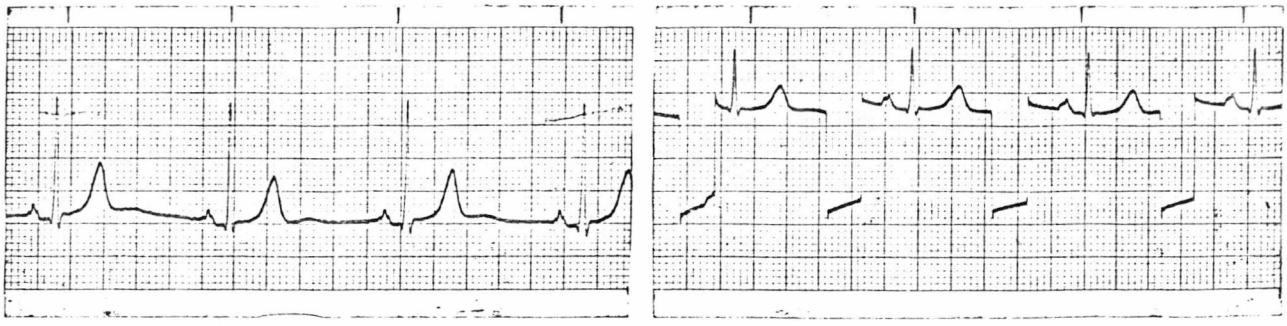
7.2b The Electrocardiogram and Input Resistance with and without Compensation

The experiments to observe the effect of low R_{in}s on the ECG was performed in the same manner as the frequency response tests *(Fig. (7))* except that the sine wave generator was replaced by a rectangular pulse generator, which introduced pulses of 0.1 sec. duration and 1mV in height in the circuit.

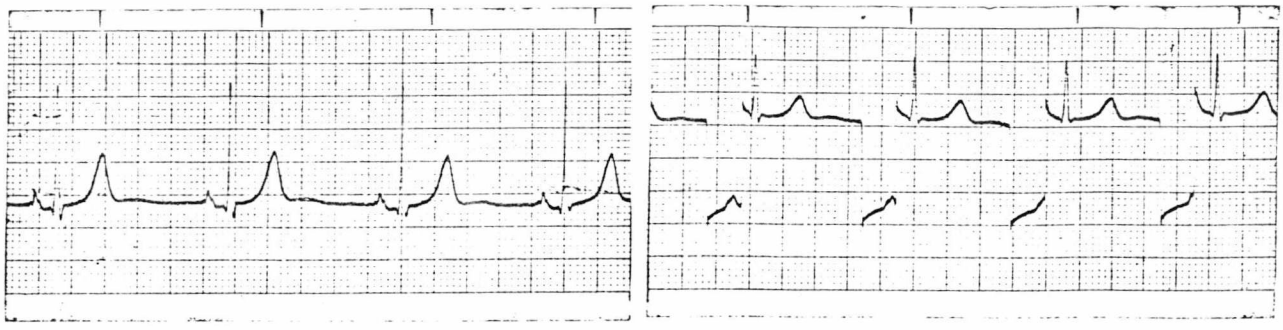
The input resistance was progressively reduced from 2.2MΩ, and the resulting ECGs recorded. The effect of this can be seen in the series of recordings in *Fig. (13)*. At first (R_{in} = 2.2MΩ) the ECG remains normal, and then a deepened S wave appears, and the P and T waves become smaller. Eventually, when R_{in} is 10KΩ both P and T waves and the QRS complex are all biphasic, and the overall amplitude is decreased. Alongside each recording of the ECG is one showing the effect upon the 1mV pulse, and the changes in this as R_{in} is lowered are similar to those of the ECG - with considerable overshoot of the leading and trailing edges developing. All of these observations are consistent with the previous results - that the low frequencies in the circuit are depressed in amplitude with respect to the high frequency components as R_{in} is lowered. *Fig. (13)* are the recordings when using large (20 sq.cms.) metal plate electrodes made from German Silver (an alloy of copper and nickel) which are commonly used in the ECG department. *Figs. (15)* and *(17)* show the results obtained from small (10 sq.cm.) metal plate electrodes and a disposable

ECG

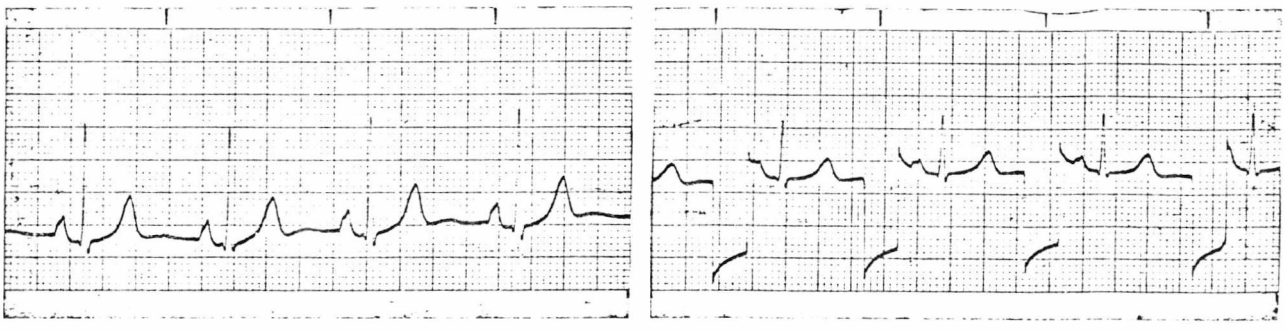
1mV 0.1sec pulse



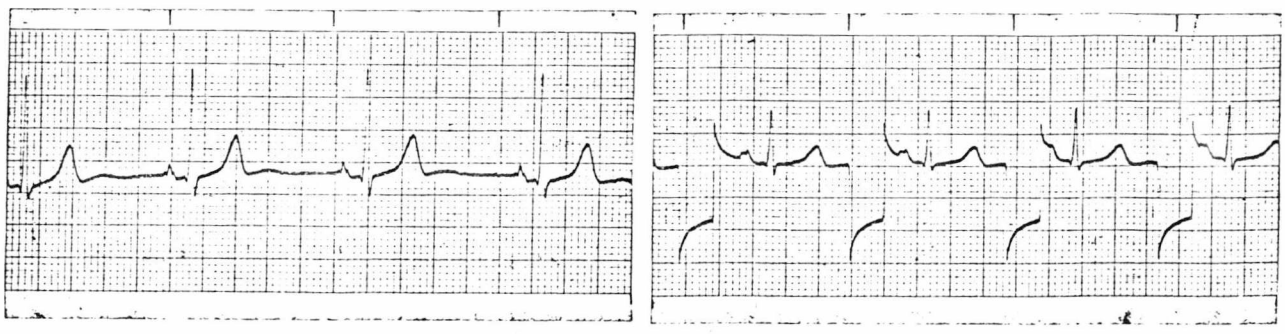
Rin = 2.2MΩ



Rin = 1MΩ



Rin = 470kΩ

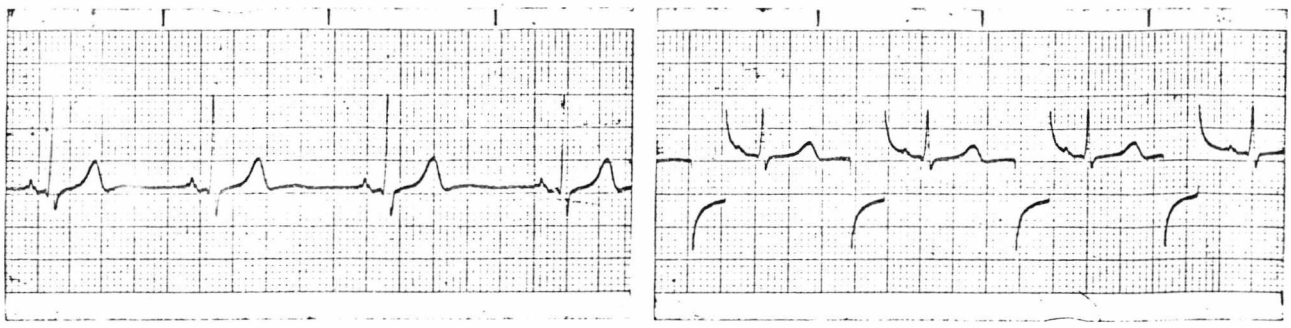


Rin = 220kΩ

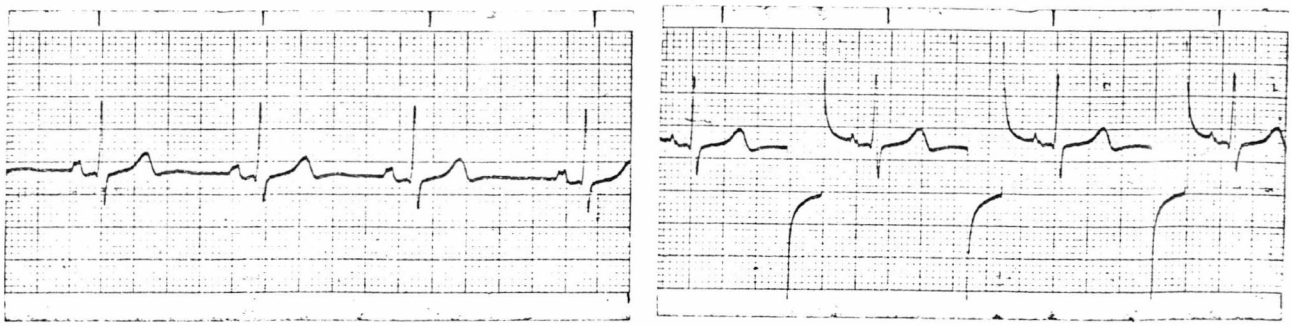
FIG. 13a

ECG

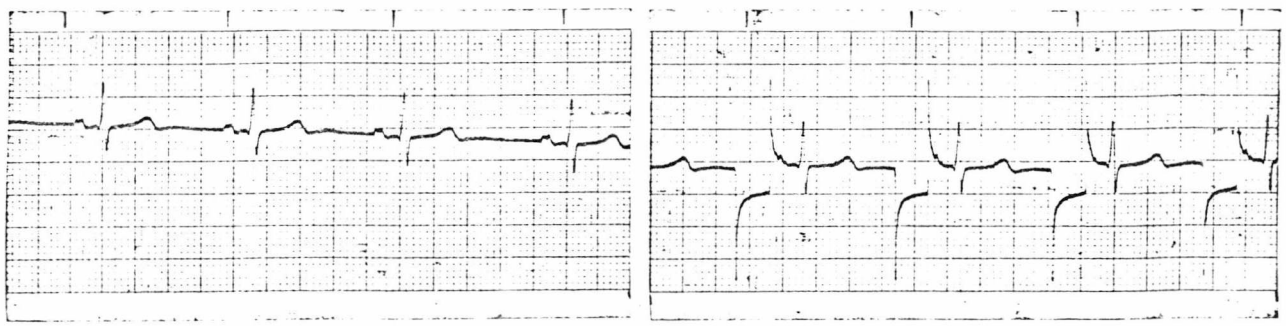
1mV 0.1sec pulse



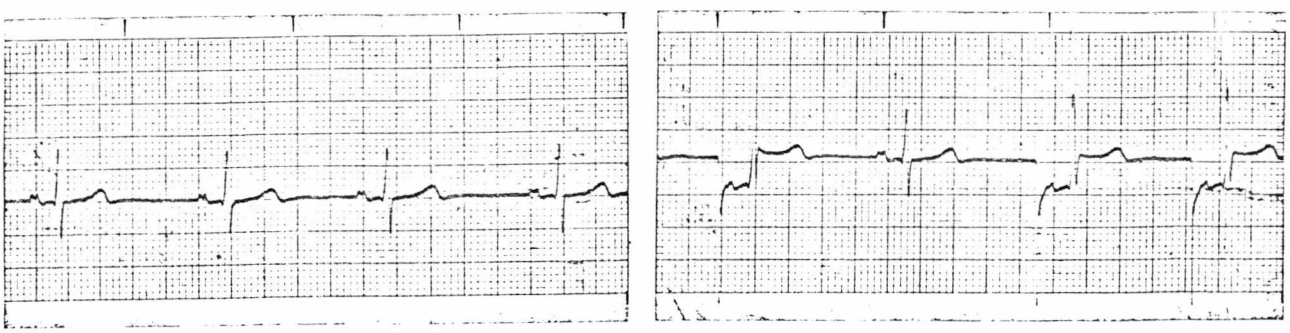
Rin = 100kΩ



Rin = 47kΩ



Rin = 22kΩ



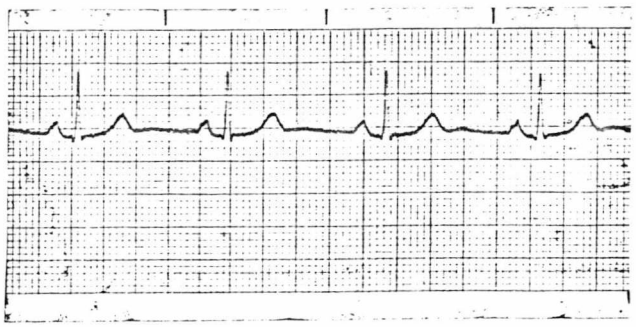
Gain x 2

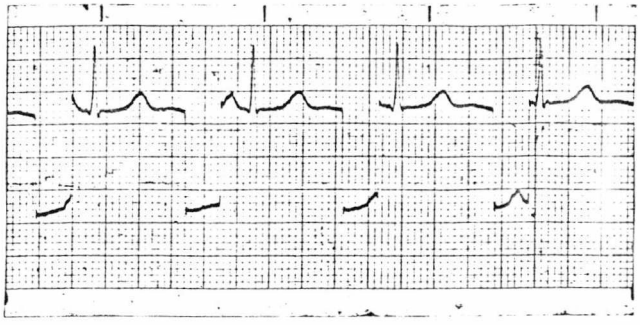
Rin = 10kΩ

FIG. 13b The effect upon the ECG of reducing Rin - large metal plate electrodes

ECG

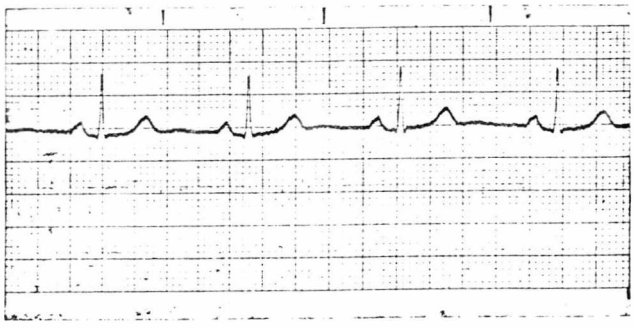
1mV 0.1sec pulse

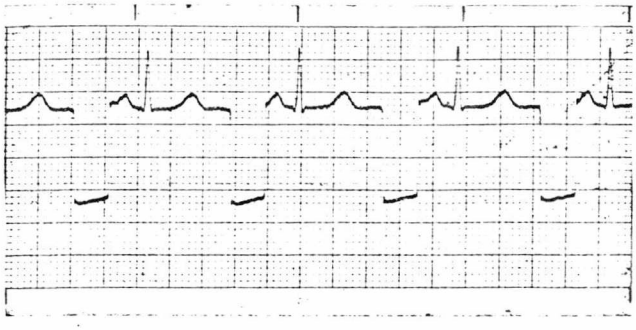




Rin = 2.2MΩ

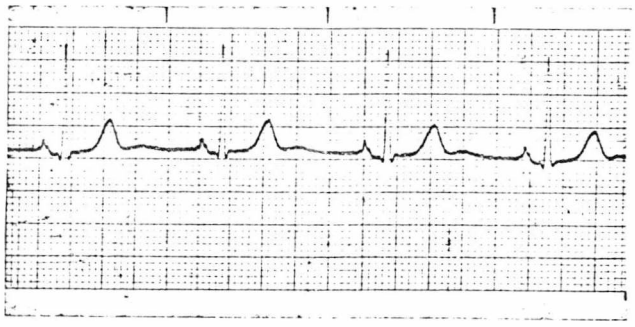
Cin = 0.025μF

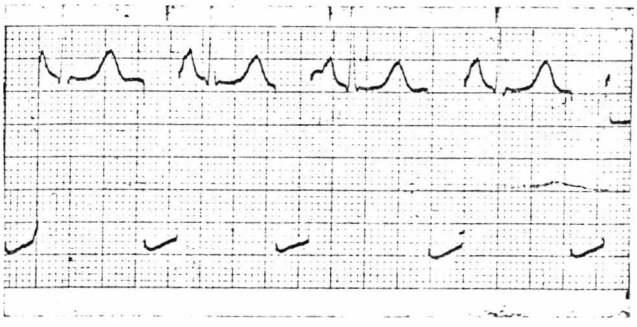




Rin = 1MΩ

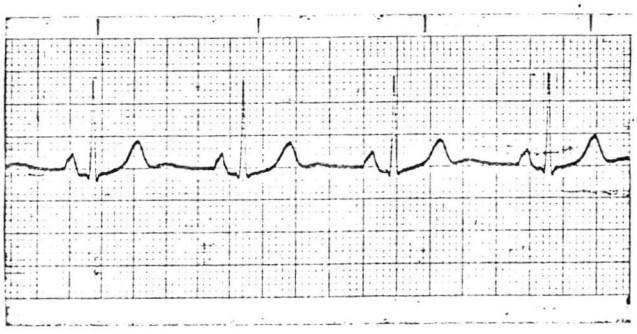
Cin = 0.05μF

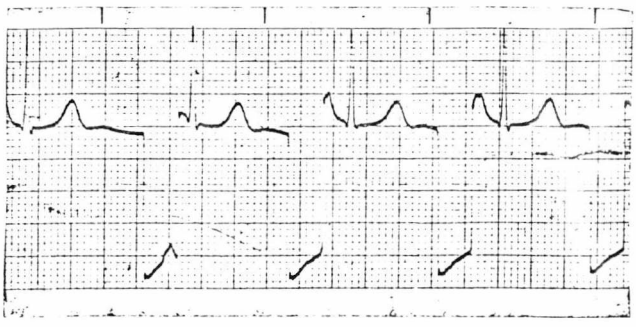




Rin = 470kΩ

Cin = 0.1μF





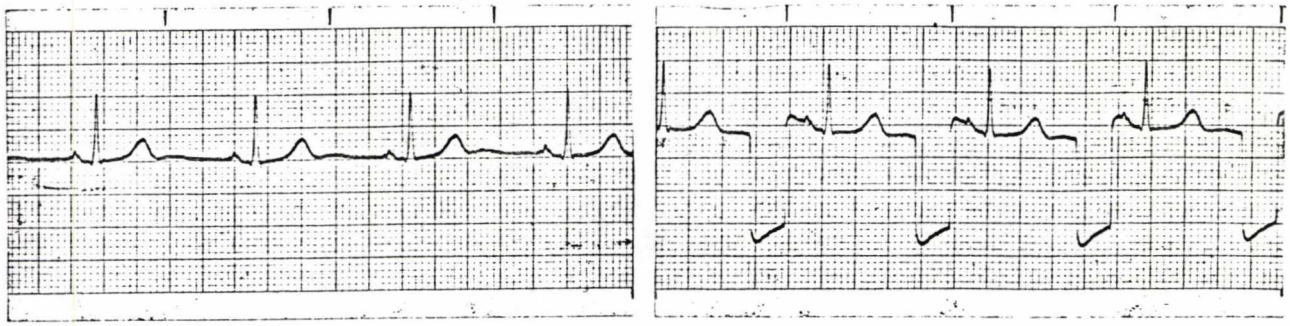
Rin = 220kΩ

Cin = 0.15μF

FIG. 14a

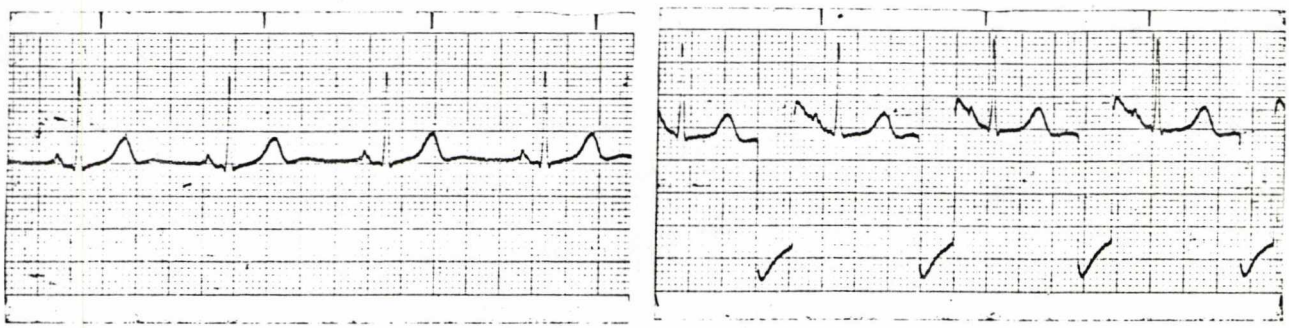
ECG

1mV 0.1sec pulse



Rin = 100kΩ

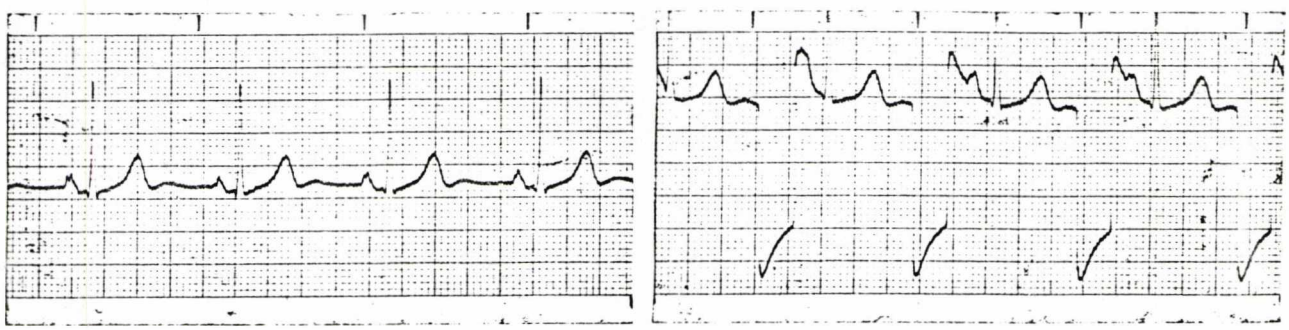
Cin = 0.35μF



Gain x 2

Rin = 47kΩ

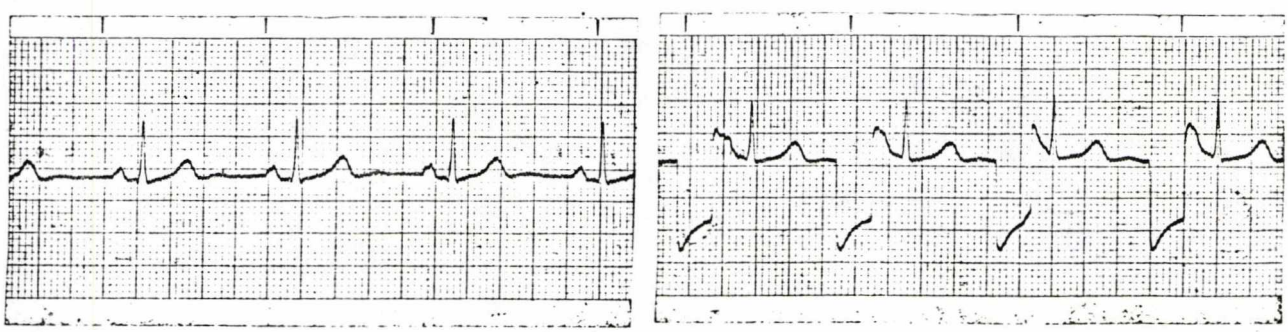
Cin = 0.7μF



Gain x 5

Rin = 22kΩ

Cin = 1.3μF



Gain x 5

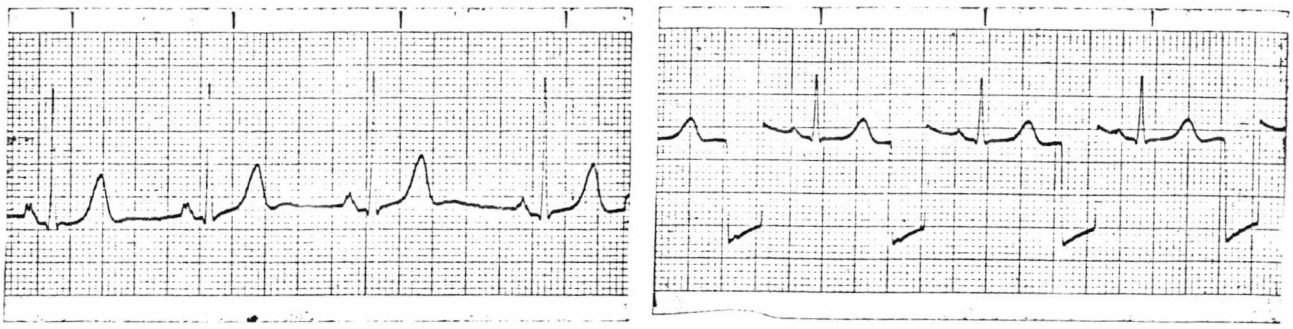
Rin = 10kΩ

Cin = 2.6μF

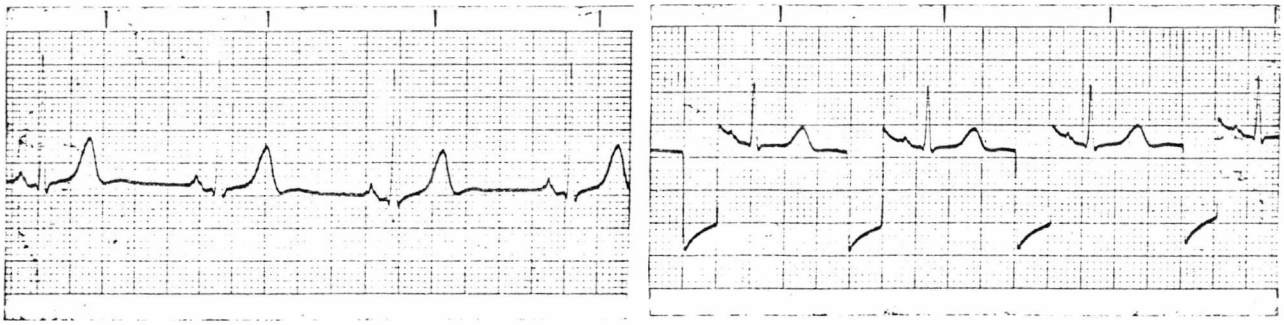
FIG. 14b The effect upon the ECG of reducing Rin with suitable compensation - large metal plate electrodes

ECG

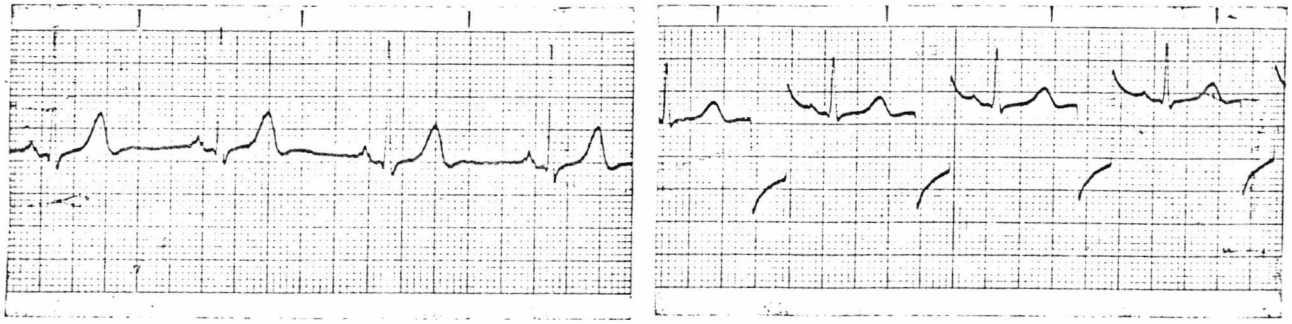
1mV 0.1 sec pulse



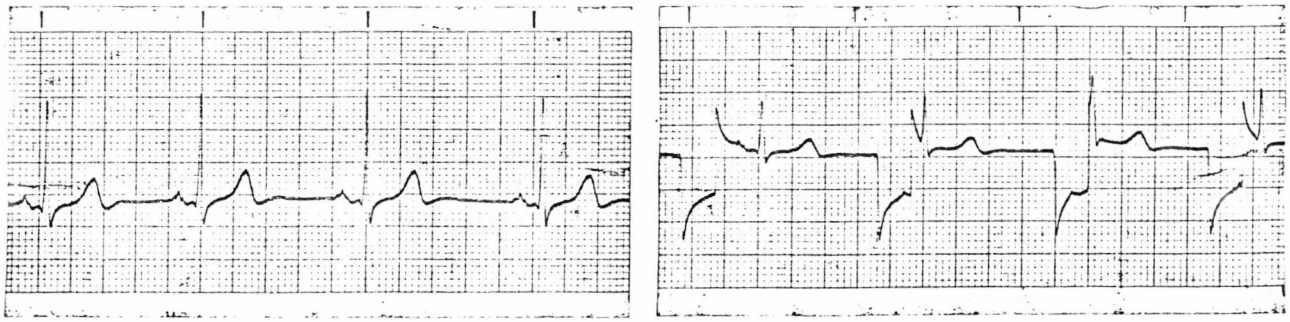
Rin = 2.2MΩ



Rin = 1MΩ



Rin = 470kΩ



Rin = 220kΩ

FIG. 15a

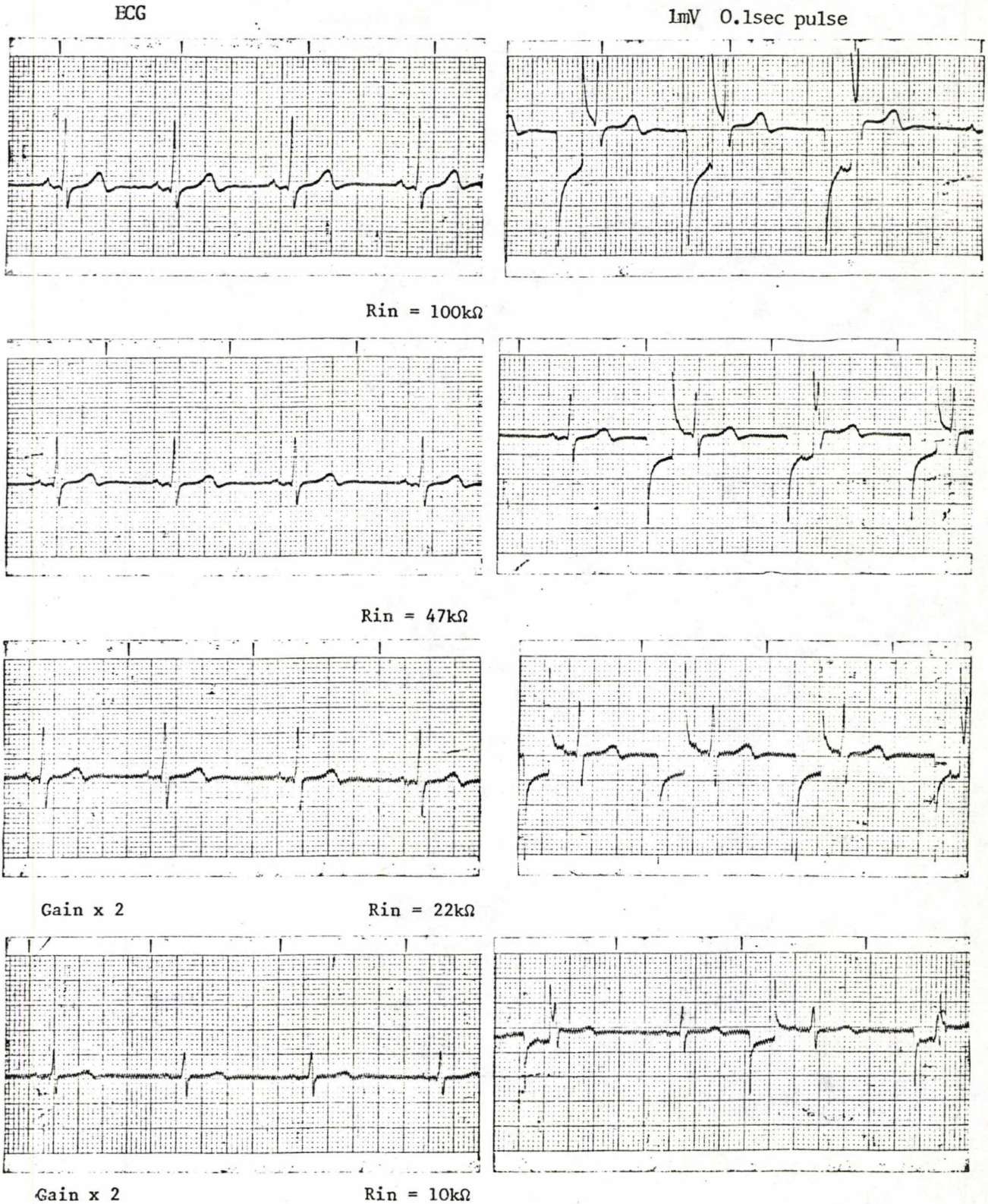
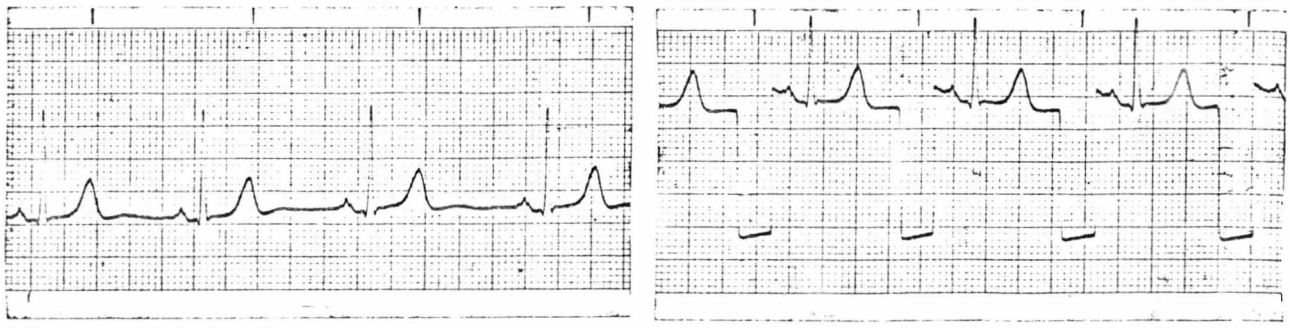


FIG. 15b The effect upon the ECG of reducing Rin small metal plate electrodes.

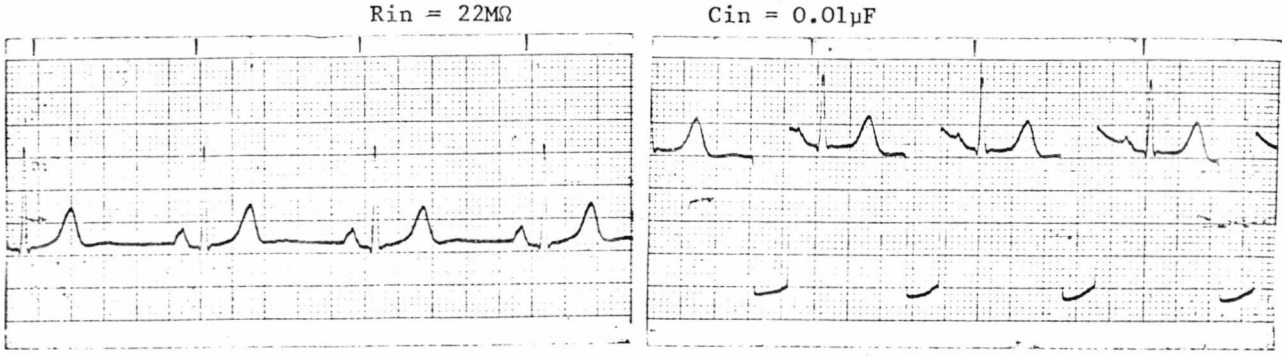
ECG

1mV 0.1sec pulse



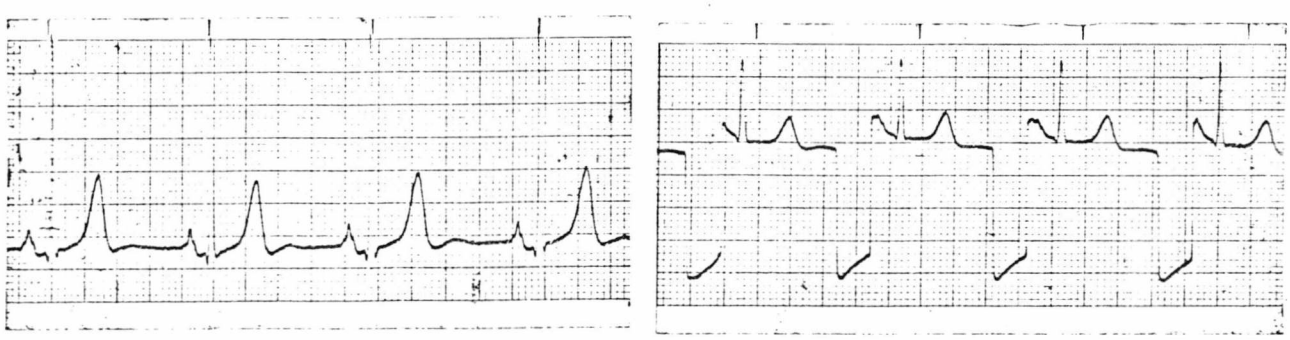
Rin = 22MΩ

Cin = 0.01μF



Rin = 1MΩ

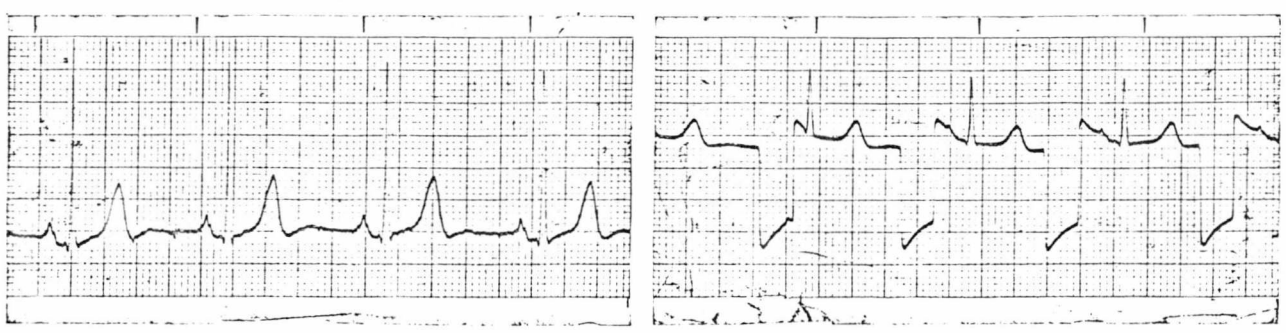
Cin = 0.02μF



Gain x 2

Rin = 470kΩ

Cin = 0.07μF



Gain x 2

Rin = 220kΩ

Cin = 0.15μF

FIG. 16a

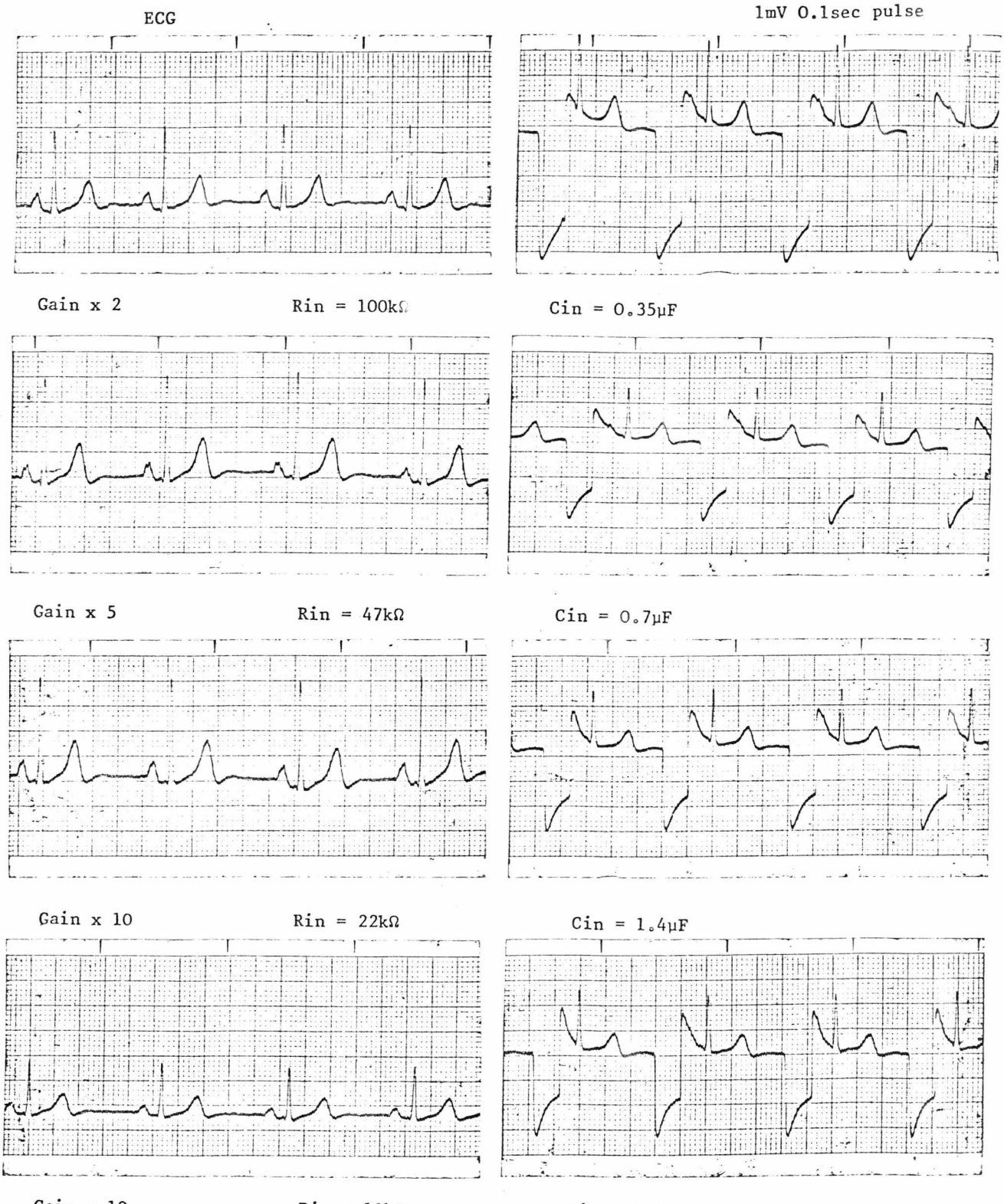
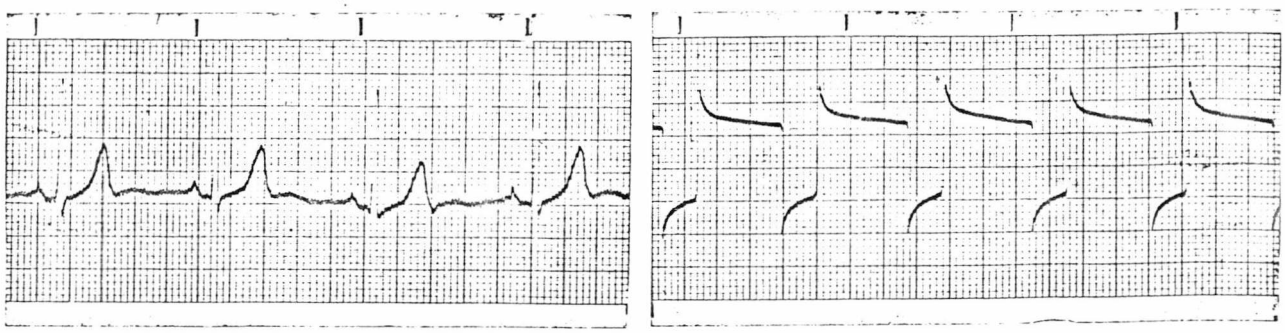


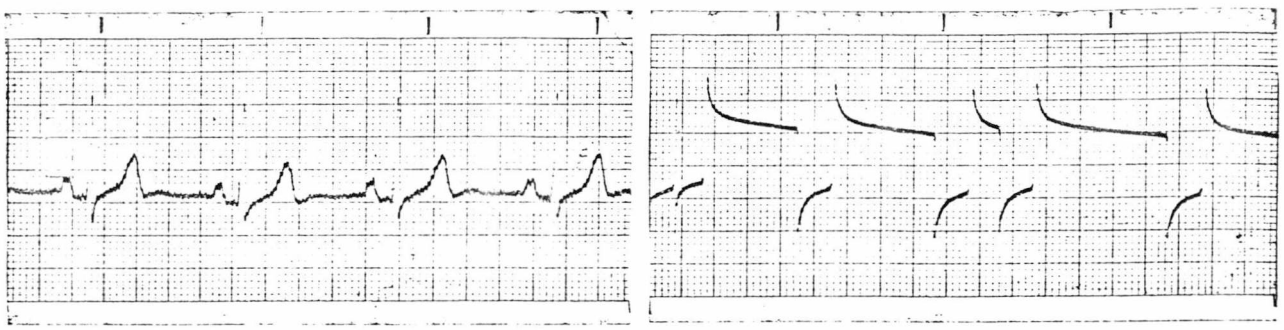
FIG. 16b The effect upon the ECG of reducing Rin with suitable compensation - small metal plate electrodes

ECG

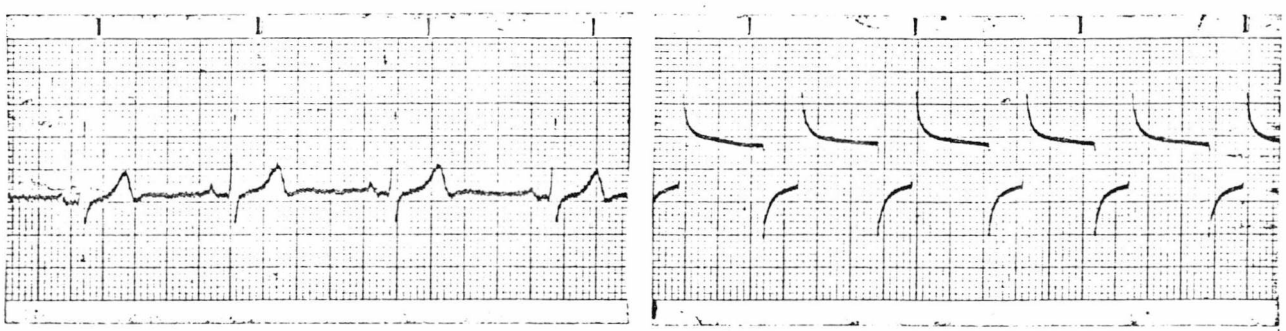
1mV 0.1sec pulse



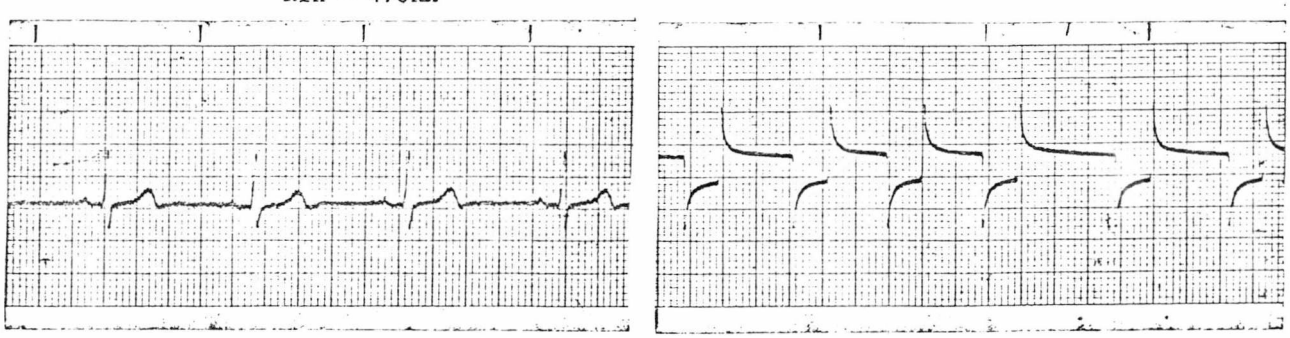
Rin = 2.2MΩ



Rin = 1MΩ



Rin = 470kΩ



Rin = 220kΩ

FIG. 17a

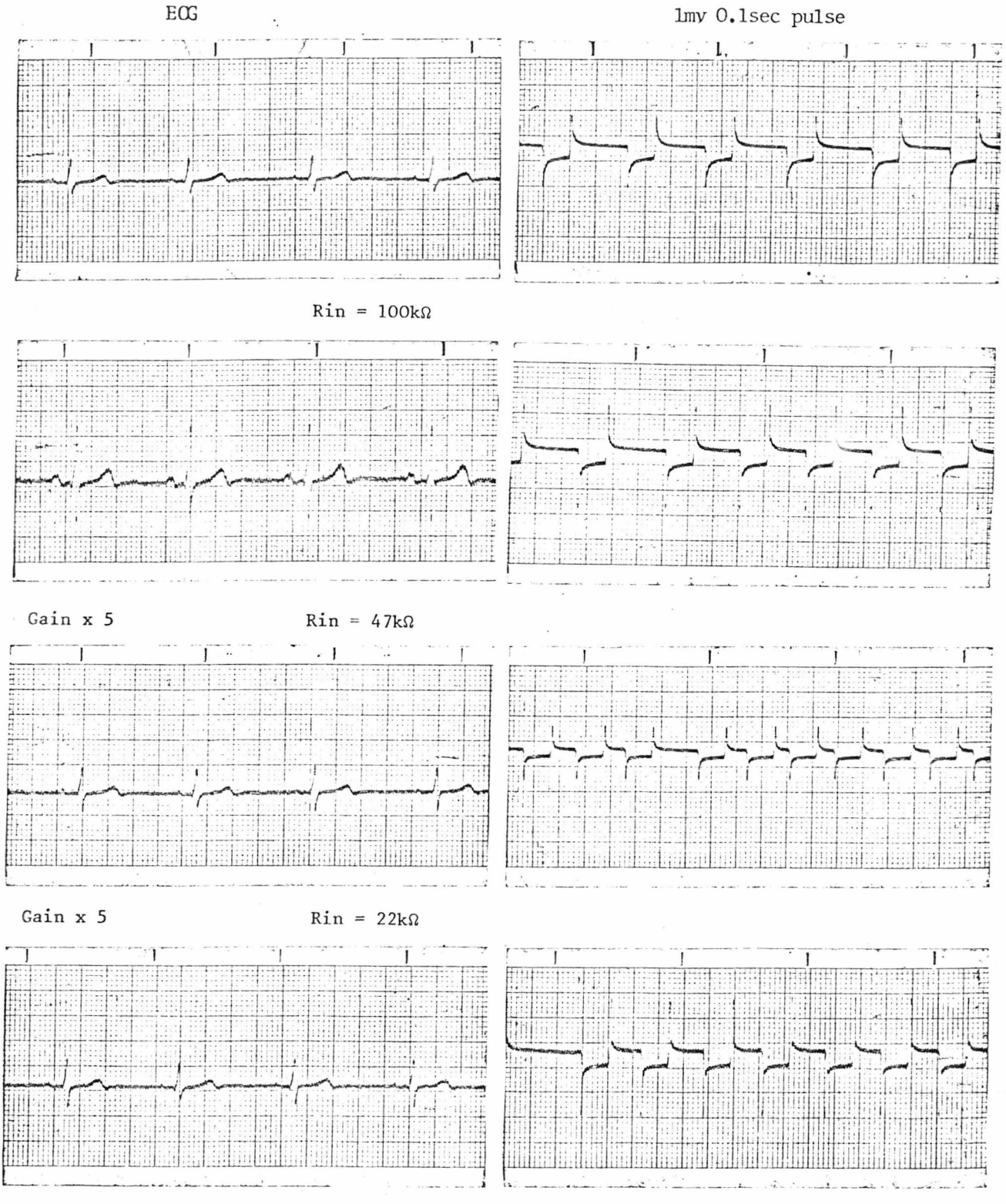
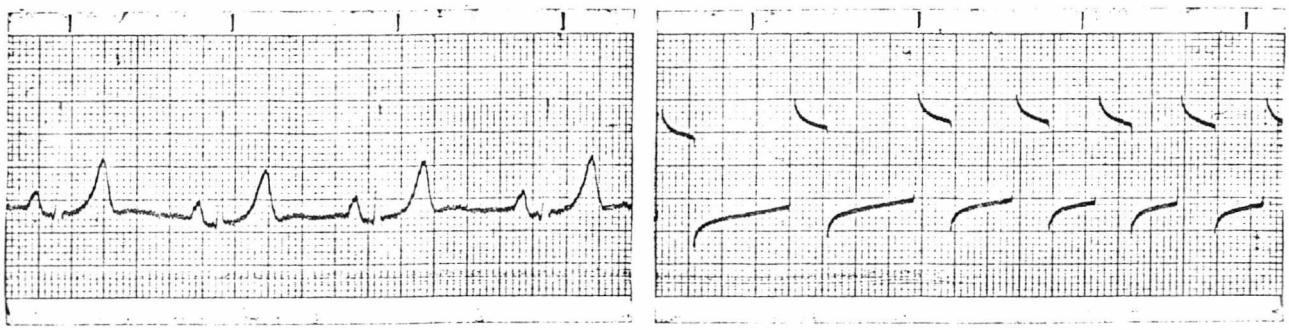


FIG. 17b The effect upon the ECG of reducing Rin - silver/silver chloride electrodes

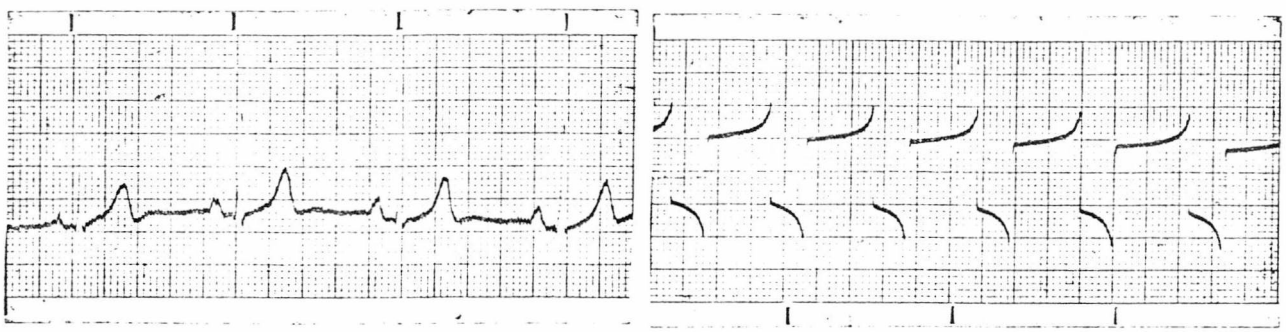
ECG

1mV 0.1sec pulse



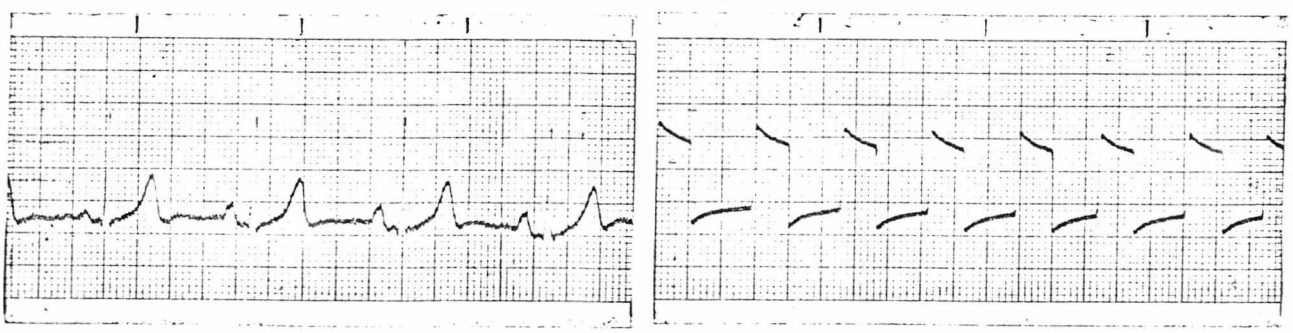
Rin = 2.2MΩ

Cin = 0.005μF



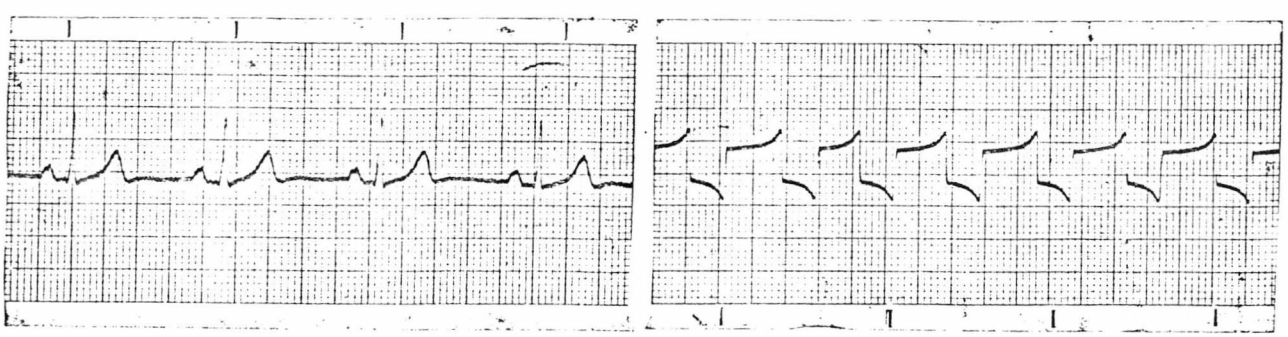
Rin = 1MΩ

Cin = 0.01μF



Rin = 470kΩ

Cin = 0.04μF



Rin = 220kΩ

Cin = 0.08μF

FIG. 18a

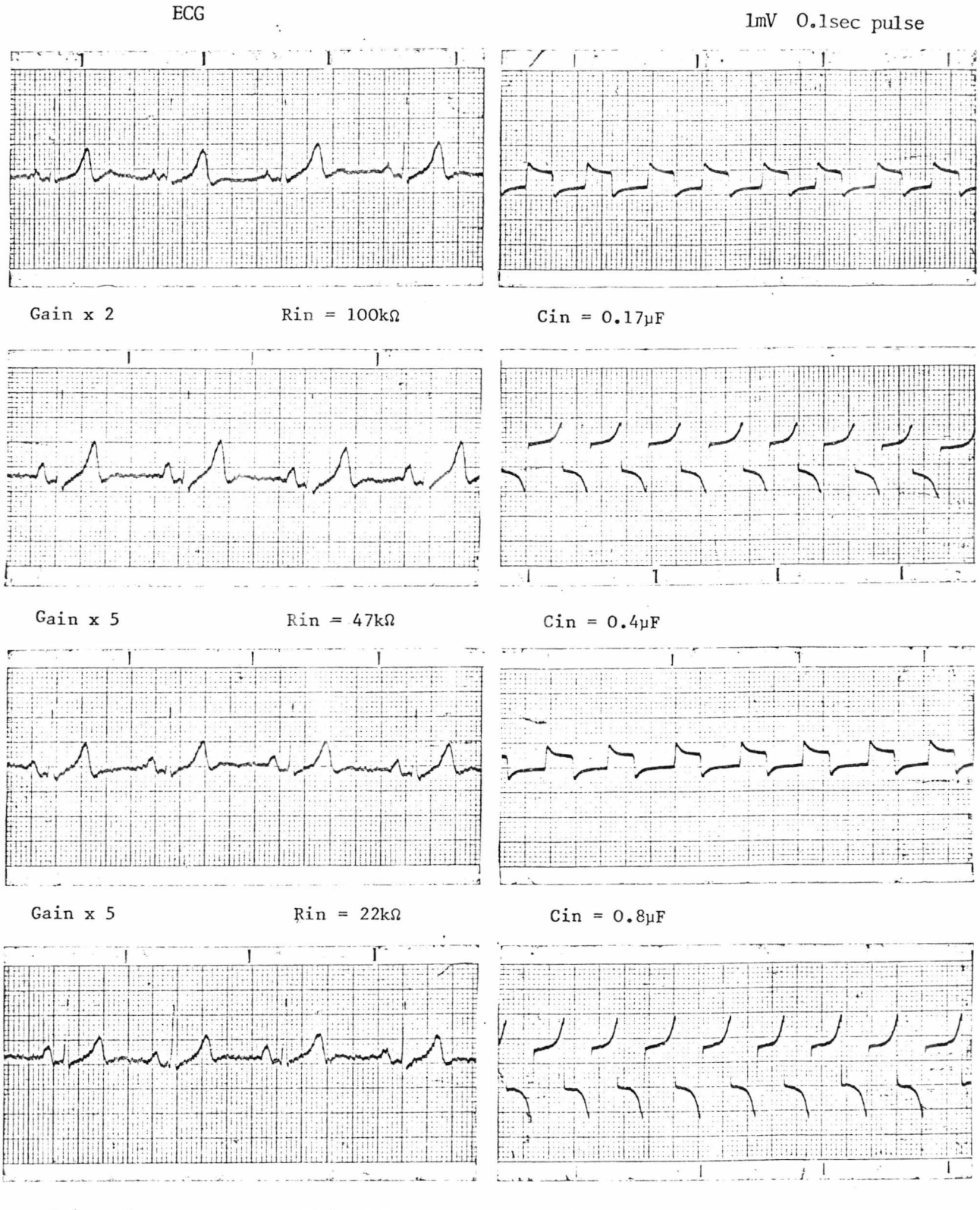


FIG. 18b The effect upon the ECG of reducing Rin with suitable compensation - silver/silver chloride electrodes

silver/silver chloride electrode respectively.

When the ECGs are compensated against a reference from a normal machine (Cambridge VS4), and the results taken, then they appear in *Figs. (14), (16)* and *(18)* for the same pairs of electrodes as used previously. It can be seen that it is possible to compensate all the values of R_{in}, for all the electrodes, and that the compensated ECGs all resemble one another - a very different situation from those recordings taken without compensation.

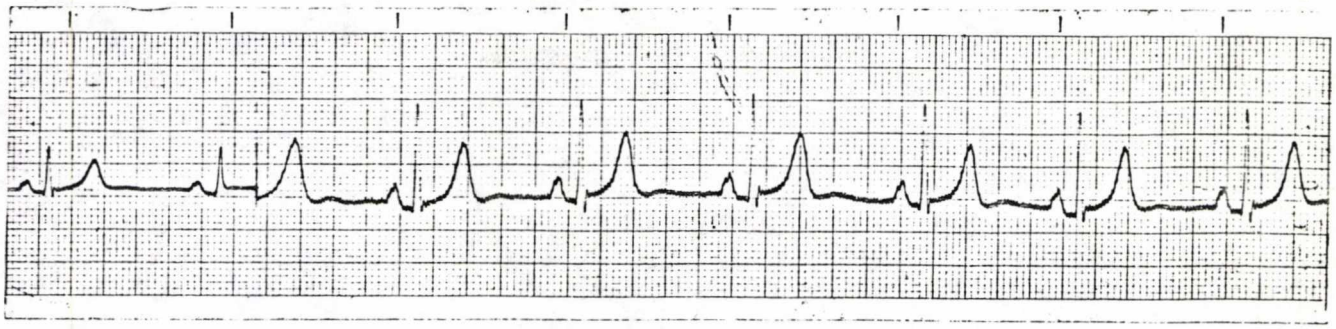
Some of the recordings are compensated better than others e.g. from *Fig. (14a)*, the traces from the 470KΩ and 220KΩ input resistors are better than that of the 2.2MΩ. There is a U wave which is clearly discernible in the control (2.2MΩ, *Fig. (3a)*), and which is also displayed in 470KΩ and 220KΩ recordings of *Fig. (14a)*, but is not at all clear in the 2.2MΩ version.

There are other similar examples, but in general, the compensated traces in *Figs. (14), (16), (18)* all resemble their controls and with small adjustments in compensation, the differences could be easily removed.

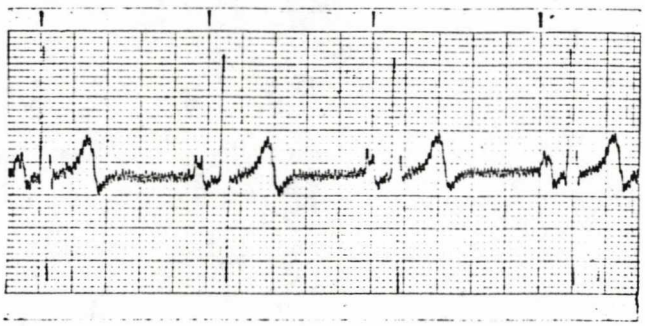
This work shows that it is possible to record clinically useful ECGs when using low values of resistance (10KΩ to 220KΩ) across the input terminals of the amplifier.

7.2c The Compensation of R_{in}

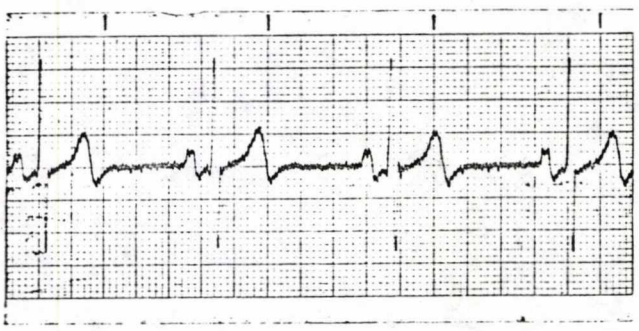
To illustrate the effect of adding compensation capacitance to an input resistor, the results of *Fig. (19)* were obtained. A control was recorded and appears at the beginning and end of the figure. Then R_{in} = 47KΩ was selected, and this can be seen



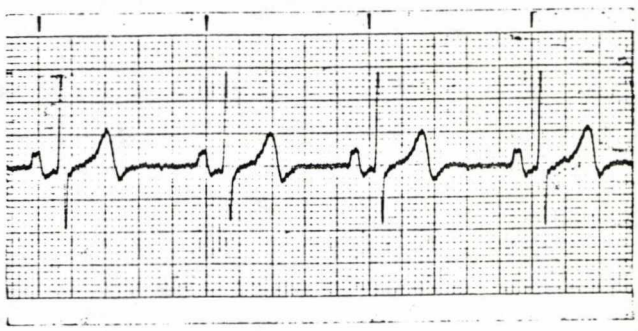
Control (2.2MΩ)



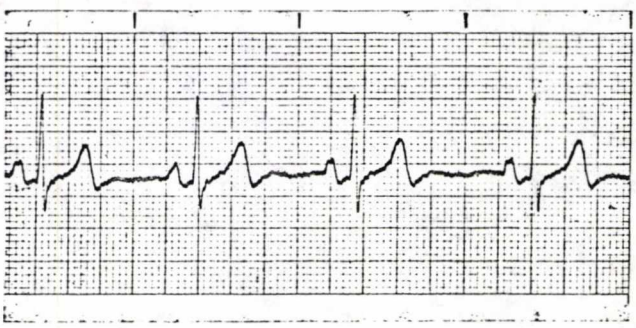
47kΩ above



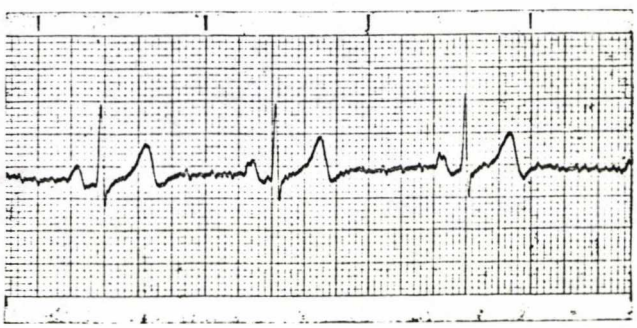
0.1μF



0.2μF

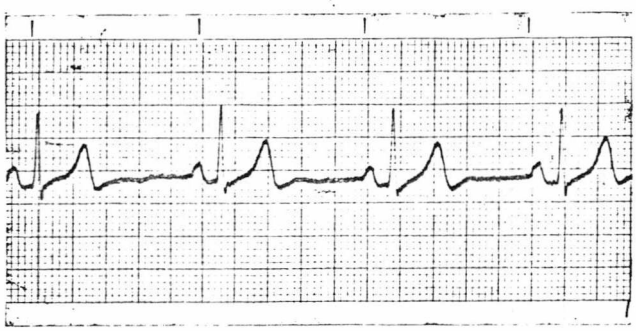


0.3μF

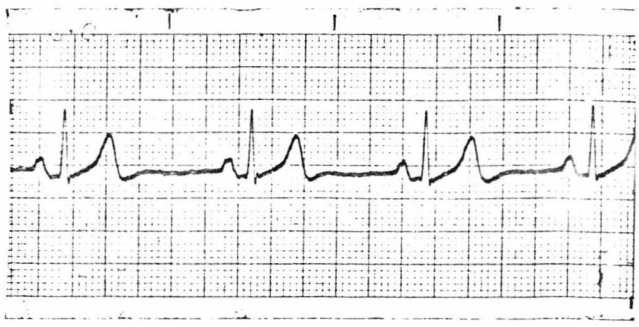


0.4μF

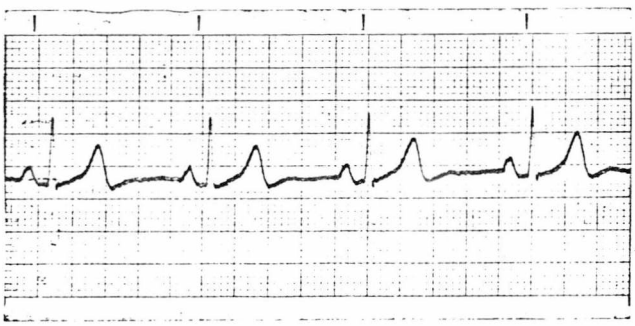
FIG. 19a



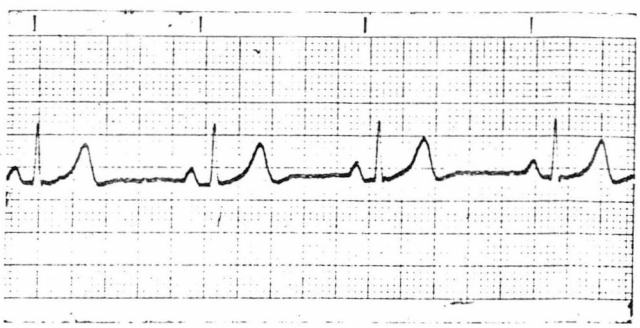
0.5μF



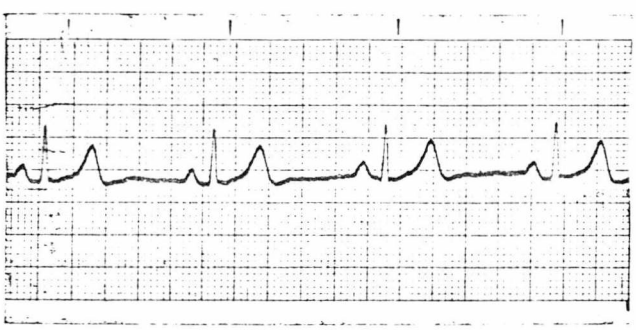
0.6μF



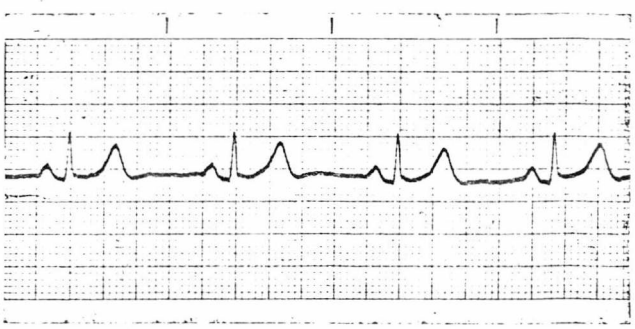
0.7μF



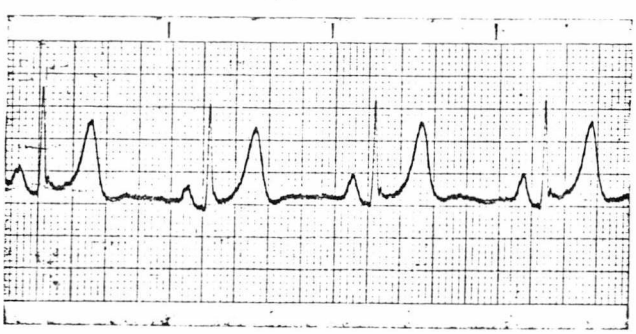
0.8μF



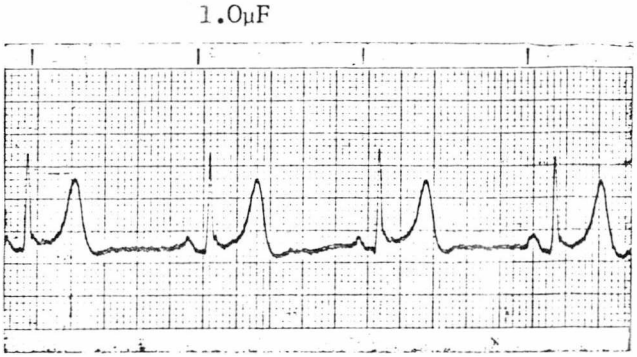
0.9μF



1.0μF

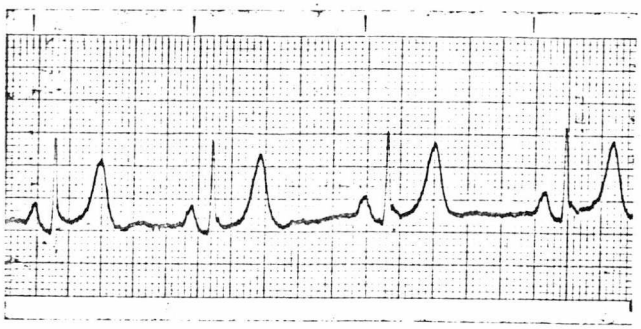


1.1μF

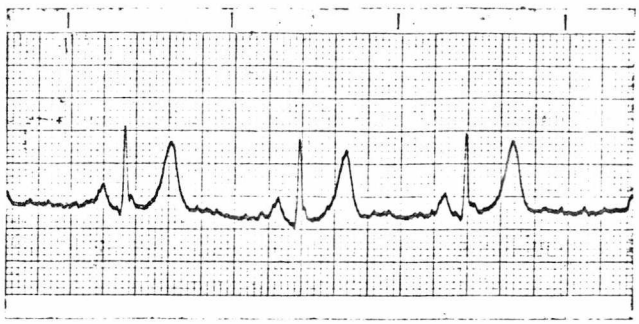


1.2μF

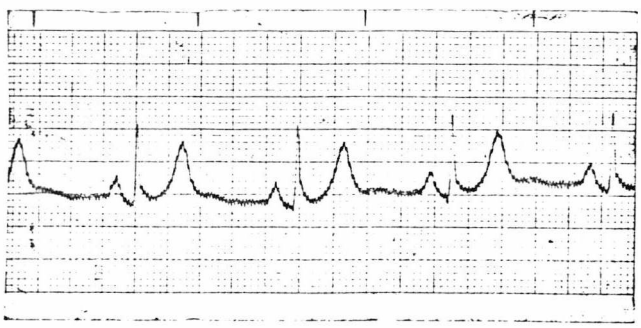
FIG. 19b



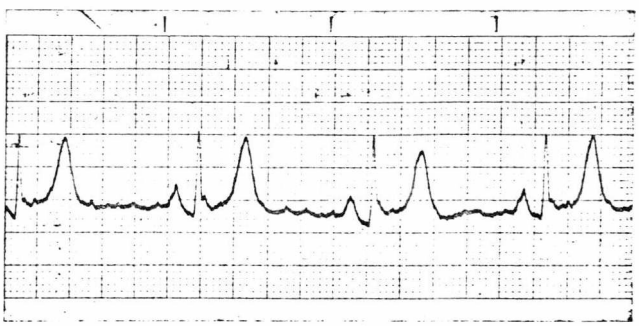
1.3μF



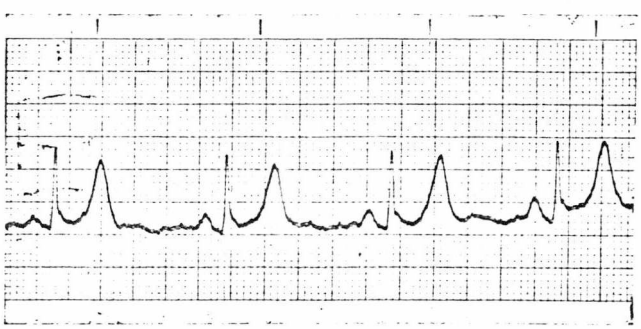
1.4μF



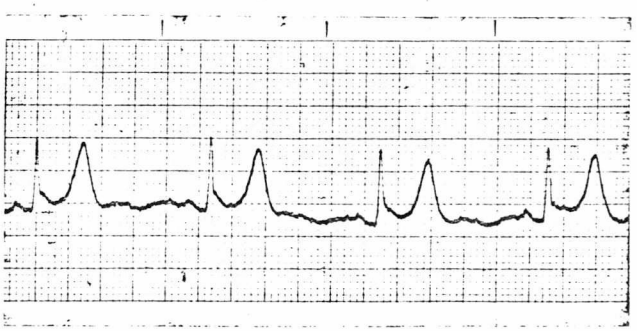
1.5μF



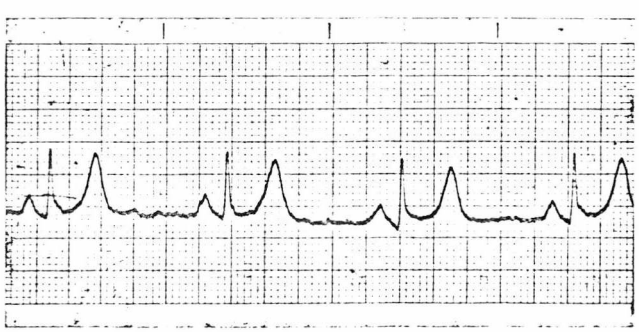
1.6μF



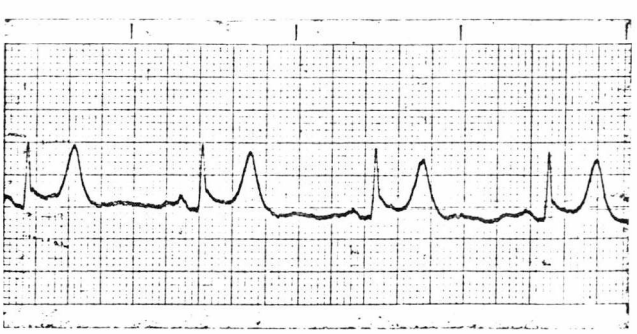
1.7μF



1.8μF



1.9μF



2.0μF

FIG.19c

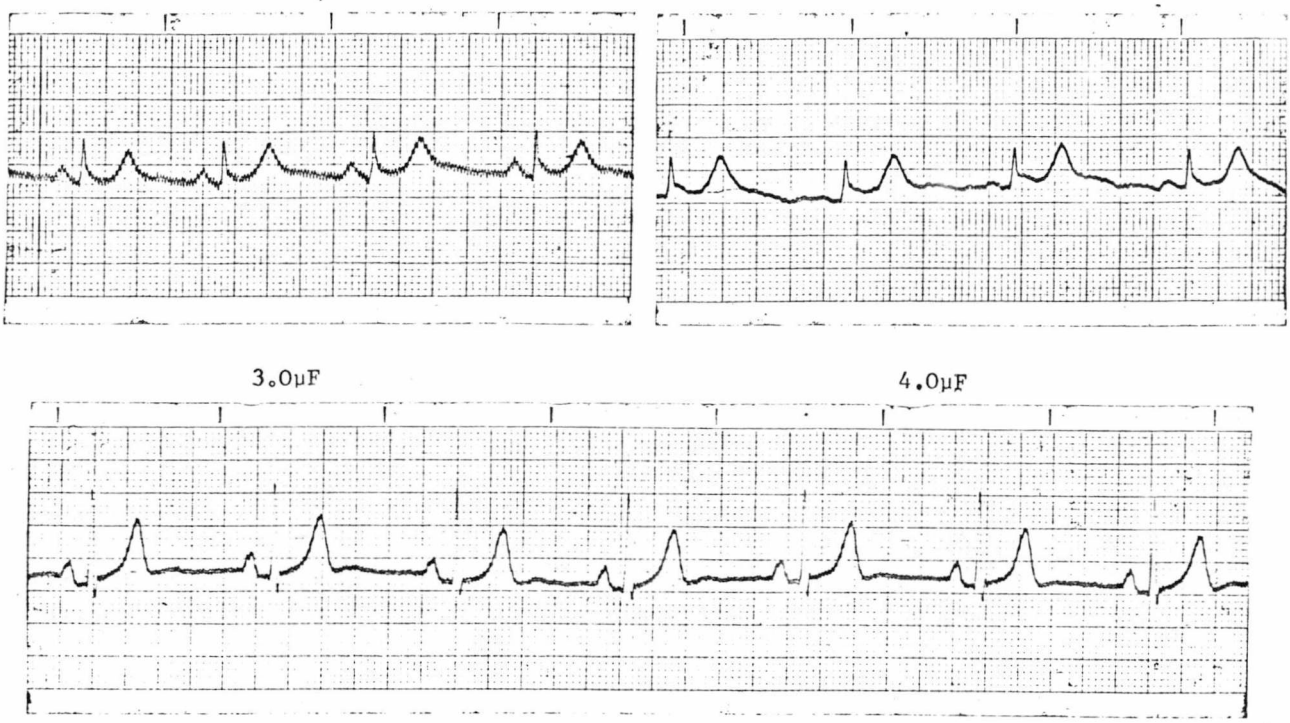


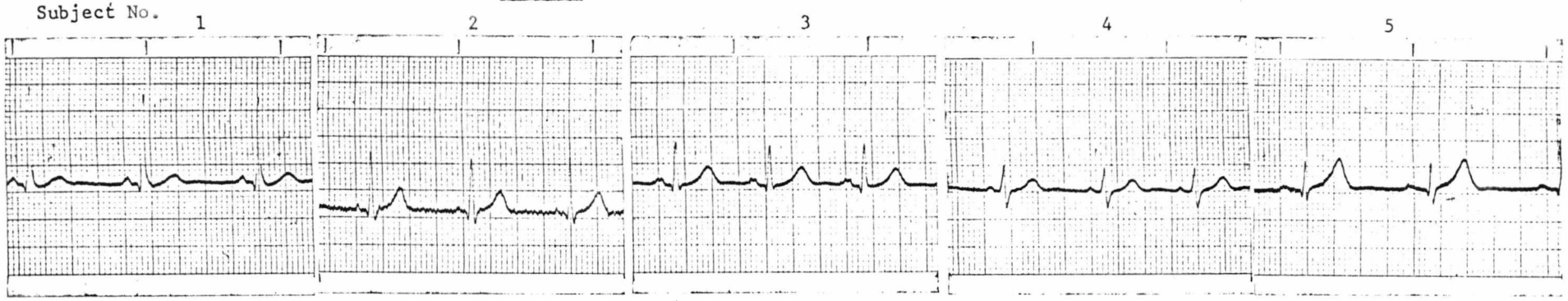
FIG. 19d The effect upon the ECG of successively adding compensation capacitors to Rin = 47kΩ. The figures underneath each recording refer to the added compensation.

directly under the reference in *Fig. (19a)*. Compensation was added to this resistor in steps of 0.1μF from 0.1μF to 2.0μF and then 3.0μF and 4.0μF were added. The various stages of change of the ECG can be seen from when it is biphasic first of all, to when it is normal at 0.6μF, and then above this value more distortion occurs - with S-T segment elevation and reduced R wave height until the R-wave and T-wave seem to merge at the highest value of compensation. This shows that there is an optimum value of compensation and that it is quite easy to overcompensate the input resistance as well as undercompensate it.

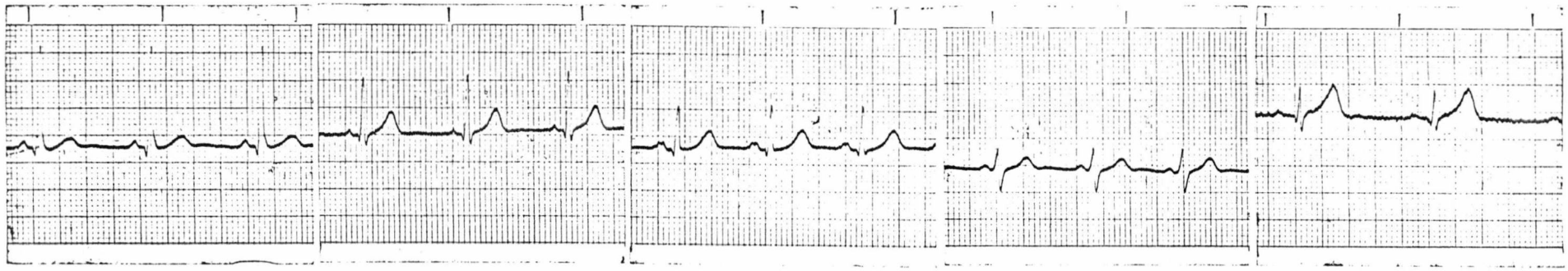
7.2d The Compensation of Electrodes between Patients

From section 7.2c it can be seen how important is the correct choice of compensating capacitor. As this was varied from 0.4μF to 0.8μF then the ECG had changed from being undercompensated to overcompensated. This raises the question of what happens when electrodes are placed on different people. Does the compensation have to be adjusted each time, or will a standard capacitor be sufficient for all cases - especially in the light of the experience of Berson and Pipberger and Almasi and Schmitt (references 41 and 42, Chapter 6)? An experiment was performed to investigate this, and the results can be seen in *Figs. (20)* to *(25)*. The three common types of electrodes were placed on each of five subjects in turn and leads I, II and III were recorded - both a control from the normal ECG machine directly, and a set with only 47KΩ input resistance, with suitable compensation values chosen from the results of 7.2b (0.4μF for the silver/silver chloride type,

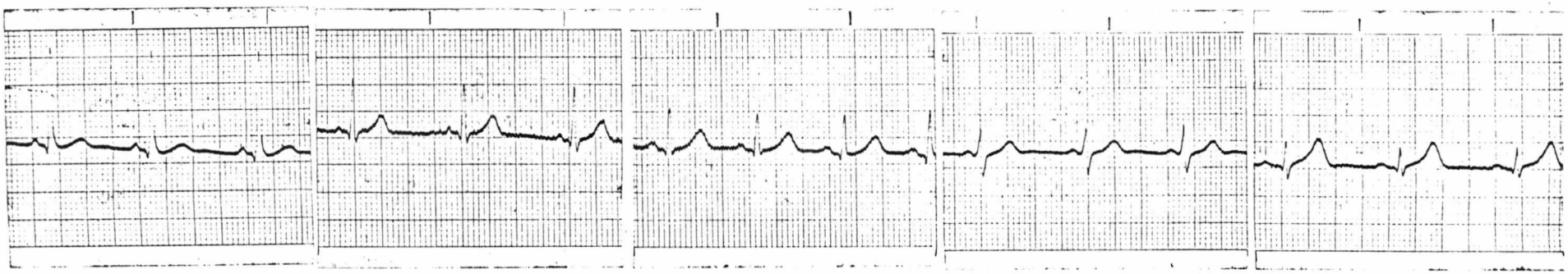
FIG. 20 Lead I - Control recordings



Small metal plate electrodes

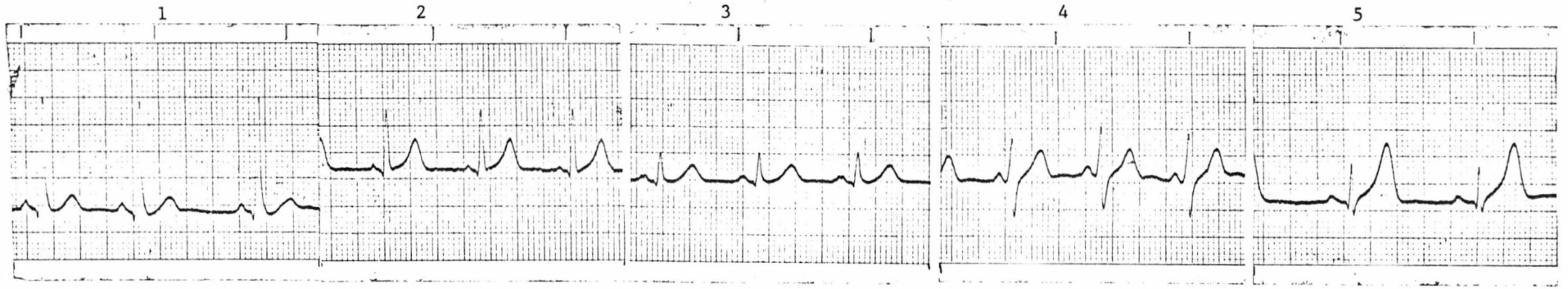


Large metal plate electrodes

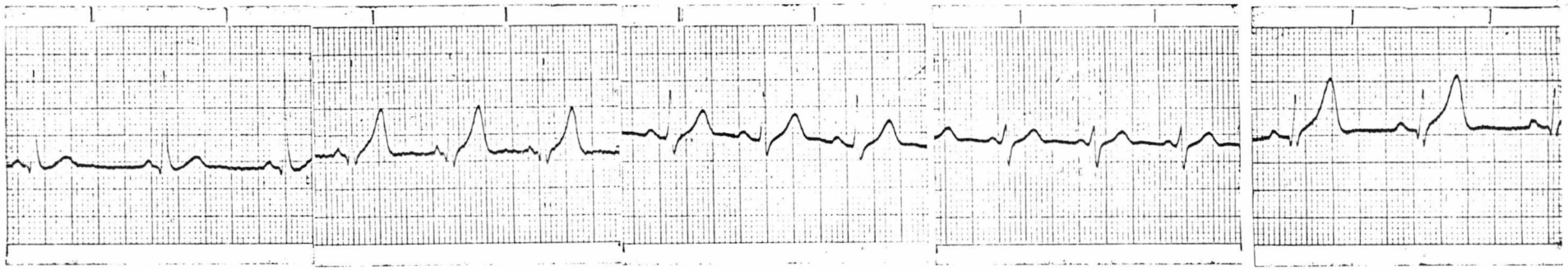


Silver/Silver chloride electrodes

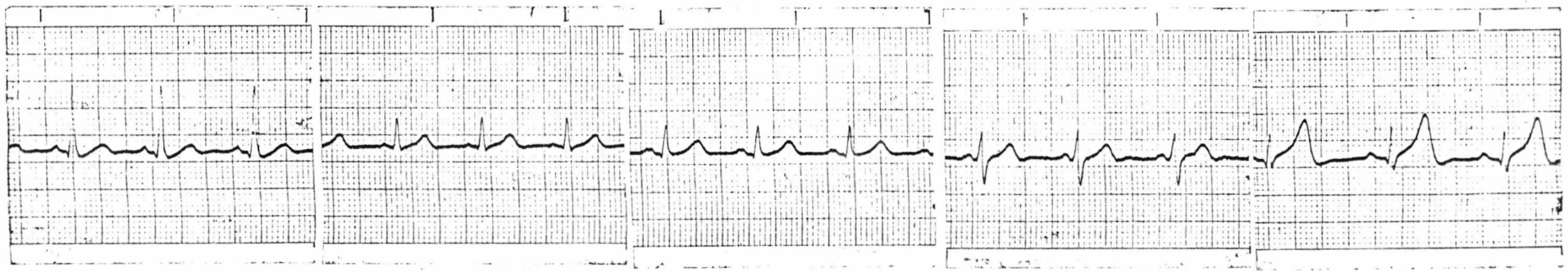
FIG. 21 Lead I - Compensated Input Resistance recordings



Small metal plate electrodes



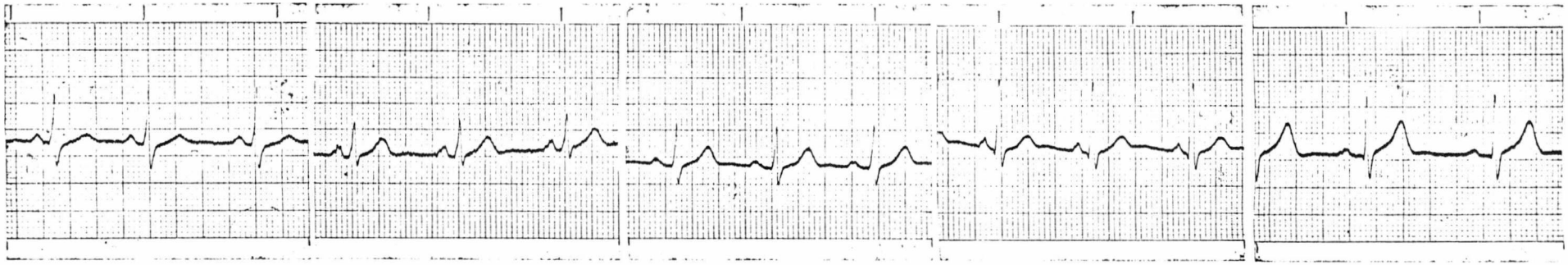
Large metal plate electrodes



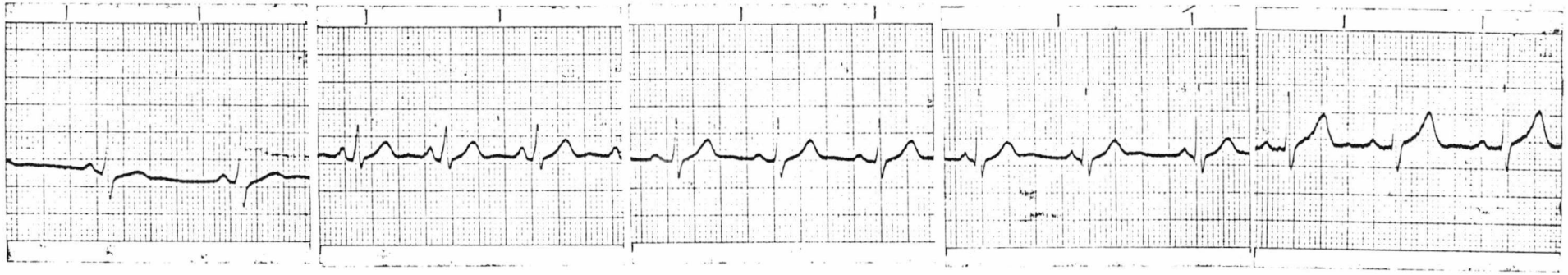
Silver/silver chloride electrodes

FIG. 22 Lead II - Control Recordings

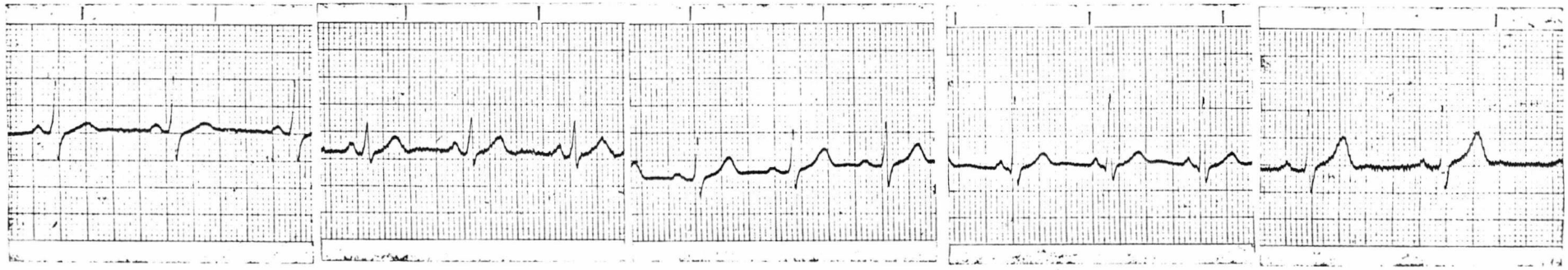
Patient No. 1 2 3 4 5



Small metal plate electrodes



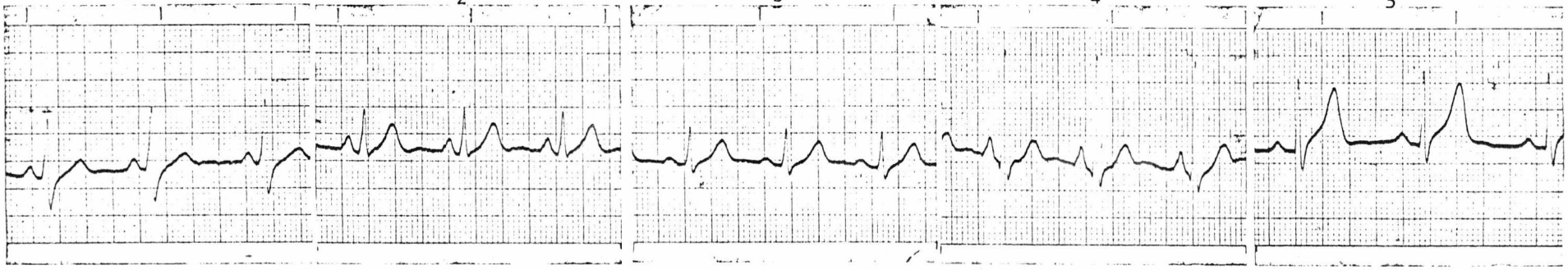
Large metal plate electrodes



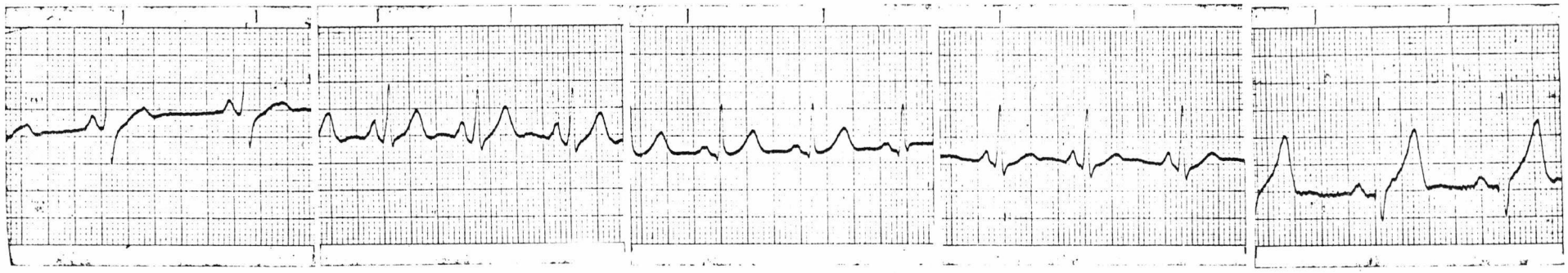
Silver/silver chloride electrodes

Patient No. 1 2 3 4 5

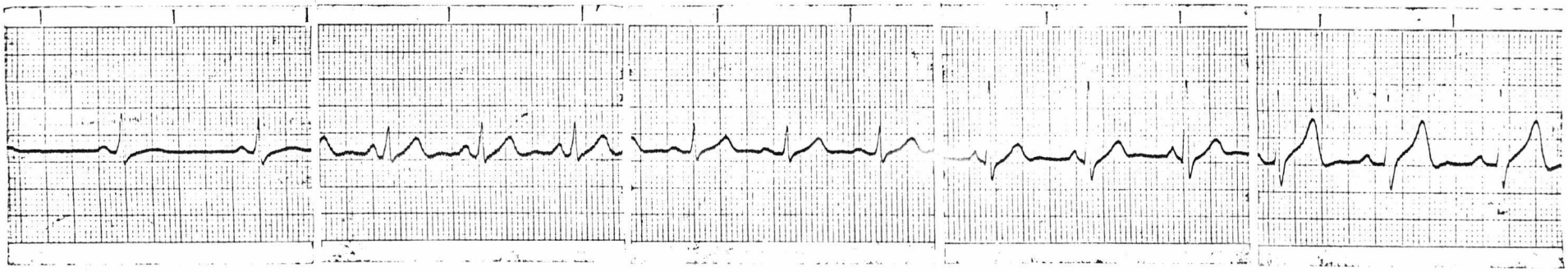
FIG. 23 Lead II - Compensated input resistance recordings



Small metal plate electrodes



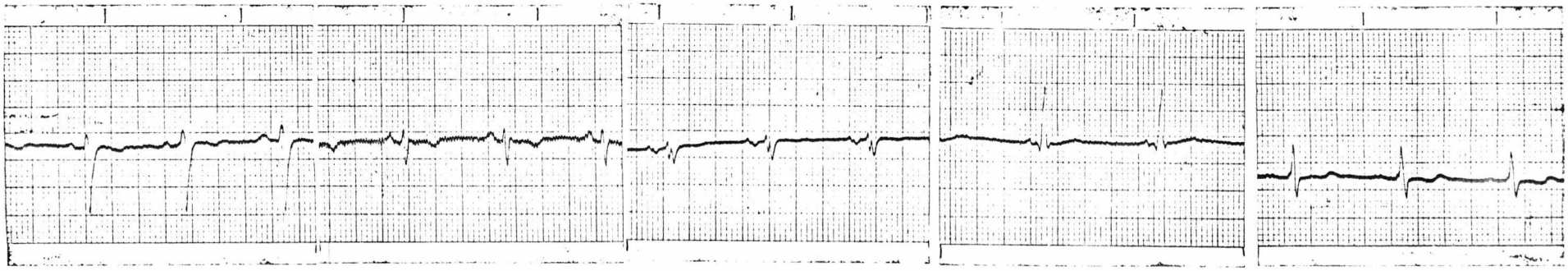
Large metal plate electrodes



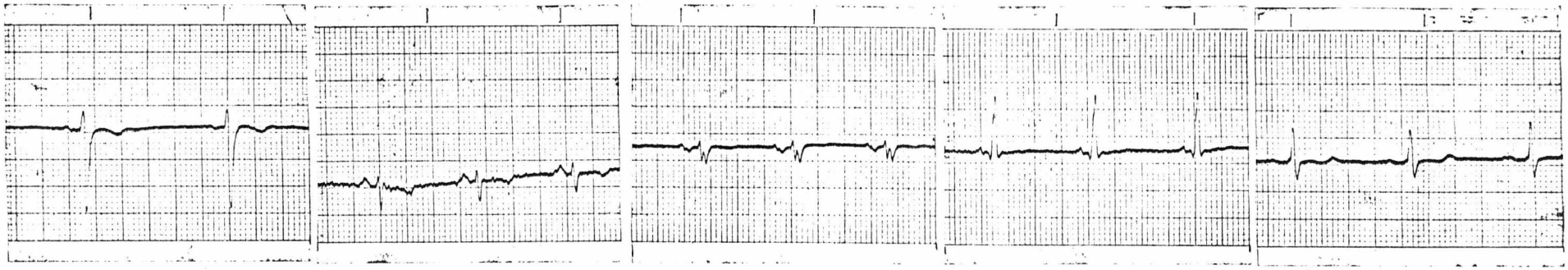
Silver/silver chloride electrodes

FIG. 24 Lead III - Control Readings

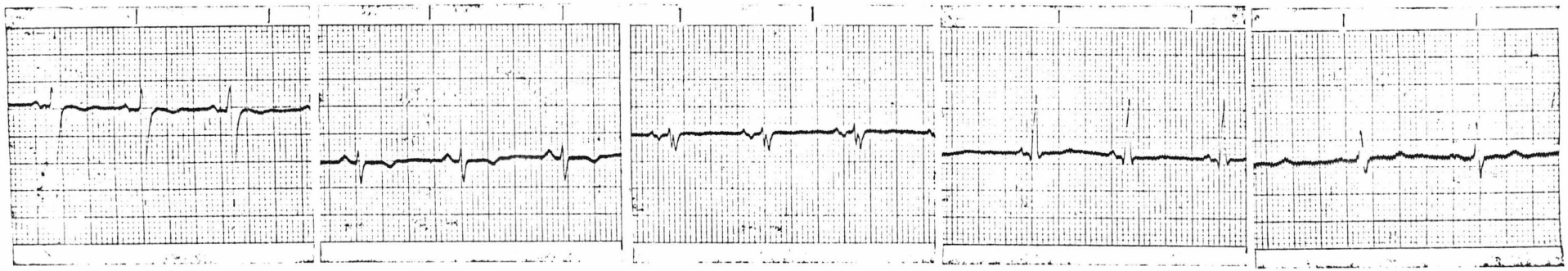
Patient No. 1 2 3 4 5



Small metal plate electrodes

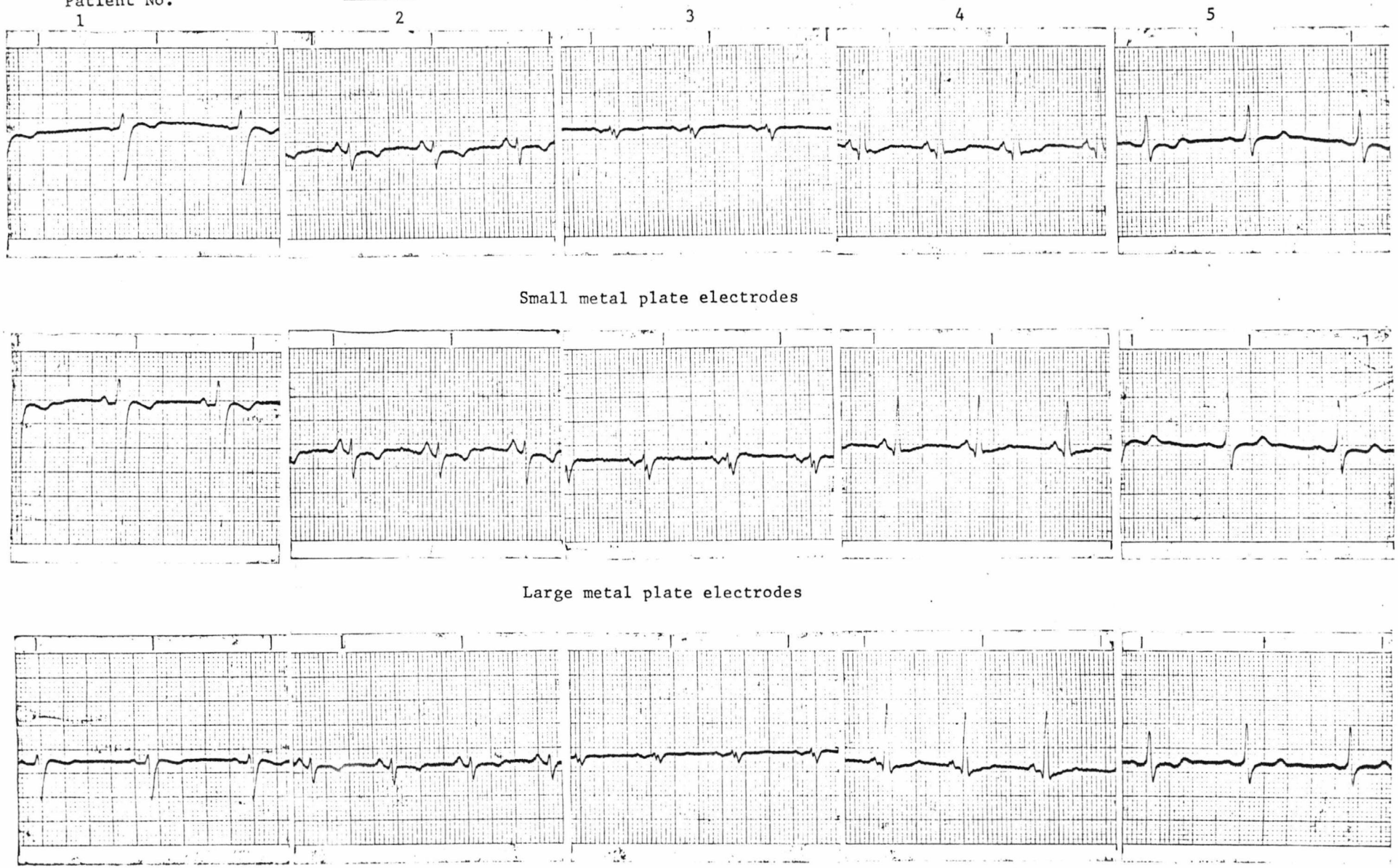


Large metal plate electrodes



Silver/silver chloride electrodes

FIG. 25 Lead III - Compensated input resistance recording



and 0.7μF for both types of metal plate electrodes).

At first sight the agreement between the controls of each subject and the low impedance recordings are very good for each lead; however, closer investigation does show up some differences. If we examine lead I taken from the small metal plate electrodes it can be seen that agreement for subject 1 between control *(Fig. (20))* and low impedance input *(Fig. (21))* is all right, whilst subjects 2 and 3 are overcompensated, with subjects 4 and 5 reasonably compensated again. For the large metal plate electrodes, subjects 1 and 2 are correctly compensated, whilst subject 3 is undercompensated and subjects 4 and 5 are all right.

These comparisons can be repeated throughout the results and more differences discovered. They are, in general, not severe and in view of the results of 7.2c not very much change in compensation would be needed to rectify the situation - ±0.1μF in most cases. It does mean, however, that for any clinical decision making on the results of a patient, the compensation would have to be special to that patient, whilst for general trend observations then a standard compensation per electrode type would be sufficient.

7.2e Frequency Spectra of Compensated and Uncompensated ECG Recordings

It was decided to examine the frequency spectra of compensated and uncompensated ECGs and to compare them with a reference as a further investigation of the effect of the amplifier input upon the signal. One second of each recording was taken, including one whole complex and transferred onto

graph paper (by means of a photographic enlarger). This was then digitised by hand into 128 samples which were put onto paper tape, and analysed by a fast Fourier Transform programme. The results were plotted out in frequencies that were harmonics of 1Hz. Although the analysis included components up to 64Hz, only the first 32 were actually plotted onto the graphs of *Figs. (26)* to *(29)*. An analysis was performed on a reference sample *(Fig. (26))* and on two recordings from each type of electrode used (silver/silver chloride - *Fig. (27)*, large metal plate type - *Fig. (28)*, small metal plate type - *Fig. (29)*). The input resistance was 10KΩ, and a suitable compensation capacitance was used in each case.

The reference spectrum of *Fig. (26)* is consistent with those found by other workers (references 2,3,4, Chapter 4). In each of the other Figures it can be seen that the spectrum of the uncompensated recording shows less low frequency content than its compensated companion - and also that the spectra of the latter are similar to that of the reference one. We can thus conclude from the latter observation that since all four spectra are similar, then the compensated ECGs must be similar to the reference ECG obtained normally. The spectra of the uncompensated ECGs are a further illustration of the loss of low frequencies that is obtained when the amplifier input time constant is much less than that of the electrodes.

7.3 THE C.M.R.R. AND THE AMPLIFIER INPUT RESISTANCE

A model circuit was constructed of the common mode rejection circuit using resistors as the electrode impedances, and the storage oscilloscope's differential plug-in unit as

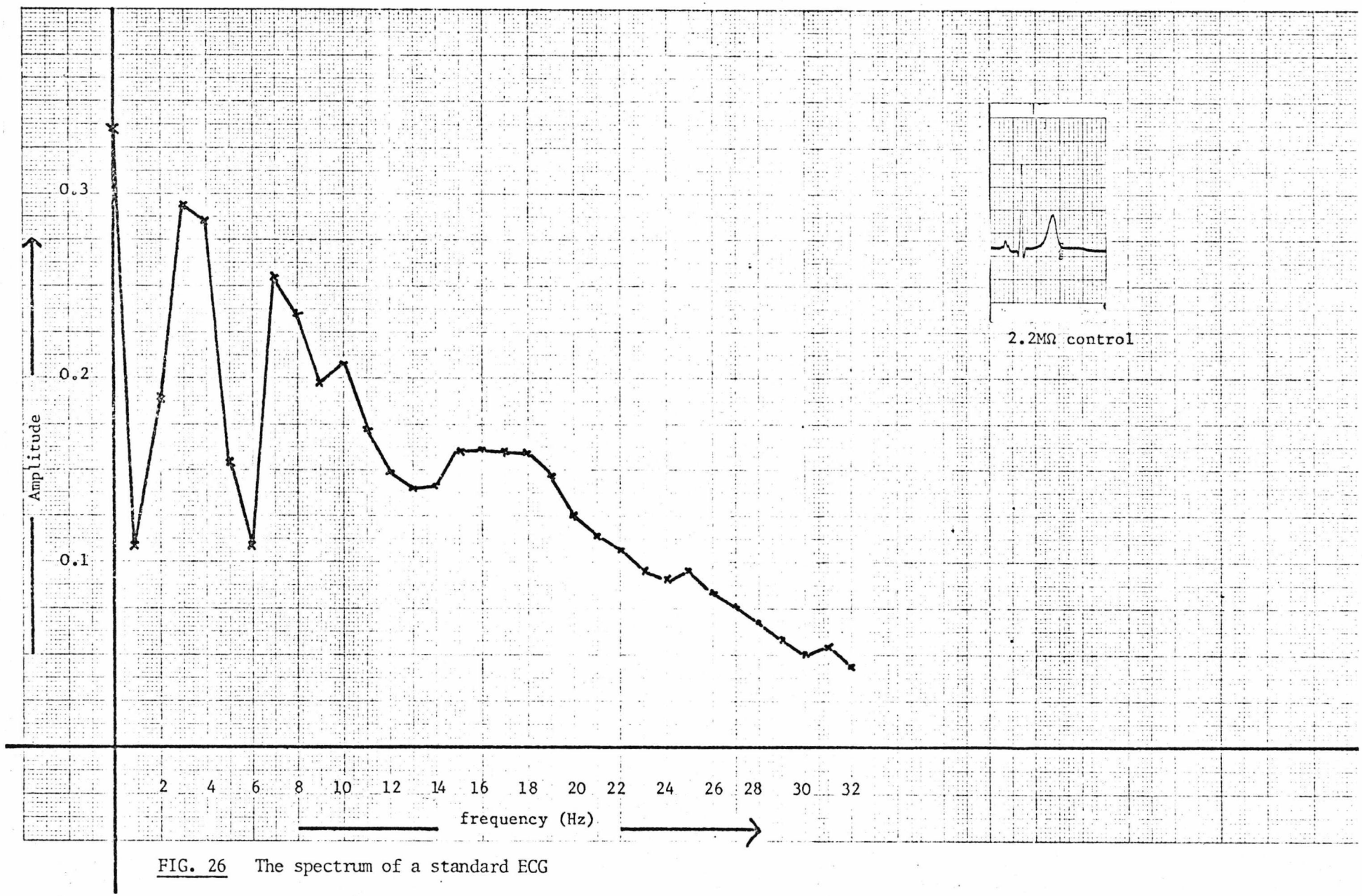


FIG. 26 The spectrum of a standard ECG

FIG. 27 The spectrum of compensated and uncompensated ECGs, silver/silver chloride electrodes

FIG. 28 The spectrum of compensated and uncompensated ECGs - large metal plate electrodes

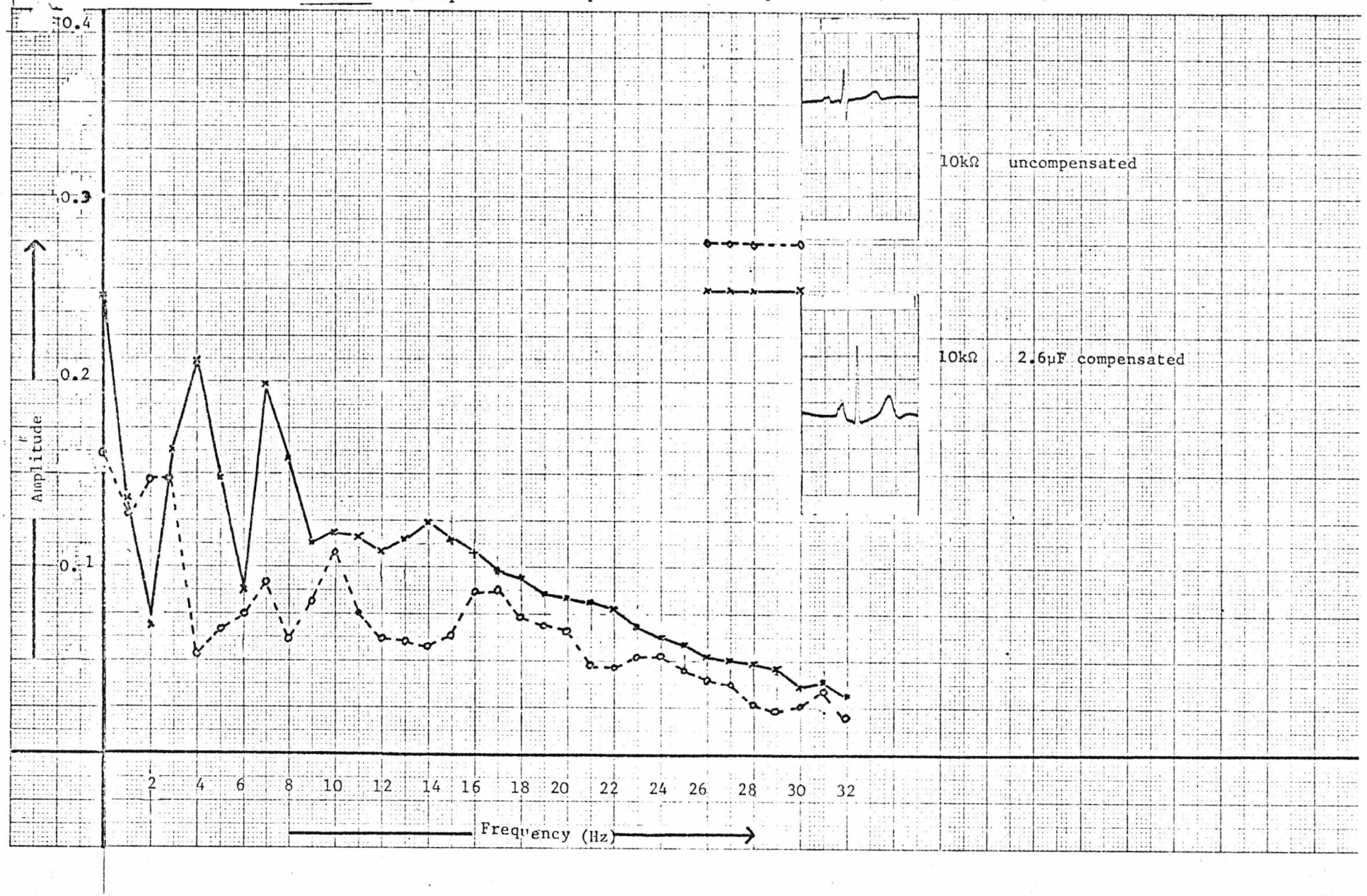
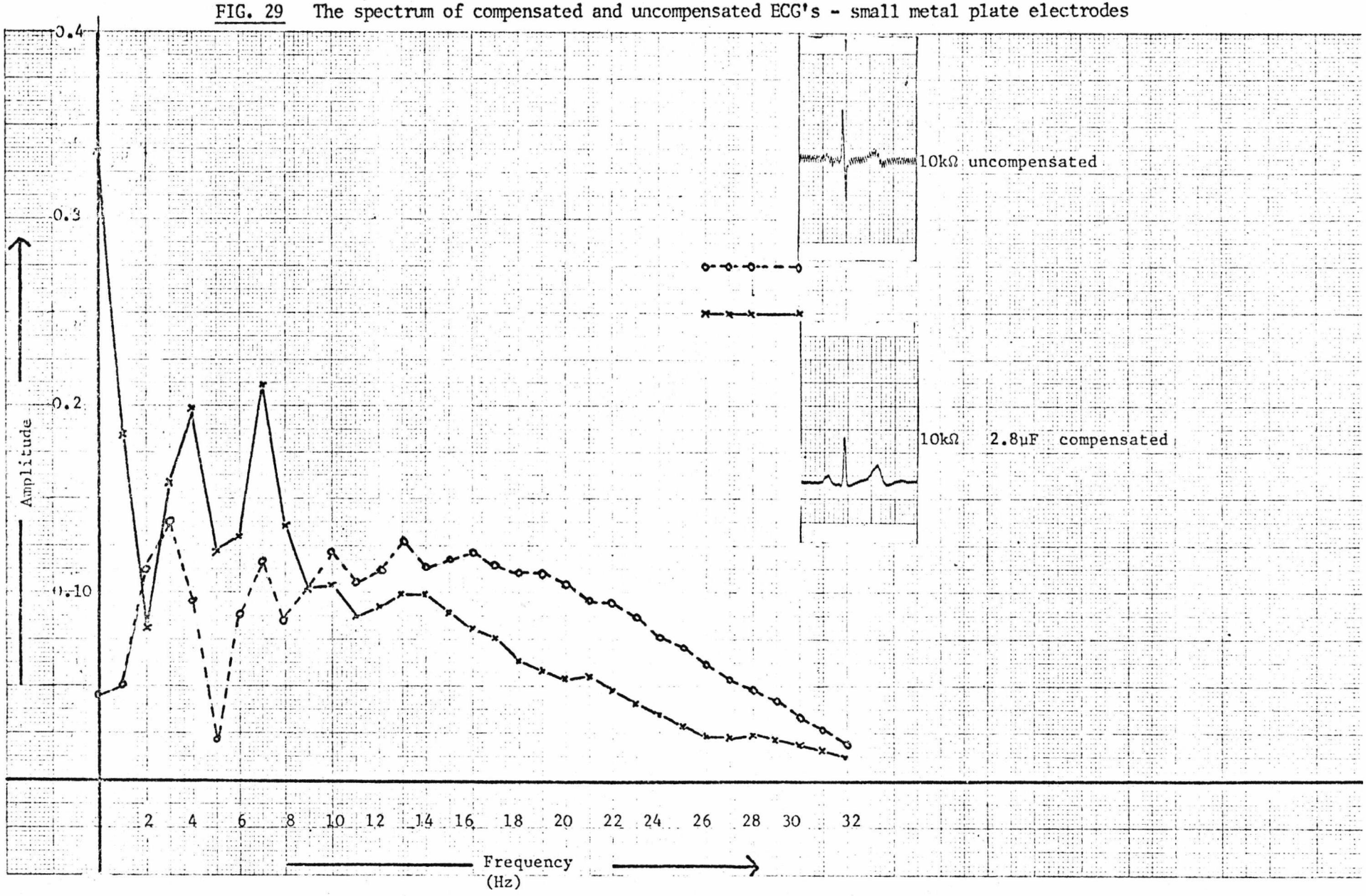


FIG. 29 The spectrum of compensated and uncompensated ECG's - small metal plate electrodes



the amplifier. This was connected to a box of resistors so that any one of them could be switched across the inputs.

At first, the circuit was connected as in *Fig. (30a)* for measuring the common mode response. The generator output was set at 20 volts p-p and with no R_{in} across the input (∞), R_T was adjusted to match the equivalent electrodes as closely as possible, and to give the minimum possible voltage displayed on the oscilloscope. Then different values of R_{in} were switched across the input, and the common mode voltage appearing for each was noted. Then the generator was moved to its position in *Fig. (30b)* for the differential signals to be applied. For each value of R_{in}, the generator output was adjusted so that the value of the differential signal seen on the oscilloscope was the same as the common mode signal for that value of R_{in}, and then the output was measured, and the CMRR calculated from the readings (for any R_{in}, $\text{CMRR} = \frac{20 \text{ volts}}{\text{differential input voltage}}$). The experiment was first performed at 10Hz and then repeated for 50Hz and 100Hz. The results are plotted in *Fig. (31)* as lines a, b, and c, and it can be seen that for each frequency, the CMRR is independent of the value of R_{in}, as predicted from the theory in section 6.6(ii).

Then capacitors were added in parallel to the two resistors representing the electrode to make the model more exact (note 1, *Fig. (30)*). The frequency was set at 50Hz, and the experiment repeated and the CMRR was found for various values of R_{in} (line d, *Fig. (31)*). Then all was repeated again with a compensating capacitor across R_{in} (so that $\tau_{in} = \tau_{electrode}$), (note 2, *Fig. (30)*) and the results plotted as line e in *Fig. (31)*. Once again these last two results show that the CMRR is independent of R_{in}.

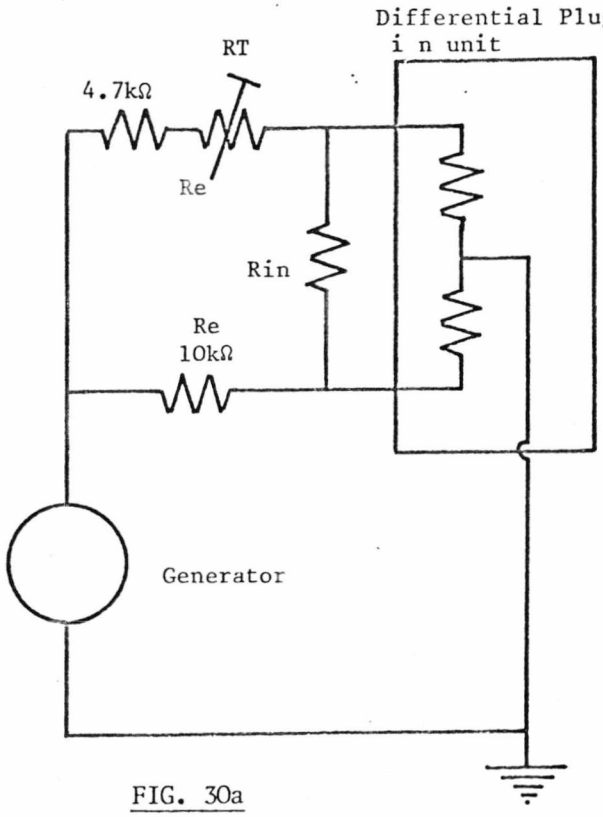


FIG. 30a

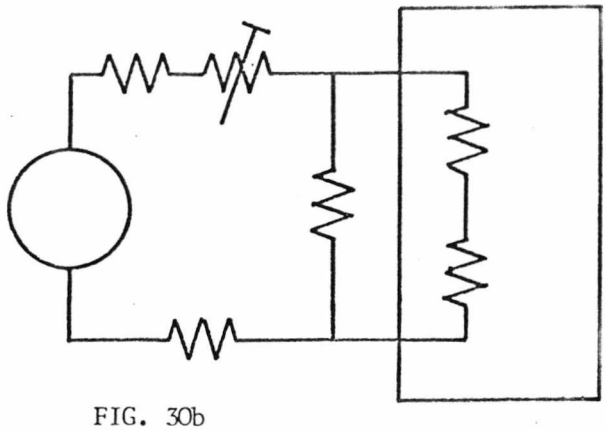
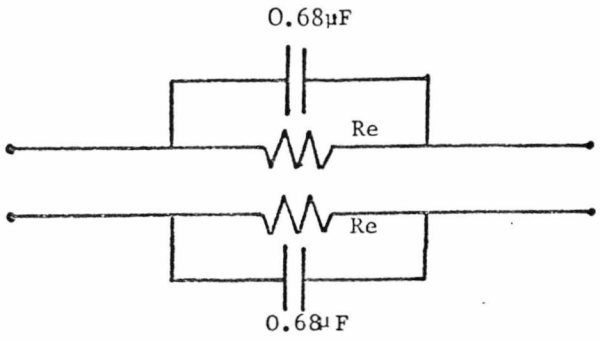


FIG. 30b

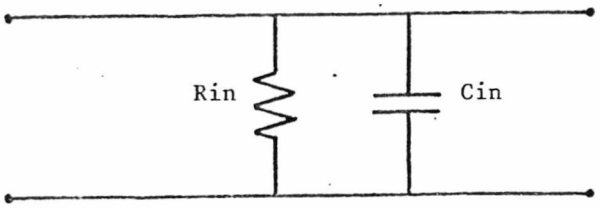
FIGS. 30 Show the model circuits for testing the common mode rejection rate

Note 1



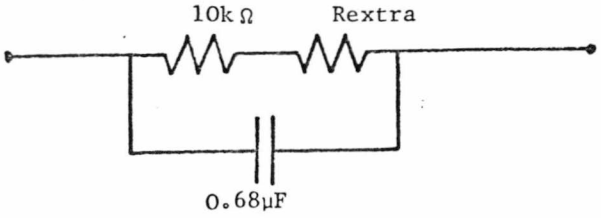
Capacitors are added to the electrode resistances for a more exact model

Note 2



A compensating capacitance is connected across Rin

Note 3



An imbalance is created between the electrodes by the insertion of one extra resistance in one arm.

FIG. 31 The variation of CMRR with Rin and frequency

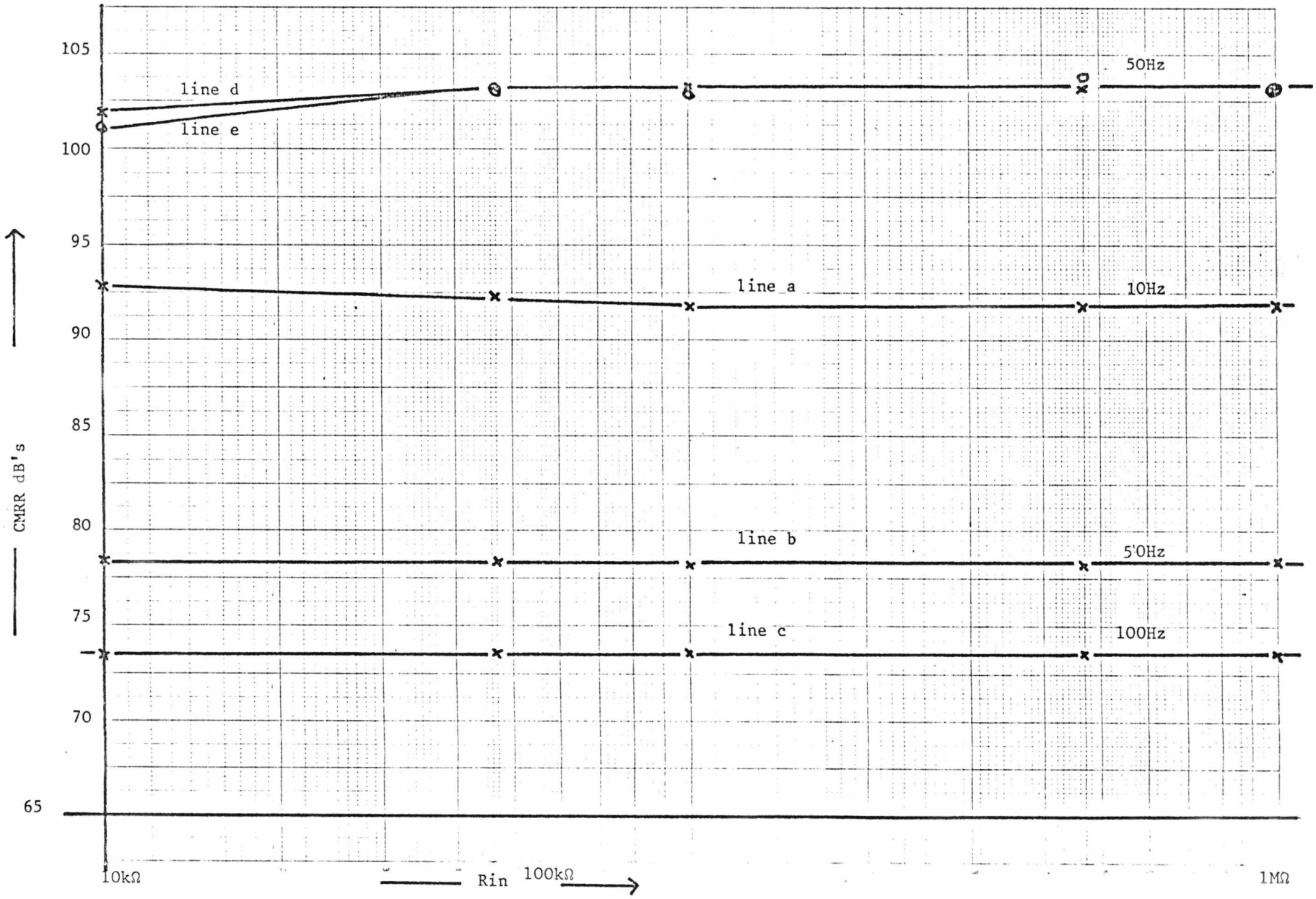
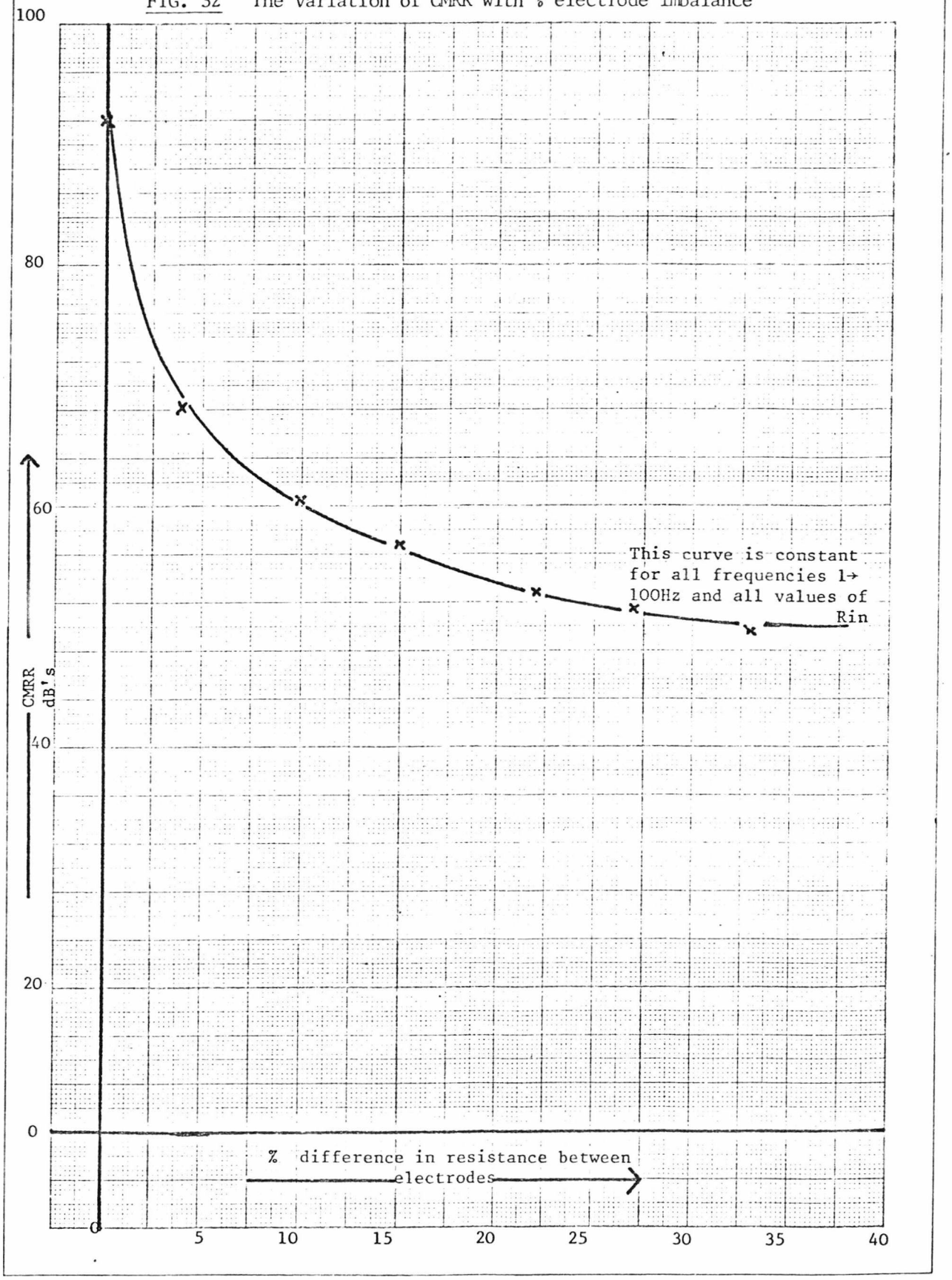


FIG. 32 The variation of CMRR with % electrode imbalance



There is a disparity in value of the CMRR at 50Hz, between the two cases of the electrode being represented by a resistance alone, and then by a resistance/capacitance network. This is believed to be due to the stray capacitances across the resistances - especially the trimmer R_T - being unequal and so setting an imbalance between them. When two large and equal (0·68μF) capacitors were placed in parallel, they dominated the strays and the two electrodes become nearer each other in characteristics and so gave a higher CMRR.

Another experiment was tried with the model (note 3, *Fig. (30)*). An extra resistance was placed in one of the arms of the equivalent electrodes to upset the balance, and then the CMRR was calculated for the readings obtained. This was repeated for various values of extra resistance and R_{in} and the results are found in *Fig. (32)*, and in fact the CMRR only varied with the % difference in value of the electrode impedances, and gave the same results for any value of R_{in} and frequency from 10 to 100Hz. Thus, the model experiments prove conclusively that the theory was correct, and that the CMRR only depends upon the electrode impedance difference and not upon the value of any R_{in} placed across the inputs.

The system was further tested on another more exact model. Since it is not good practice to connect up a human subject to a generator and drive them with 20 or more volts at 50Hz to generate a common mode potential - another model was used. A glass cylinder had gauze padding wrapped round it, and two small metal plate electrodes were prepared with electrode gel and strapped to the gauze, *Fig. (33)*. Then the gauze was dampened with saline solution. Two more electrodes were applied to the gauze, but lower down from the others and the axes of the two pairs were perpendicular. The bottom set of

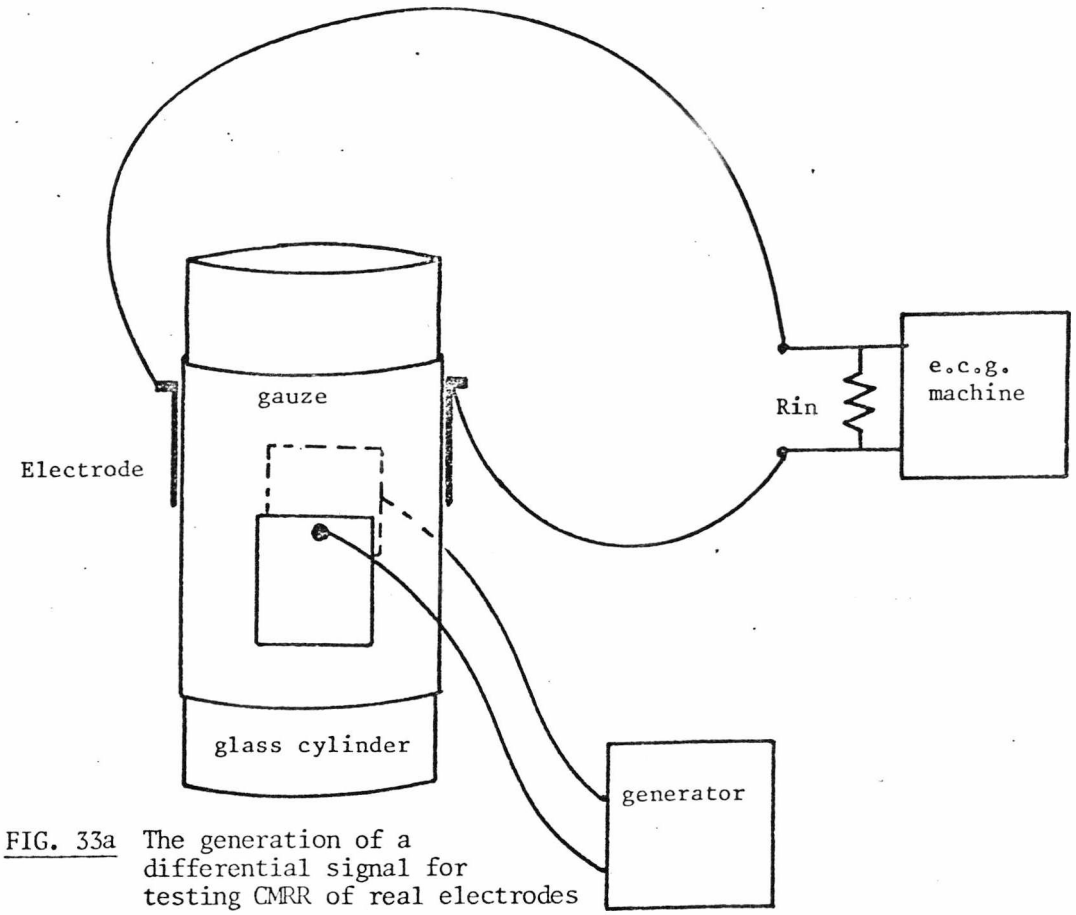


FIG. 33a The generation of a differential signal for testing CMRR of real electrodes

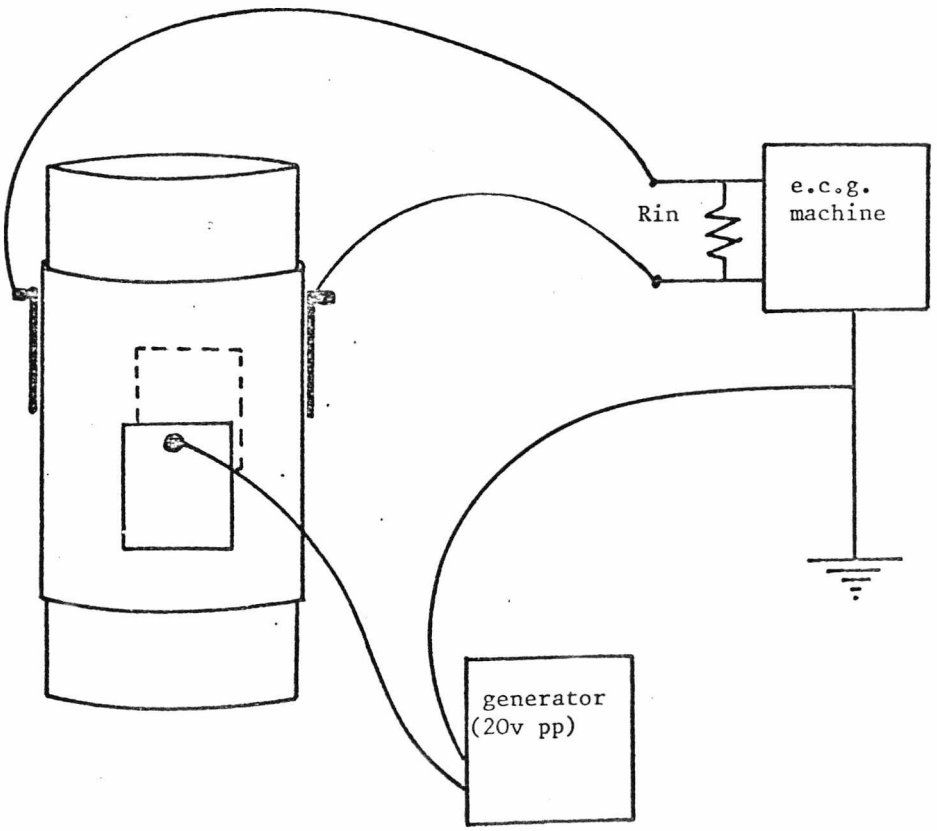


FIG. 33b The generation of a common mode signal to test CMRR of real electrodes

electrodes was used to provide a differential signal for the upper set when both were connected across the generator *(Fig. (33a))*, and a common mode signal when just one was driven *(Fig. (33b))*.

Then the experiment was repeated as previously, and it was found, once again, that the common mode rejection ratios were constant for any value of R_{in}. The values of the CMRR were 70dBs(10Hz), 68.6dBs(50Hz) and 70dBs(100Hz). Although a further drop in the size of the CMRR at 100Hz would be expected from the previous work, the fact that real electrodes alter their parameters with frequency means that the imbalance of the electrodes alters in a more complicated way than that of the simple fixed component model used previously, and at 100Hz they are better balanced than at 50Hz.

7.4 MOVEMENT ARTEFACT

7.4a Movement Artefact and Input Impedance

The previous sections have shown that it is possible in practice, as well as in theory, to satisfactorily record an ECG with an amplifier having a low input resistance across its terminals. In this section, the purpose for which the previous work was investigated i.e., the effect of R_{in} upon the offset potential and movement artefact is examined.

Two small, metal plate electrodes were strapped to a subject, and these were connected to an ECG amplifier and across the input of the latter was a variable resistor box (as in *Fig. (7)*). The two electrodes were placed on one arm of the subject, so that no ECG would be detected only the offset potential between them, and this was measured for

various values of input resistance. Then a small signal was injected into the system and the amplitude of this across the various values of R_{in} was measured. This was first tried at 10Hz and then repeated at 100Hz. The change in amplitude of the ECG itself was not made a parameter, as the various components of it each change differently with a change of R_{in}. The results were plotted on a graph, and the experiment was repeated for three other electrode types - large metal plates, silver/silver chloride and stainless steel disc electrodes. The results can be seen in *Figs. (34)* and *(35)*. In all of them the changes in amplitude of the injected signals are exactly as expected from the previous results with the 10Hz signal being reduced more than the 100Hz. Also in all of them the offset potential is reduced as R_{in} is lowered - and in the silver/silver chloride *(Fig. (34a))* and stainless steel *(Fig. (34b))* results, it almost disappears completely.

It is important to determine whether or not this is just a case of attenuation of the offset by the combined effects of input resistance R_{in} and the internal resistance of the generator - or whether the offset is discharged by the load as well as attenuated. The equivalent circuit of *Fig. (36)* was used and circuit values discovered from each set of recorded values. The generator voltage was taken to be the offset measured when $R_{in} = \infty$ and the value of the internal resistance was found from this and the offset voltage when $R_{in} = 2.2M\Omega$. (It was assumed that at this high value of R_{in} only attenuation would take place). Then using all of these the values of offset were calculated for each value of R_{in} and the results for each electrode were plotted on their respective graphs. It can be seen, that in all cases, the measured and predicted

WELL Graph Data Ref. 5561

Log 6 Cycles x mm, ½ and 1 cm

FIG. 34a Silver/silver Chloride Electrodes

FIG. 34b Stainless steel disc electrodes

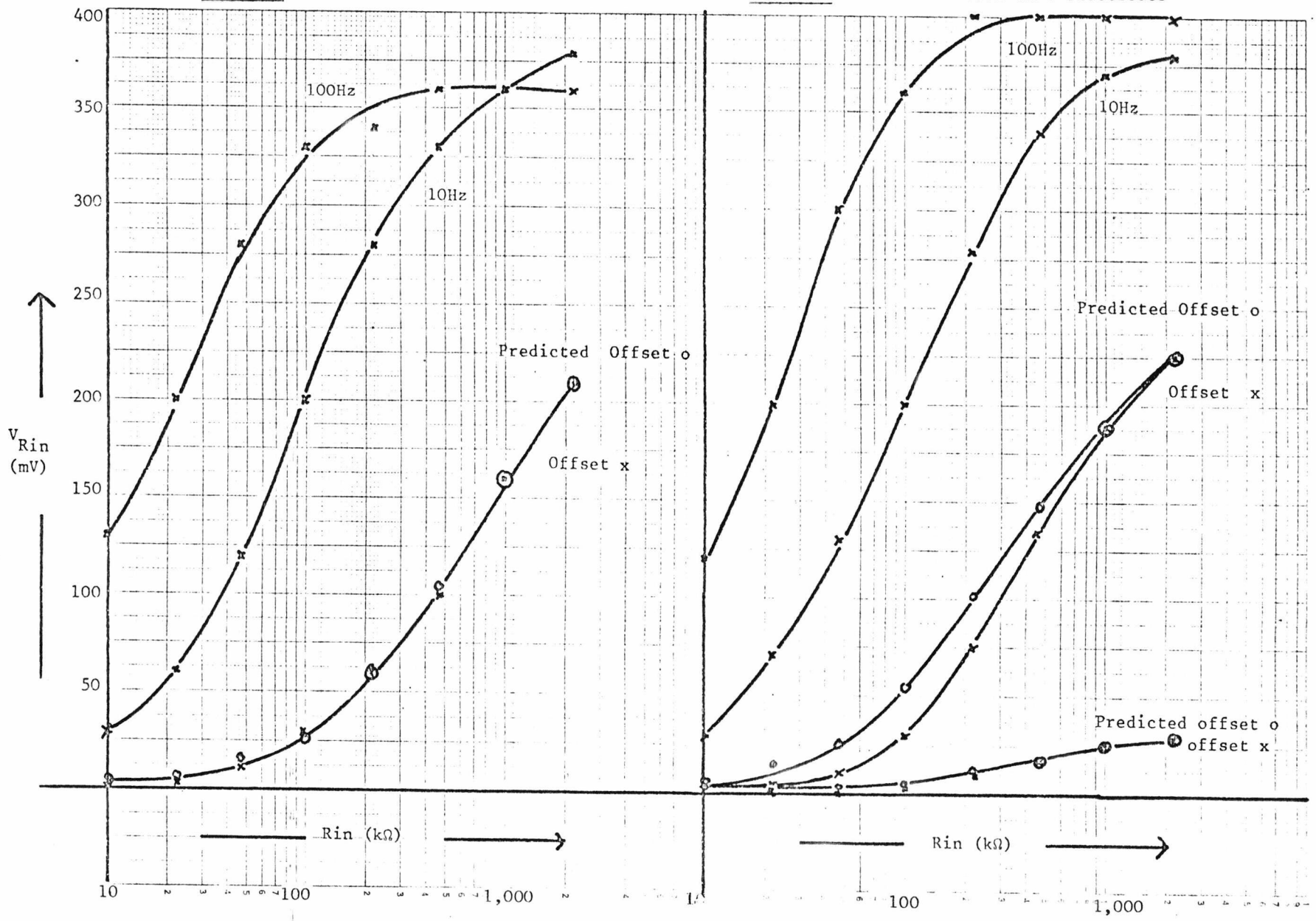


FIG. 35a Small metal plate electrodes

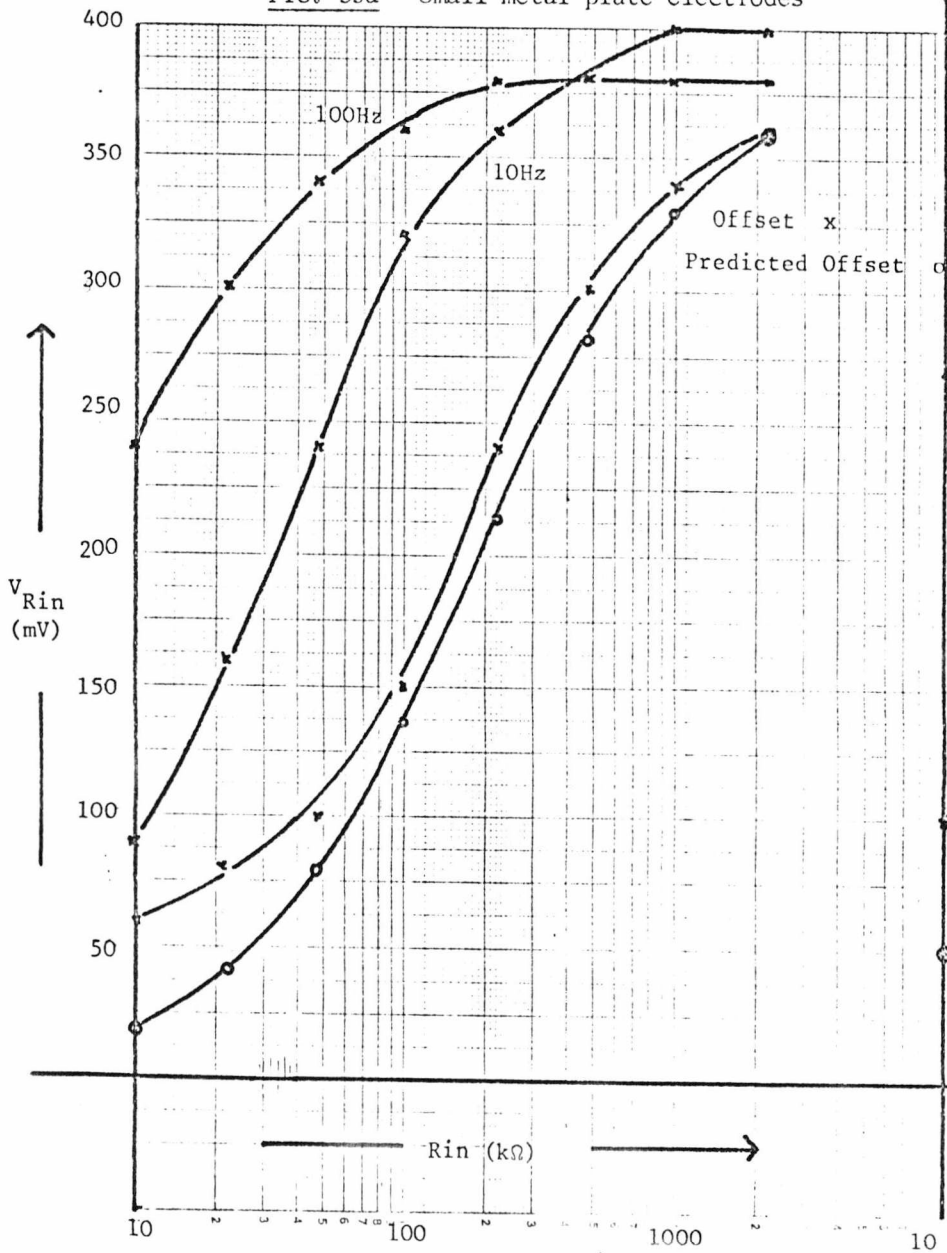
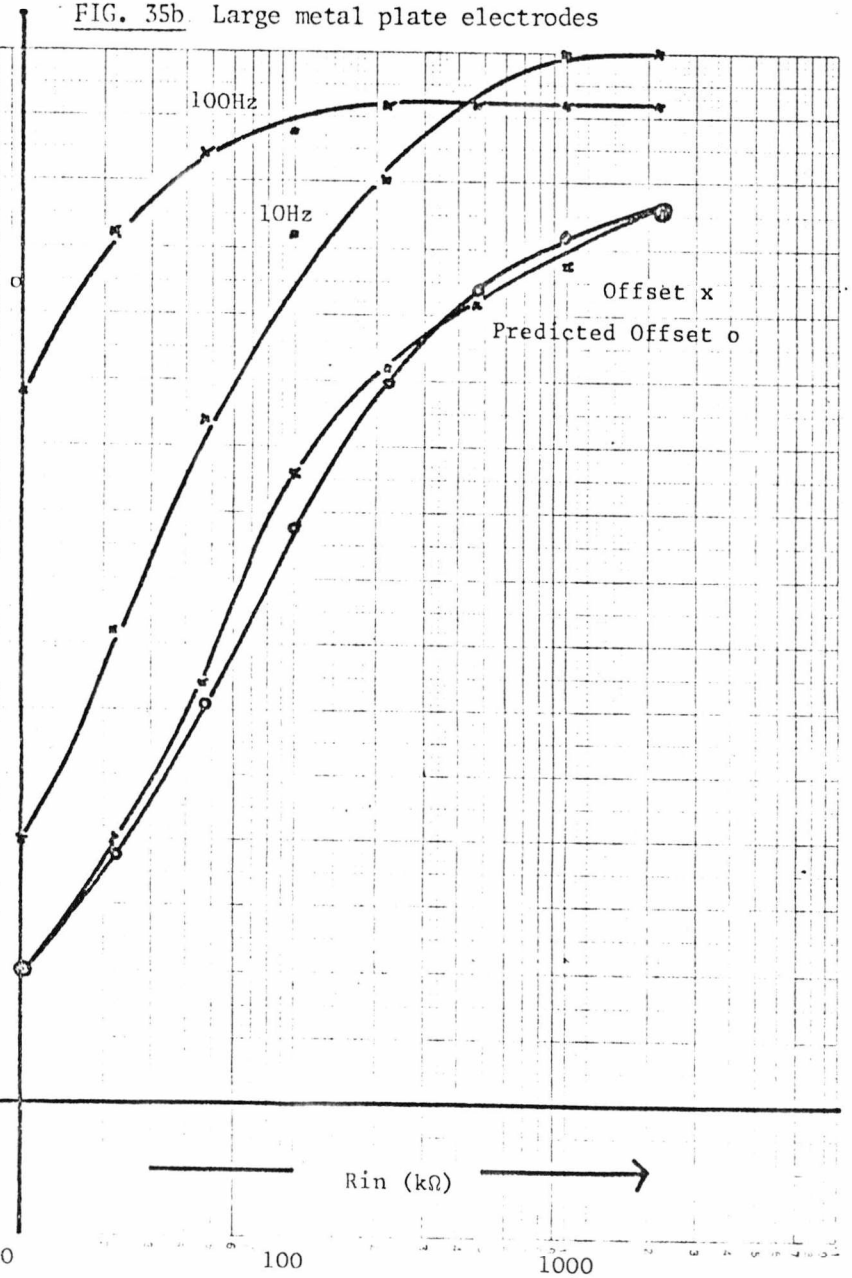
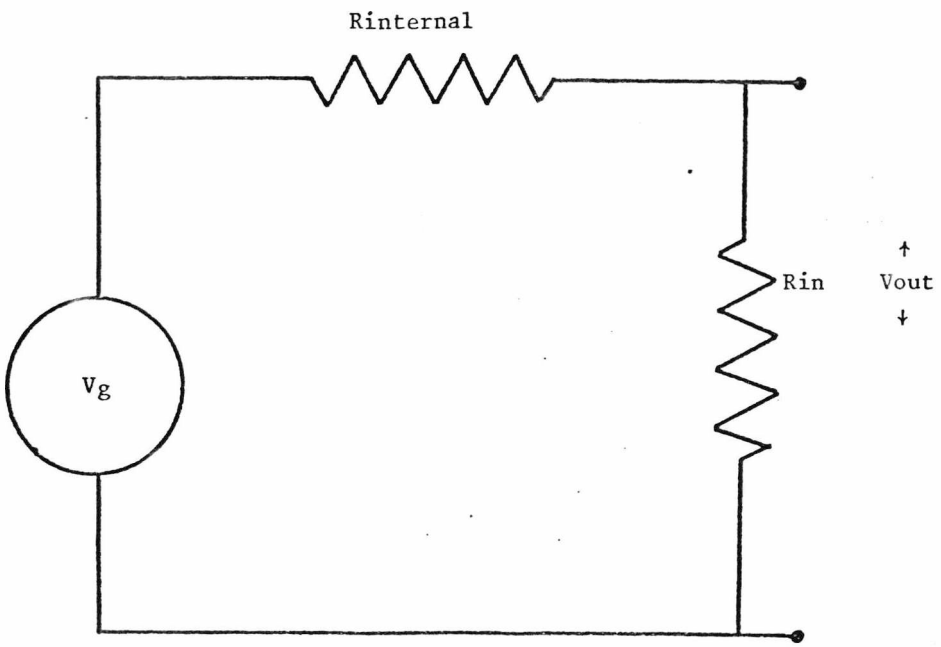


FIG. 35b Large metal plate electrodes





$$Vout = \frac{VgxRin}{Rin+Rinternal} \quad \text{for each Rin}$$

Vg = Offset measured when Rin = ∞

$$Rinternal = \frac{2.2M\Omega(Vg-Vout^{*})}{Vout^{*}}$$

$Vout^{*}$ = Vout at 2·2MΩ.

FIG. 36 The equivalent circuit used in the calculation of the attenuation of the offset potential

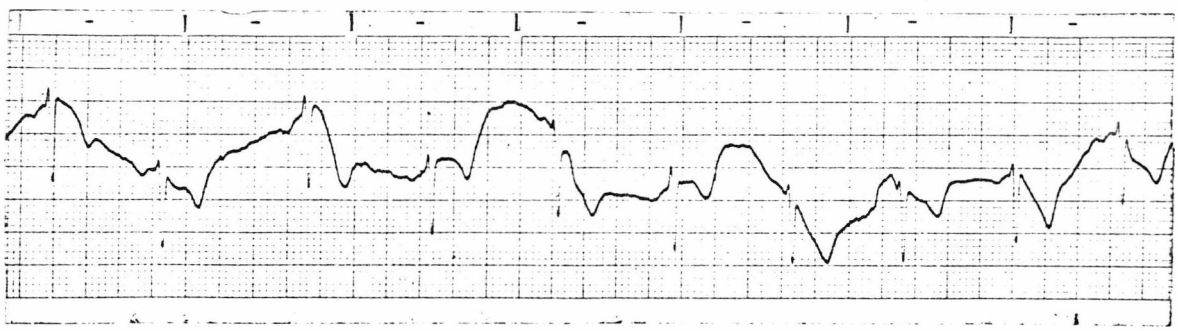
offset voltages were quite close in value so that it would appear that only attenuation of the offset is taking place. If we examine, however, the signal to offset ratio then from *Fig. (34a)* for 100Hz, the ratio is 1.7 for R_{in} = 2.2MΩ and 22 for R_{in} = 47KΩ. For 10Hz in *Fig. (34a)* it changes from 1.8 at R_{in} = 2.2MΩ to 9.6 at R_{in} = 47KΩ. Thus, although only attenuation is taking place some improvement is still obtained, as similar results to these can be obtained from any of *Figs. (34)* and *(35)*

Dynamic studies for the different types of electrodes are shown in *Figs. (37)* to *(41)*. Two electrodes of each type were stuck or strapped to the chest, and then the subject pedalled the ECG department's exercise bicycle. The traces recorded for R_{in}s of 2.2MΩ, 47KΩ and 10KΩ are shown in the figures. The first three show that for metal plate electrodes and Walsh suction cup electrodes, the low input impedance does not reduce the movement artefact produced by the exercising subject. In *Fig. (39)*, it appeared that some improvement was present when R_{in} = 10KΩ. When the gain was adjusted, however, so that the amplitude of the QRS was the same as that of the recording for R_{in} = 47KΩ, then the artefact (the 4th trace of *Fig. (39)*) can be seen to be as bad as ever.

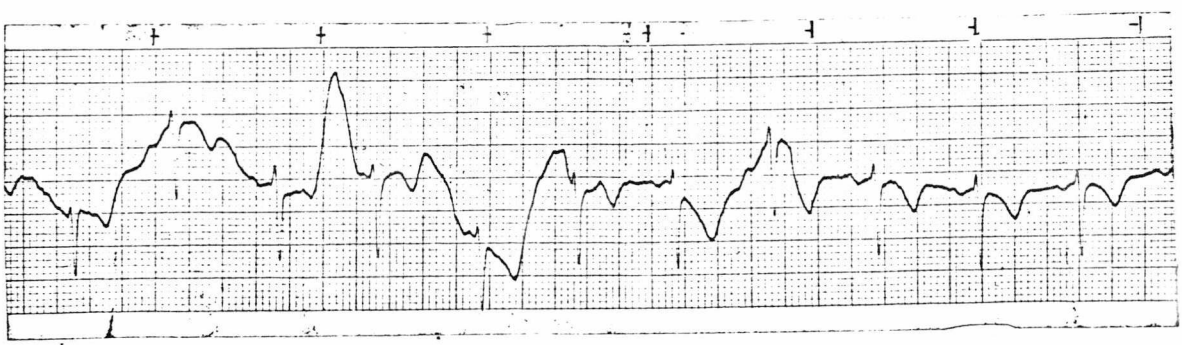
In *Fig. (41)*, using stainless steel electrodes, the drift, apparent with the large input resistance, disappears with the two low values, and so the proposal works for these electrodes if not for the others.

The low resistance was found very useful, however, when discharging electrodes that had been well overpolarised. The model of *Fig. (33)* was used and two small metal plate electrodes were strapped to the gauze. Then a 10 volt power source was connected across them, for varying times (1, 5, 10 and 20

2.2MΩ

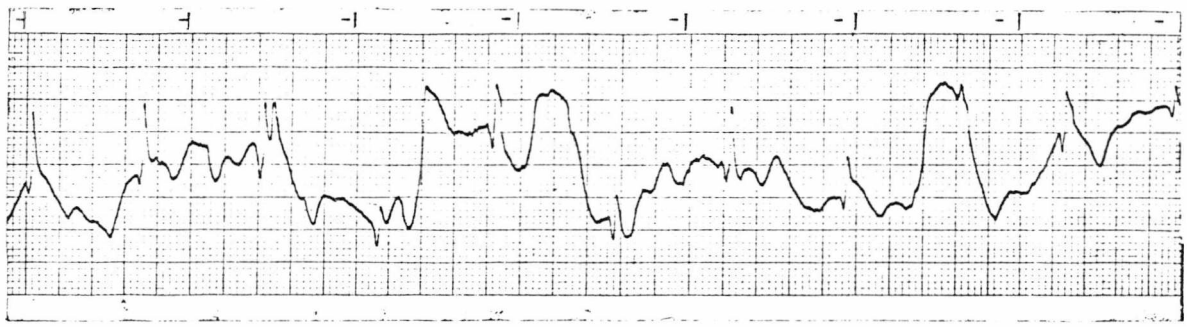


47kΩ

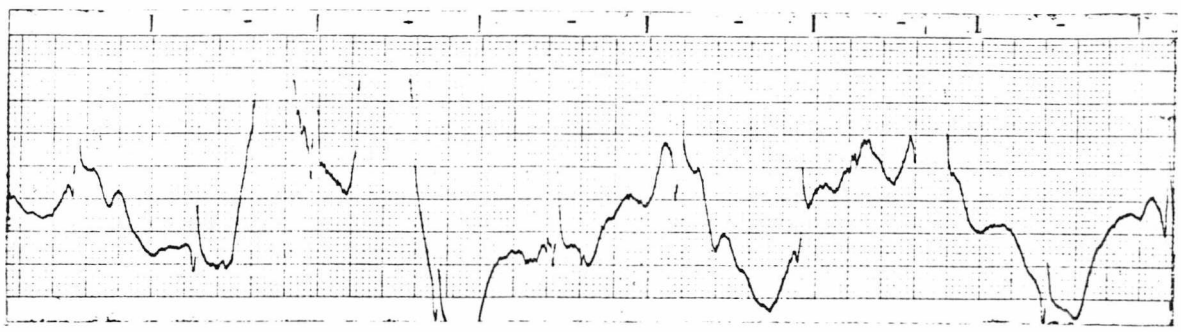


10kΩ

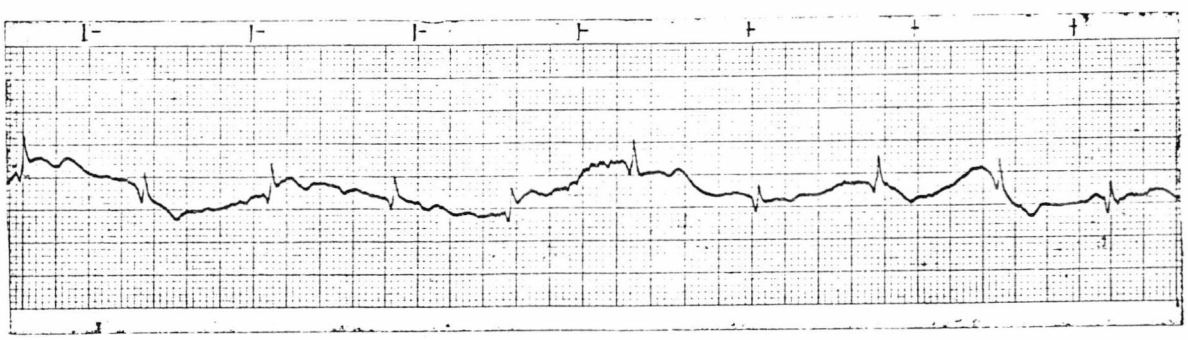
FIG. 37 The ECG's recorded, for different values of Rin, from the subject on the exercise bicycle with small metal plate electrodes



2.2MΩ



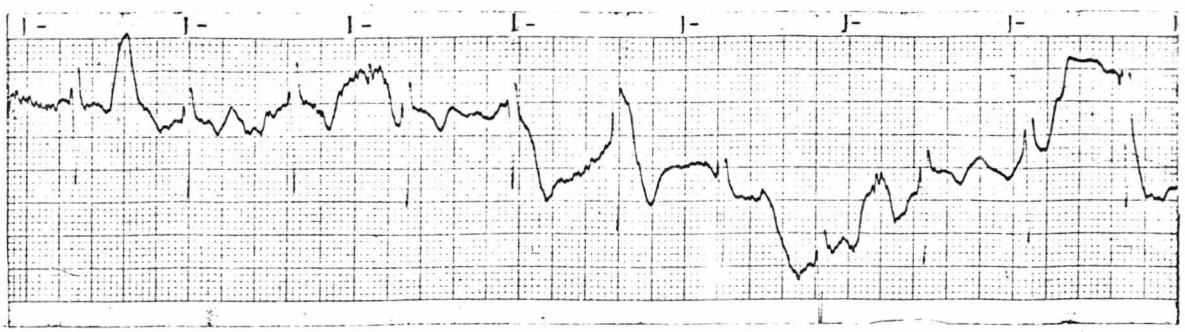
47kΩ



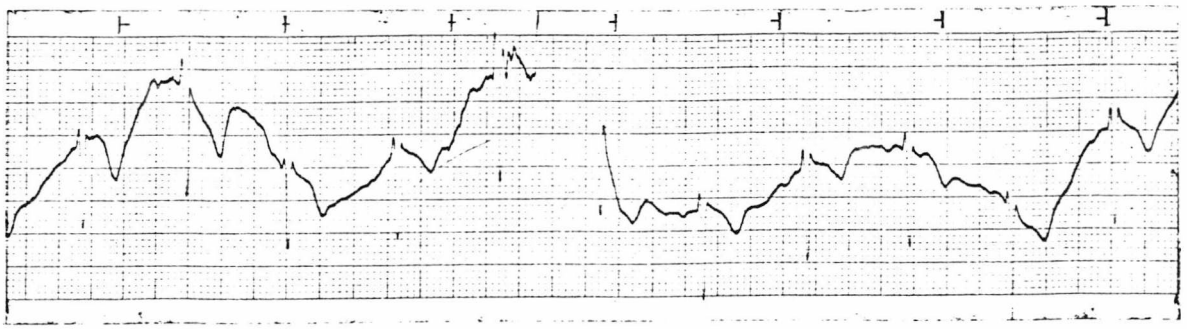
10kΩ

FIG. 38 The ECG's recorded for different values of Rin, from the subject on the exercise bicycle with Walsh suction cup electrodes

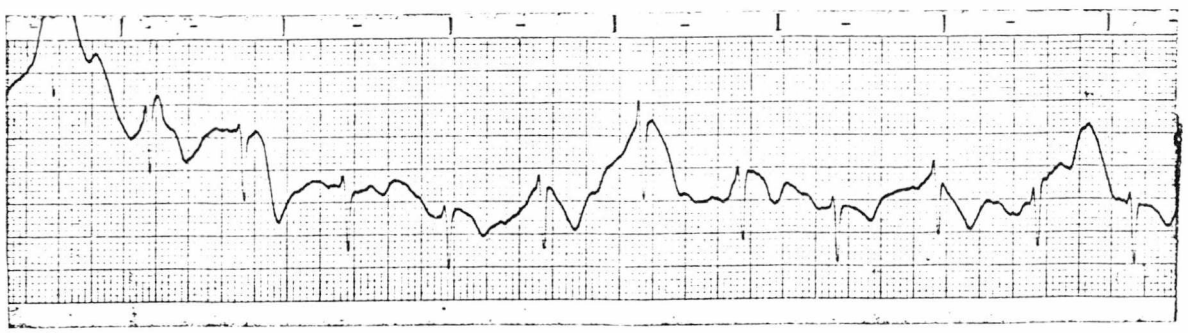
FIG. 39 The ECG's recorded, with different values of Rin, from the subject on the exercise bicycle with large metal plate electrodes



2.2MΩ

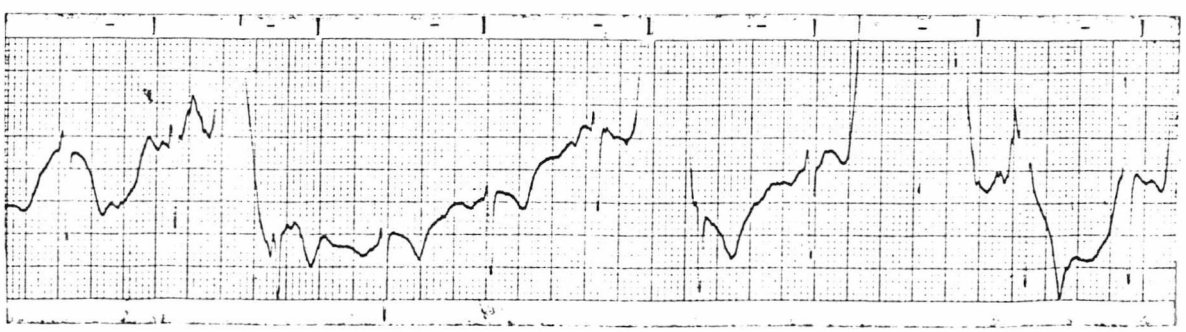


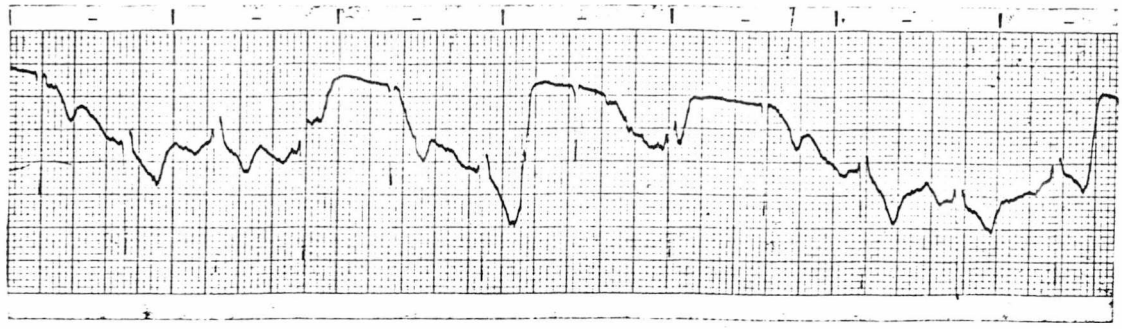
47kΩ



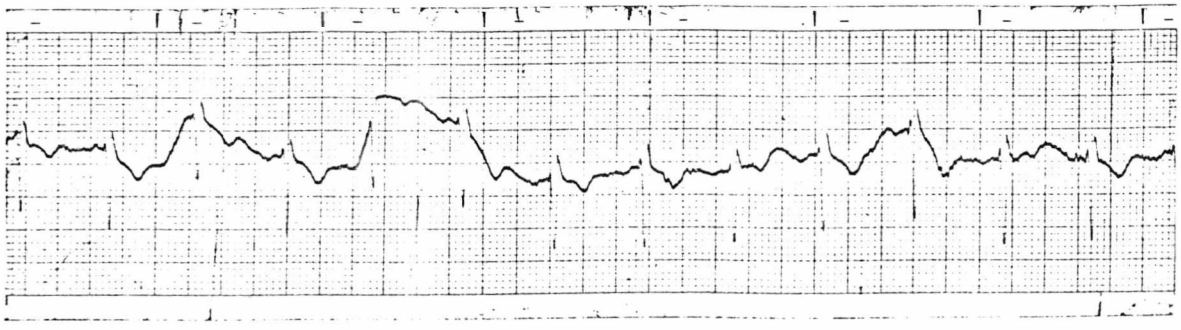
10kΩ

gain x 2 10kΩ

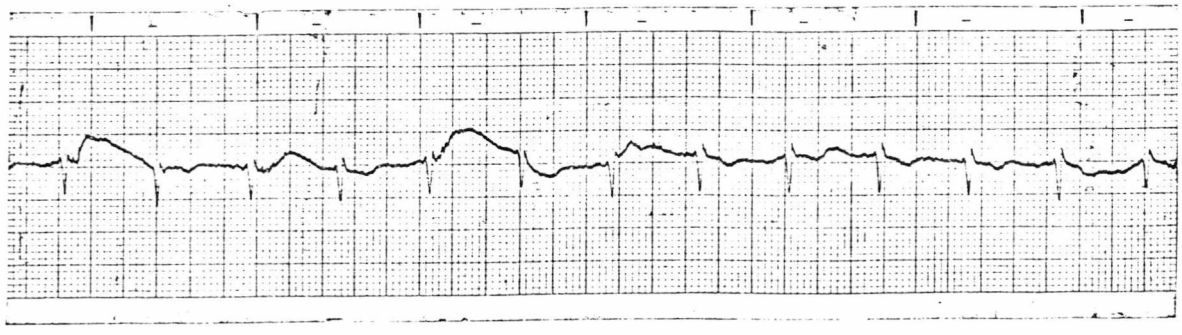




2.2MΩ

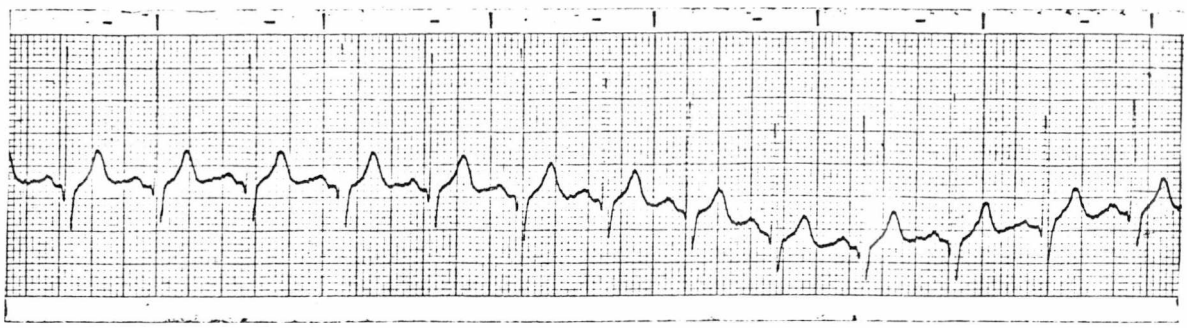


47kΩ

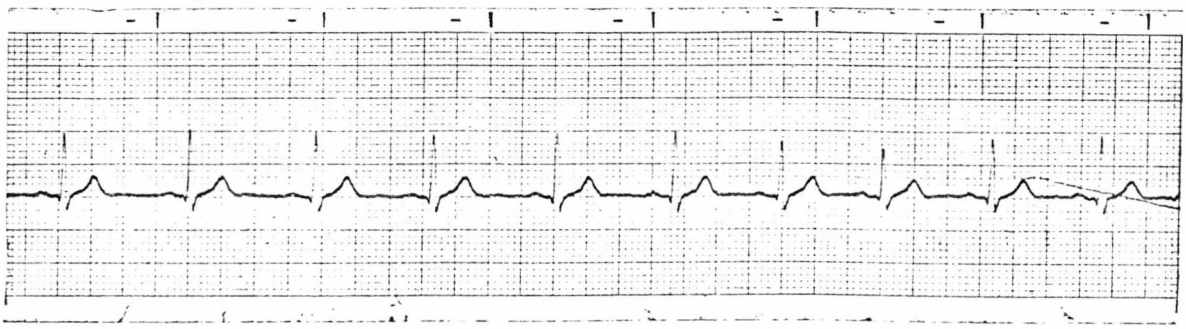


10kΩ

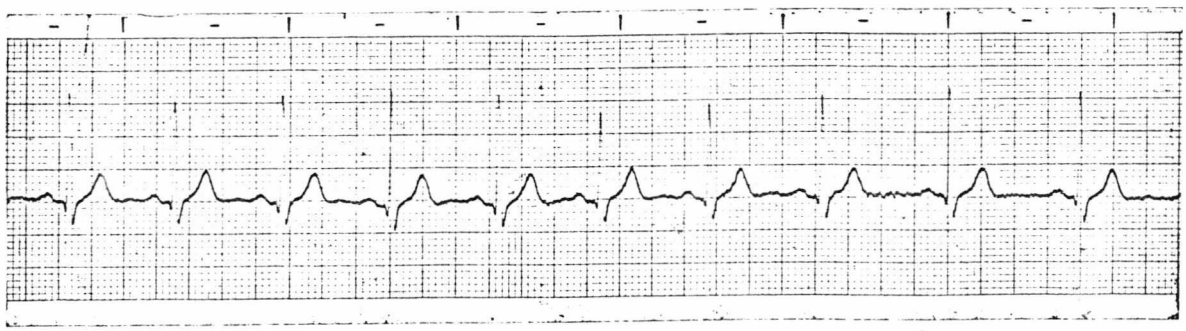
FIG. 40 The ECG's recorded, with different values of Rin, from the subject on the exercise bicycle with silver/silver chloride electrodes.



2.2MΩ



47kΩ



10kΩ

FIG. 41 The ECG's recorded, with different values of Rin, from the subject on the exercise bicycle with stainless steel electrodes.

seconds) and when disconnected the decay curve of the over-potential was traced on a storage oscilloscope. The experiment was performed with R_{in} = 2.2MΩ, 47KΩ and 10KΩ, and the results can be seen in *Fig. (42)*. It is quite obvious from the photographs that the low value of R_{in}, discharges the electrodes much quicker than with the high values. *Fig. (43)* shows the results when different values of resistances are used across the amplifier input when the overpotential charging time is constant of 5 seconds and these confirm the previous result.

This could be very useful during the defibrillation of a patient, since it means that the ECG trace after the defibrillating shock will settle down much quicker to a steady value with a low R_{in}, than otherwise. In addition, since gross changes in the ECG complexes are being sought i.e., the difference is between a sinus rhythm and fibrillation or no trace at all, then an average value of compensating capacitance can be safely used.

7.4b A Discussion of the Preceding Results

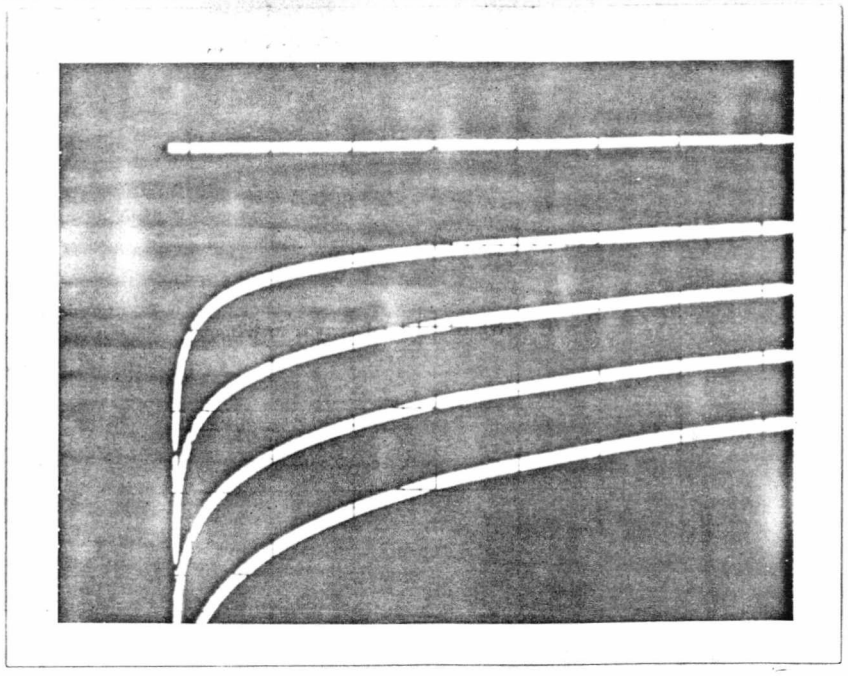
It would appear, then, that artefact is not reduced by the use of a low input impedance across surface electrodes - except where the latter are of stainless steel. The original model in Chapter 6 used for the calculation of the decay in offset potential (*Fig. (17)*, Chapter 6) used a single semi permeable membrane separating the two halves of the concentration cell. In practice the situation is as in *Fig. (6)*, Chapter 6 and since both electrodes rest on a skin surface there are two membranes. Also, the calculation did not take into account the

Vertical

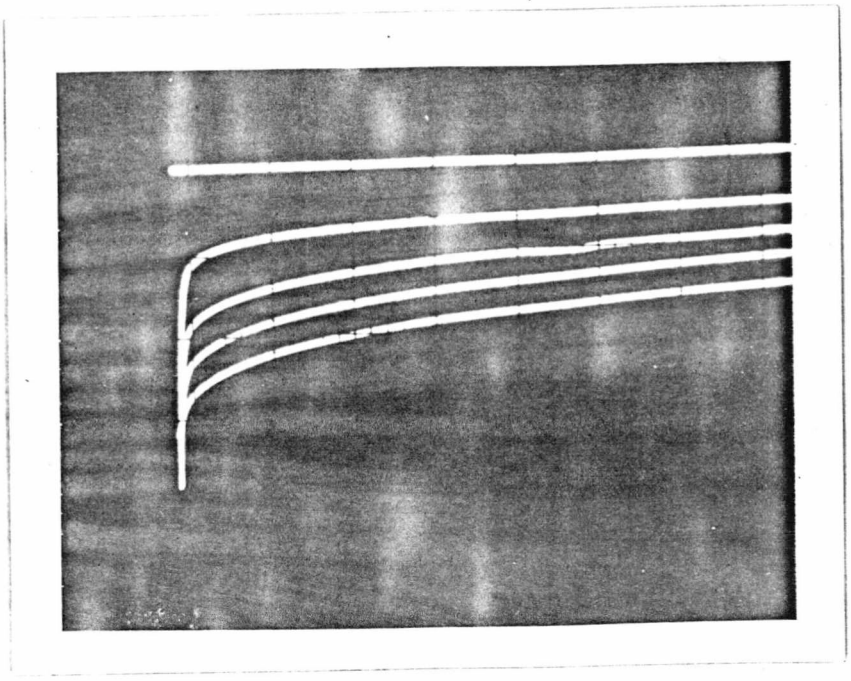
All at ↑
0.2volts/cm

Horizontal

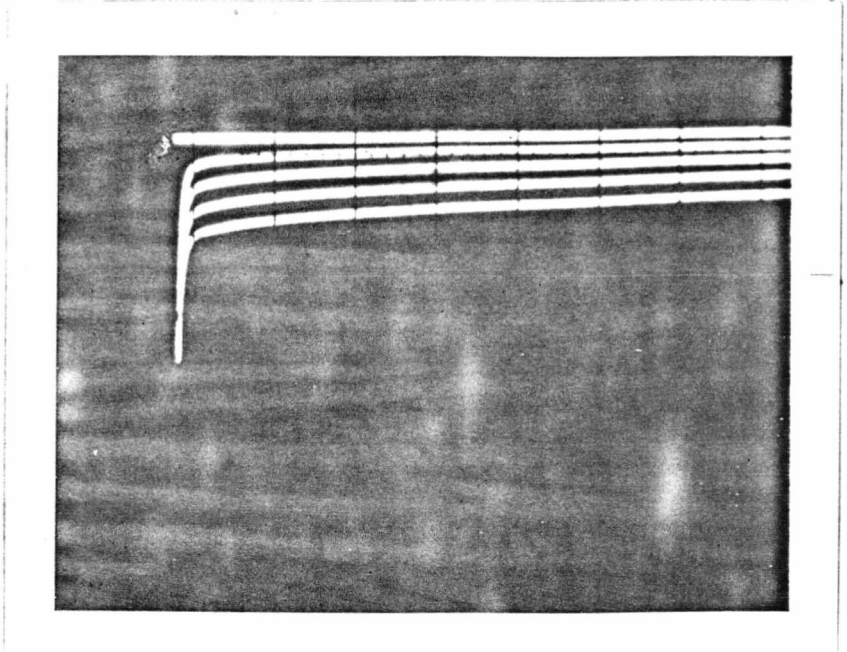
All at
0.5secs/cm →



2.2mΩ



4.7kΩ

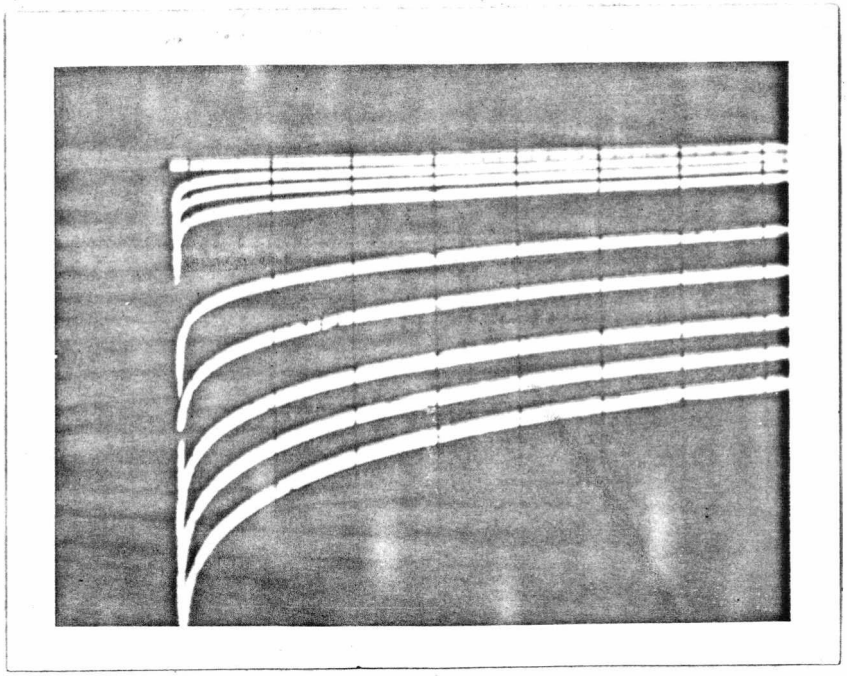


10kΩ

FIG.42 The discharge of overpolarised small metal plate electrodes. Each electrode was overpolarised for 1,5,10 and 20 seconds

Vertical

0.2volts/cm↑



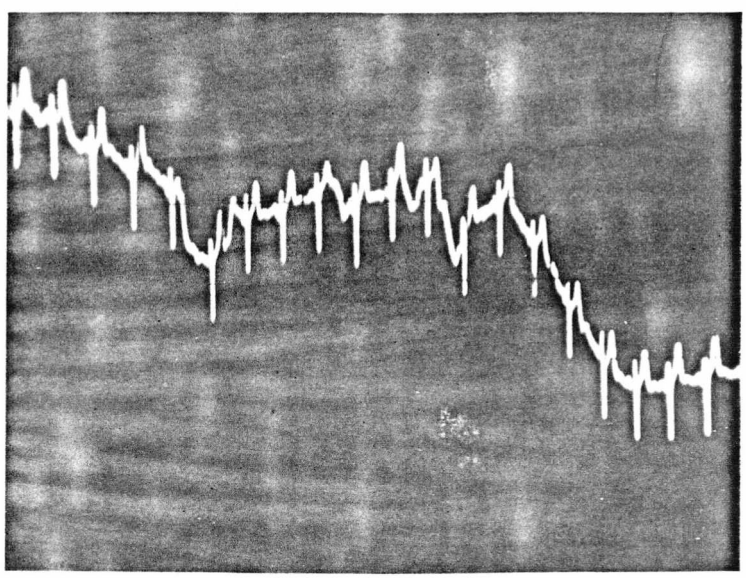
Horizontal

0.5secs/cm→

FIG. 43 The discharge of overpolarised (5 secs) small metal plate electrodes through various values of Rin

Vertical

Both at 50mV/cm↑



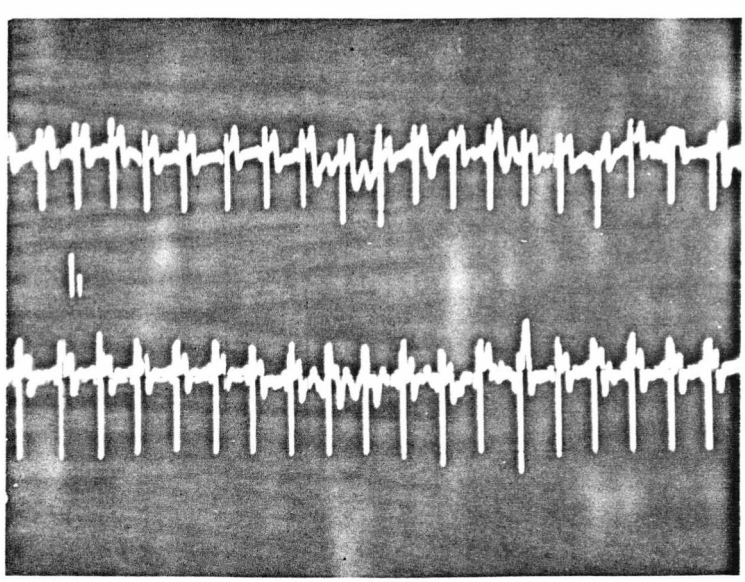
Horizontal

Both at 2secs/cm →

2.2MΩ

FIG. 44a

FIGS.44 Offset Potential behaviour of implanted electrodes



47kΩ

10kΩ (gainx2½)

FIG. 44b

transfer of charge across the membrane - it assumed that the current was determined solely by the external resistance. What probably happens when using real electrodes is that the skin acts as the principal rate determining factor in the chain of reactions and so the concentration cell ends up with a high internal resistance formed by the two semi-permeable membranes (skin under the electrodes). Thus, this prevents (relatively) large currents from flowing when low external resistances are used and prevents the offset potential from being removed - there is just the attenuation effect due to the potential divider of the internal and external resistances.

Other evidence to support this is provided by Strong (reference 4, Chapter 6) who describes the offset potential behaviour of several types of electrodes, and shows the offsets slowly decaying away over some considerable time. The initial conditions are set when the electrodes are applied and since there is a concentration difference between the two electrolytes immediately under the electrodes in the absence of an external path, they will slowly diffuse towards each other, to balance out the differences, through the skin and body fluids. Strong describes the decay times as several tens of minutes and body fluids are good conductors so one can infer that the diffusion of electrolytes through the skin is a slow process.

When the electrodes are disturbed, the electrode/electrolyte interfaces are disturbed as well. No matter how large the concentration differences that occur, the skin only allows a slow diffusion of electrolyte to take place, so the equalisation takes a long time and the artefact remains.

Stainless steel has a layer of chromic oxide on its surface, and this prevents it from coming into equilibrium with the

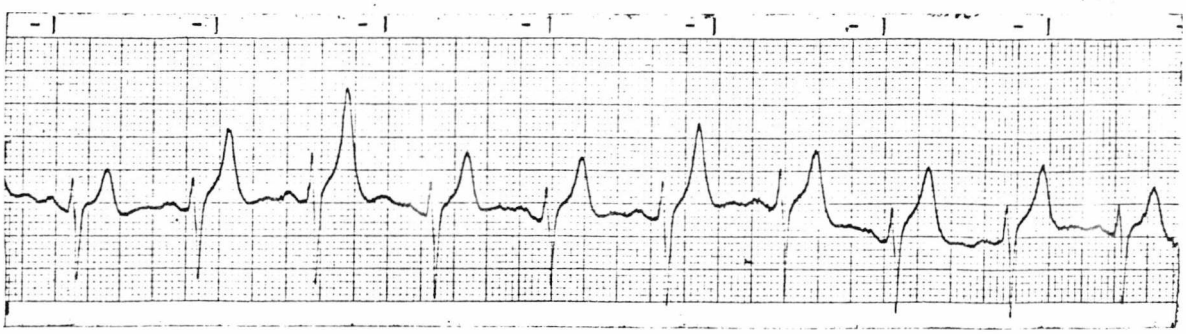
electrolyte - a reason why the offset potential of stainless steel is so unstable (Strong reference 4, Chapter 6). Few ions are exchanged at the electrode/electrolyte interface and so only very small currents are needed to equalise the differences between electrodes, and the external resistance can have a significant effect upon the offset - which it does *(Fig. 40)*.

Since the skin layers appear to limit the effectiveness of low external resistances it would seem a good idea, then, to try to remove them, so that they play no part in the reaction. An electrode that is passed under the skin, then, should prove a lot easier to stabilise with a low R_{in}.

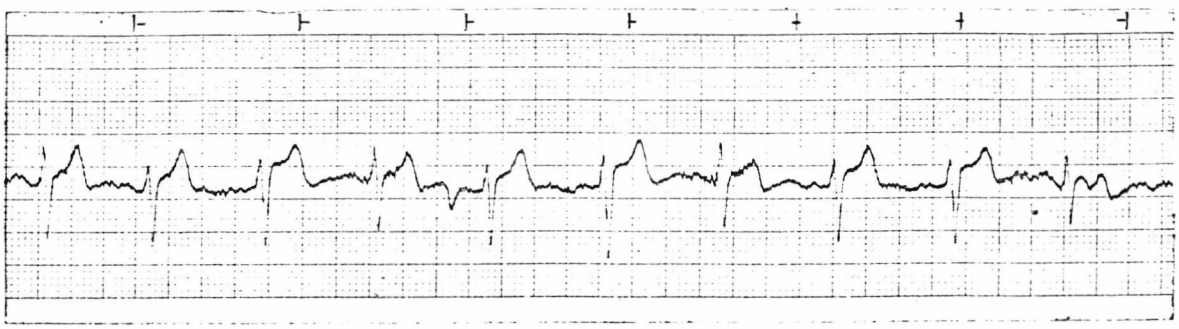
7.4c Under the Skin Electrodes

Three, sterile, stainless steel surture wires were used as electrodes in a triangular pattern on the chest, and were passed into, under and out again of the author's skin, and tied to form a loop. Thus, the electrodes were in direct contact with the body fluids. Then recordings of the ECG were taken, whilst tugging and shaking the electrode lead wires and the results can be seen in *Fig. (44)*. These were taken from an oscilloscope after passing through a d.c. coupled amplifier. *Fig. (44a)* shows the offset drift detectable when R_{in} = 2.2MΩ, but none is apparent in *Fig. (44b)* when R_{in} = 47KΩ(top) or 10KΩ(bottom). They were then used in conjunction with the exercise bicycle *(Fig. 45)*. Once again when R_{in} is of a low value no shift in the baseline is observed - even when the author was tensing his chest muscles (as well as vigorous movement) so as to produce the muscle noise seen in the bottom recording.

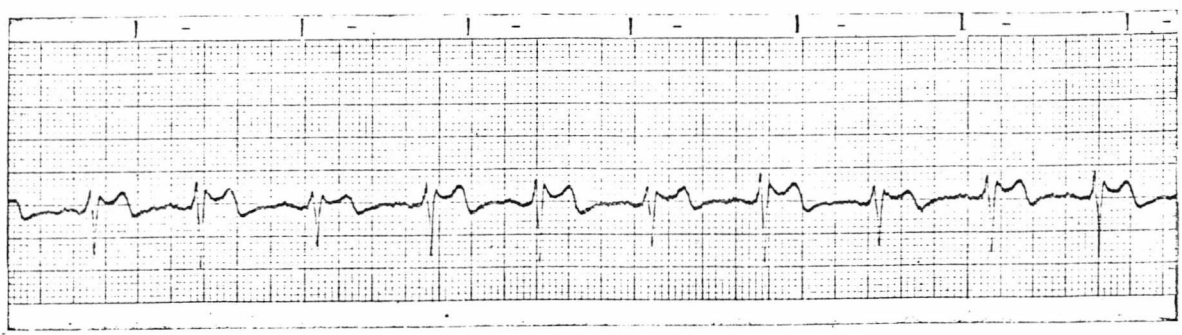
FIG. 45 The ECG's recorded, with different values of Rin, from the subject on the exercise bicycle with stainless steel under the skin electrodes.



2.2MΩ

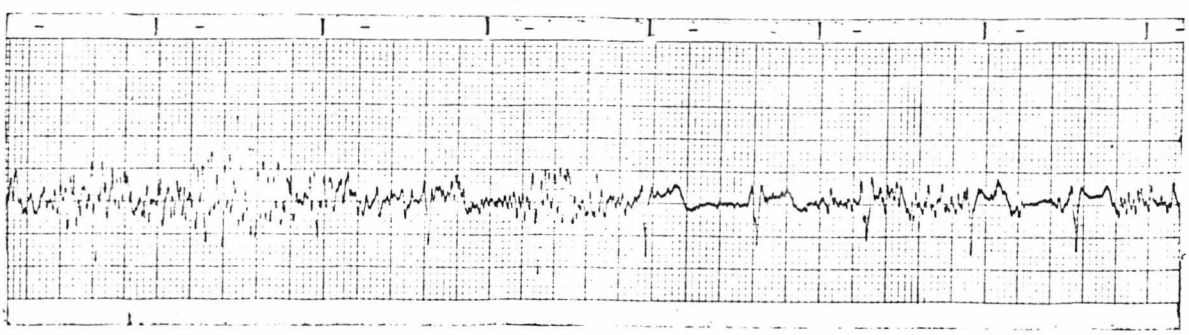


47kΩ



10kΩ

Excess Muscle Noise 10kΩ



It would appear then, that under the skin electrodes and a low input impedance both combine to produce ECG recordings that are free of significant movement artefact. Also surface stainless steel electrodes with low input impedances can be used to record ECGs from exercising patients, and the low external resistance should enable the electrode to be used during defibrillation since the baseline is quickly restored after significant overpotentials have been applied (ref 49, Chapter 6).

CHAPTER 8

CONCLUSIONS

8.1 A Summary of the Work

The underlying theme that has run throughout this thesis is that of clinical measurement - and in particular, the measurement of the ECG. At each point in the course of the patients' illness - right from the time of the attack, throughout the time in hospital until the patient (hopefully) leaves to return to a near normal life, then measurements have been made to enable the clinician to give the best possible care and attention. Although the ECG is by no means the only parameter that interests the medical staff, it is one that is easily obtainable at all times. It is impossible, for example, to perform an electrolyte analysis or partial pressure measurements of blood gases from the back of an ambulance. It is possible, however, with a relatively simple piece of equipment to detect, amplify and transmit the ECG, so that a diagnosis can be made by experienced staff, and treatment commenced well before the patient reaches hospital.

Whilst in the intensive or coronary care units, then the ECG is a signal that is continuously monitored as it can give so much information, so quickly of the cardiac state - whether or not sinus rhythm is present, the occurrence of ectopic beats, how often they appear and whereabouts in the cardiac cycle they occur. The presence of fibrillation can be detected immediately, without waiting for other symptoms to develop - it can even be anticipated if ectopic beats begin to develop at the same time as the T wave of the ECG.

The transmitter that was developed helps to ensure that

these signals reach the clinician in the best possible manner, and also increases the reliability of the monitoring by enabling the long, trailing wires, used at present, to be removed. It is also used in the assessment of the patient after treatment. This is another time where measurement of the ECG is important. It would, again, be difficult to measure parameters such as blood pressure and pulse rate from a patient pedalling on an exercise bicycle or performing simple exercises in the physiotherapy department. The ECG is easily obtained and the transmitter allows a trace as free from artefact as possible to be recorded.

At all times when the ECG is being measured transducers are being used - the electrodes - to detect the generated signals. A source of error during measurements is due to the electrode and its adjacent electrolyte being disturbed, which leads to movement artefact. A method of using low input impedances in the ECG amplifier has been tested and, especially, in the case of stainless steel, the artefact is considerably reduced using this new approach. Hitherto, it has been considered impossible to obtain clinically useful ECGs with low input impedances, due to the loss in low frequency content of the signal that occurs, and the resulting distortion. It has been shown that this can be overcome by suitable compensation, so enabling low impedances to be used, and a considerable reduction in motion artefact to be obtained.

8.2 Comments on the Preceeding Section

Neither the ambulance telemetry nor the ward telemetry work is original in concept - it is, however, original when taken into the context of the situation of the local hospital.

Should the Kent and Canterbury Hospital decide to proceed with its own cardiac ambulance, then the thesis has recommended the best approach with the conditions that applied at the time of writing. Each hospital seems to have approached the topic from a direction sensitive to their own situation, and this review of methods will be useful to those planning the Kent scheme.

There is still controversy about their usefulness, and some have found it difficult to operate with calls from the general public, as so few are genuine (infarction). This is regrettable, since it is in this situation that the mobile unit is so beneficial. Nottingham[1] has tried a system of only responding to emergency calls where the patient has been described as having collapsed, and so enables coronary care ambulances to be responsive to calls from the general public. They discovered that it was unnecessary to send out the special coronary ambulance to every call that came, since all sorts of other conditions were encountered (i.e. pregnancies and even five cases of drunk and incapable). Of the first 200 responses to collapse calls, 54 were found to have myocardial infarction and 18 of these had the symptoms for less than 30 minutes. Whether or not Canterbury will find it necessary to use the same method will remain to be seen as the unit develops. From a survey of 26 American[2] emergency ambulances schemes, the information in the following table was presented. It can be seen that 89% of the schemes carry paramedical personnel and 66% have ECG telemetry. So the method suggested for Canterbury in Chapter 3 seems to be comparable with the current American practice.

Voice communications (professional advice)	21
Portable ECG	22
Telemetry ECG	17
Portable defibrillator	23
Physicians	7
Nurses	13
Paramedical	23

TABLE (1)

A Summary of emergency ambulance services from 26 cities.

There are many ECG transmitters that are available from commercial concerns, but none were found that were completely suited to the situation, thus a purpose built system was designed. One transmitter that was available at a reasonable cost (approximately £300) turned out to have a very short battery life, whilst others that were suitable in this respect seemed to cost £1,000 or more per set. One common factor with all the units available was that they were only single channel, and having four beds simultaneously covered would mean four transmitters and four receivers - in one instance this would be a cost of over £4,000 (+ VAT).

The equipment was designed to overcome this unsatisfactory state of affairs. Tunnel diodes were used in the transmitters and an original method of biasing them was developed to overcome the previous problem of power wastage in the normal

resistive bias method. When the small output amplifier has been added to the circuit, and the constant current feedback used in the sub-carrier oscillator *(Fig. (1))*, then the transmitter will be ideally suited to its purpose. Another advantage is that the four transmitters need only one receiver and that is an easily obtainable Quad type. Thus the system could be produced at a reasonable cost by any hospital with an E.B.M.E. department.

The under-the-skin electrodes are an ideal complement to the transmitter. With a suitable network across the latter's input then the ECGs received should be free of all artefact - even when the patient is being moved, since there are no trailing wires to create noise or displace the electrodes. The normal stick-on electrodes can suffer several disadvantages. The first is that sometimes two or even three electrodes need to be used on some coronary patients in one afternoon, due to the large amounts of perspiration overcoming the adhesive compounds used. Those disposable types that use micropore tape to attach the electrode suffer less from this, as they allow some of the perspiration to evaporate[3]. The conditions under the adhesive are also advantageous to bacterial growth[4]. The secretions through the skin, and the normal perspiration can create an ideal environment for bacteria - of which there are many on the skin surface. Normally the constant shedding of the outer skin naturally removes these products, but when areas of skin are enclosed by an electrode this may lead to problems. The under-the-skin electrode is not prone to this problem, although care must be taken with it, since it pierces the skin surface, and long term studies ought to be completed to see whether any adverse reactions occur. The method should be all right when used in

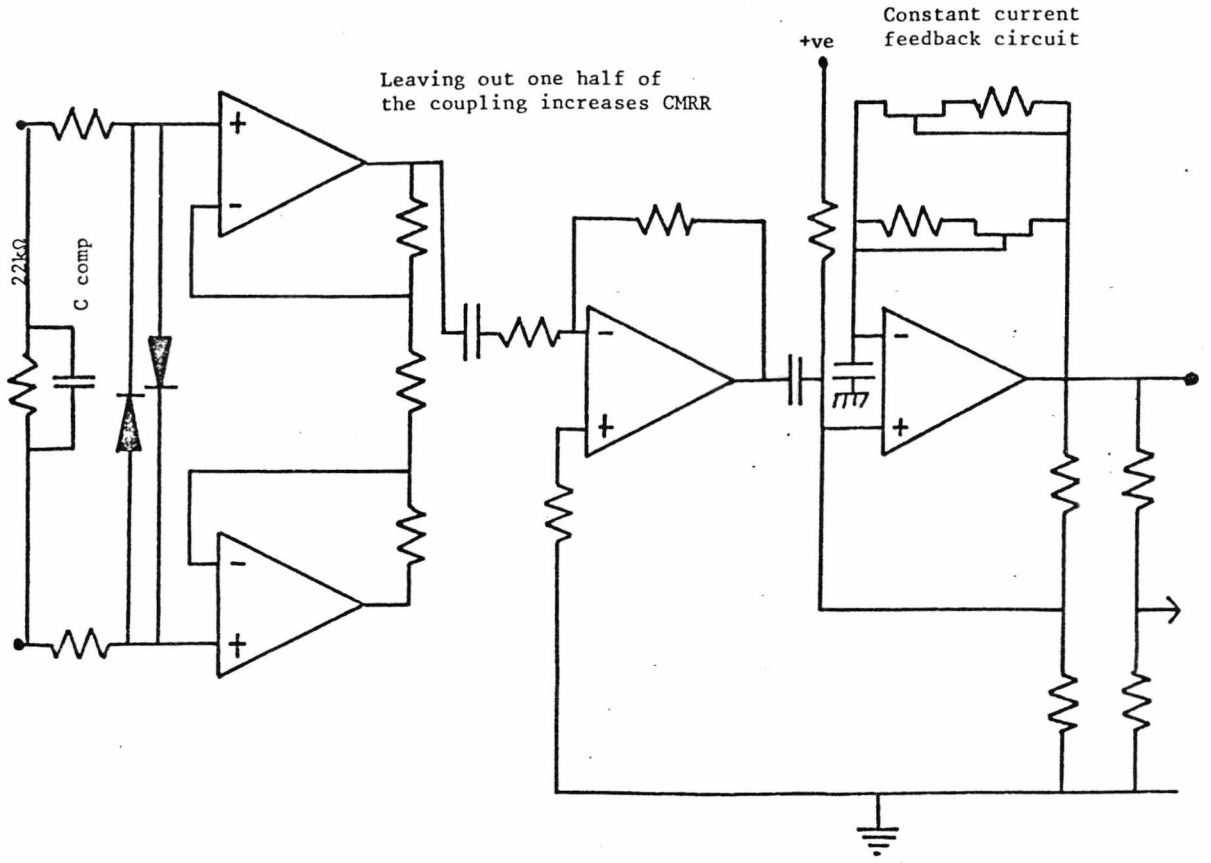
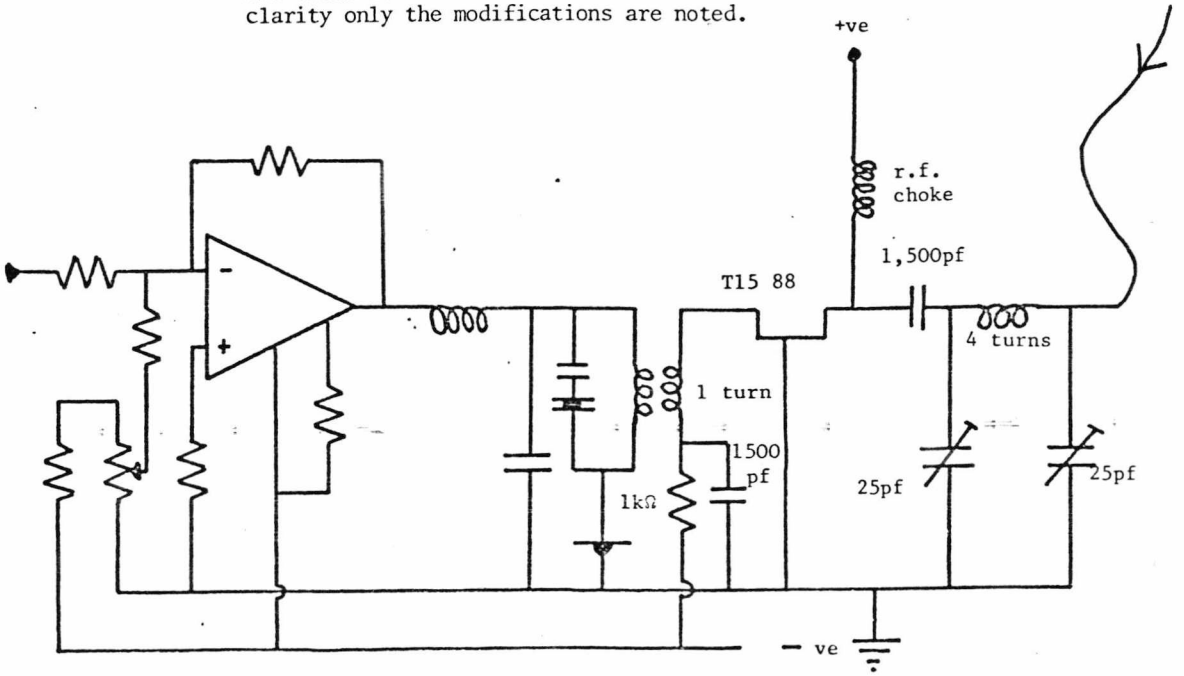


FIG. 1. The modified circuit diagram of the ECG transmitter. For the sake of clarity only the modifications are noted.



the intensive and coronary care units, especially since relatively few problems seem to be encountered with infections at the site of drip feeds, catheter insertion points - or even stitching of surgical wounds.

The under the skin electrodes would be no use in exercise or ambulance situations as they are of a more permanent nature, but normal disposable ones could be used, with a low input impedance amplifier to minimise artefact.

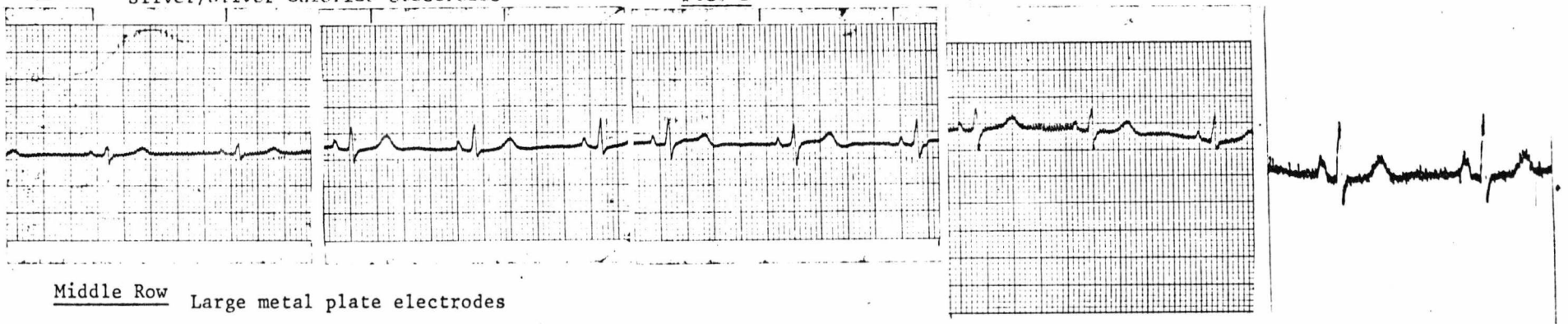
Lewes[5] has, earlier, devised a form of surface electrode which consists of a square of nutmeg grater - strapped on as a normal metal plate electrode. The fine points of the metal all penetrate the skin and so come into contact with the body fluids. Good results were claimed for this method, and a similar electrode could be used on the exercise bicycle, or in the ambulance with a suitable low input impedance amplifier.

An interesting piece of work that was carried out, has meaningful implications for routine ECG recording in the hospital. It arises from the earlier work of matching the input of the amplifier to the electrode characteristics. Since this is not done with normal ECG machines used about the hospital, and different electrodes are used with different machines it was decided to conduct some comparison tests. *Figs. (2), (3)* and *(4)* show the results of leads I, II and III when recorded from three different electrode types and five different machines all from the same patient. (The machines were three standard clinical ones, one with an input of 12MΩ, and one with an input of 1MΩ and 0.01μF capacitor). It can be seen from the *Figs.* that the results show considerable variations i.e. lead III; all machines with small metal plate electrodes show oscillatory P waves, whilst for V trace and

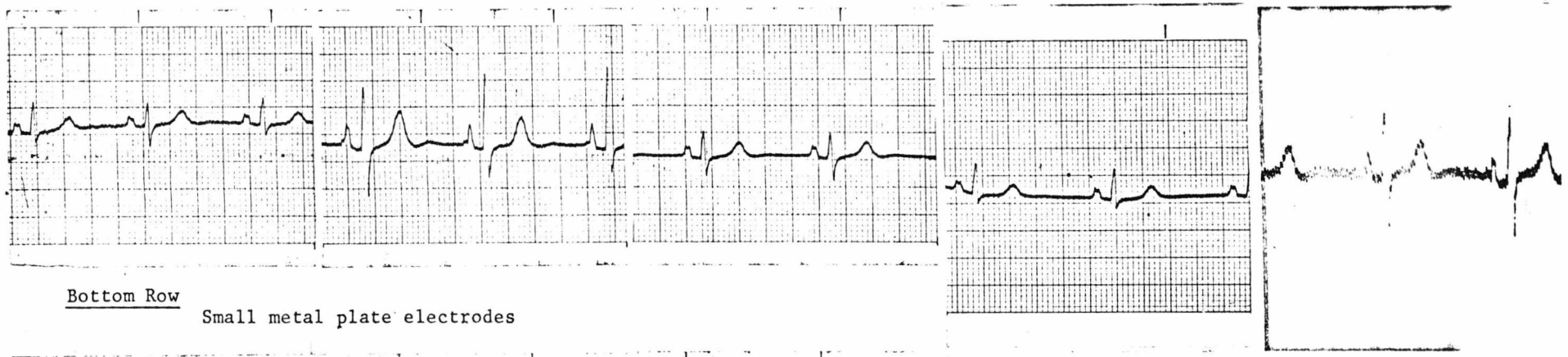
Top Row Silver/Silver Chloride Electrodes

FIG. 2

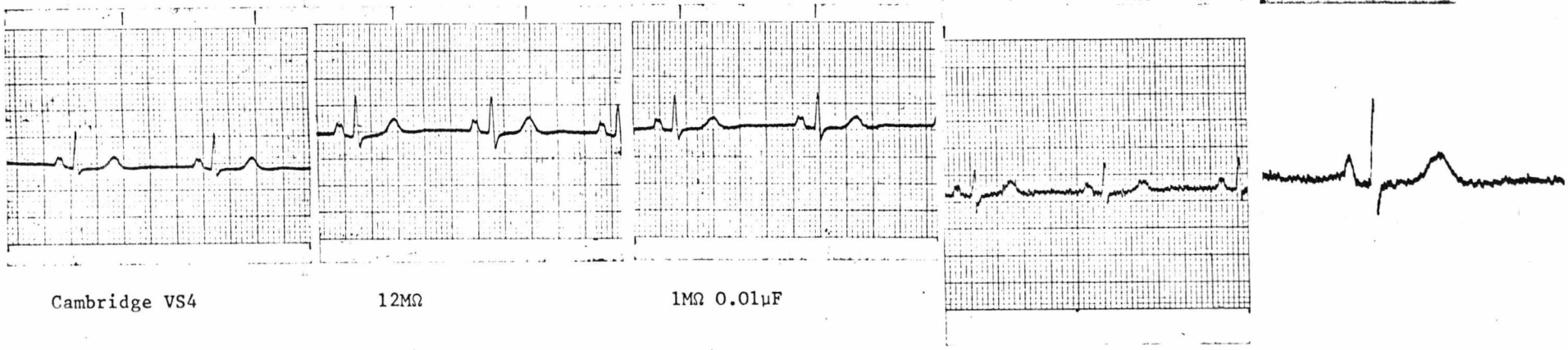
Lead I



Middle Row Large metal plate electrodes



Bottom Row Small metal plate electrodes



Cambridge VS4 12MΩ 1MΩ 0.01μF

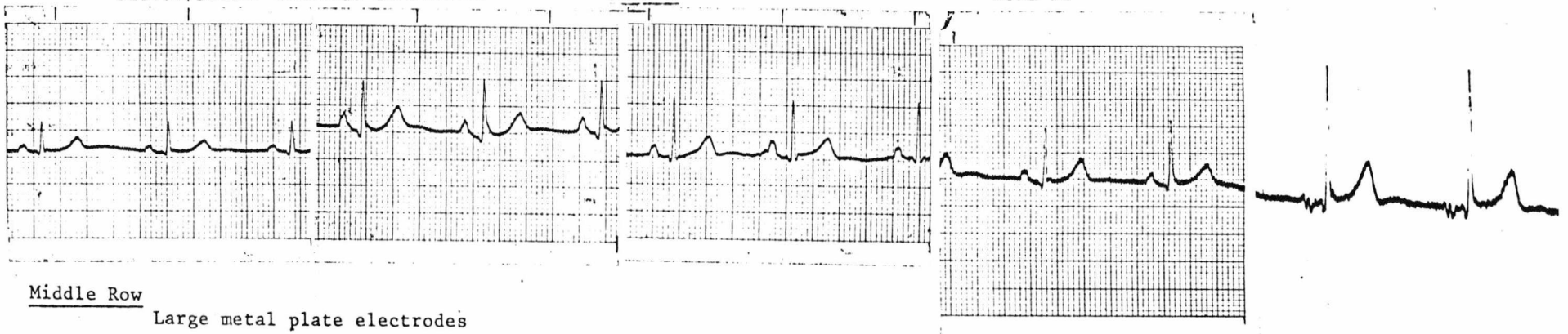
Hewlett Packard

Cambridge multi-channel analyser

Top Row Silver/Silver Chloride Electrodes

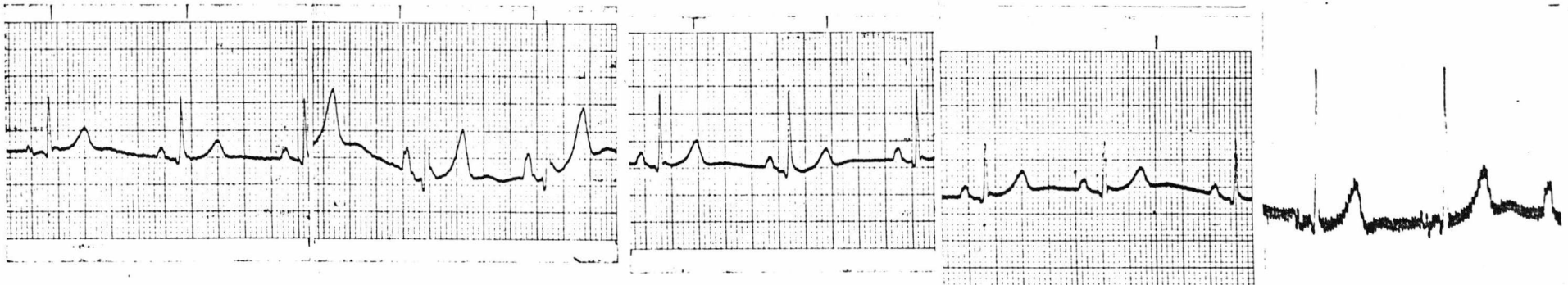
FIG. 3

Lead II

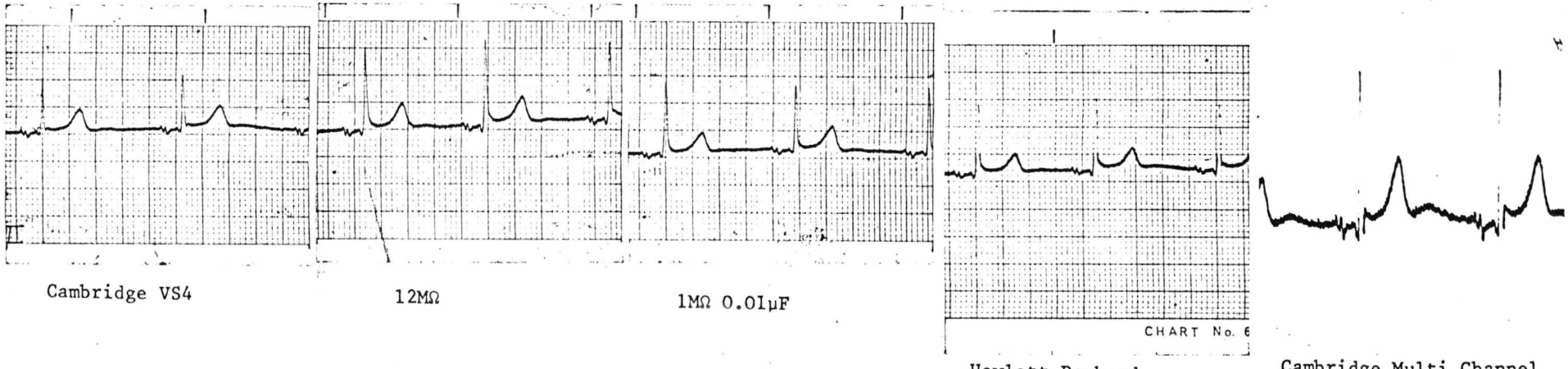


Middle Row

Large metal plate electrodes



Bottom Row Small metal plate electrodes



Cambridge VS4

12MΩ

1MΩ 0.01μF

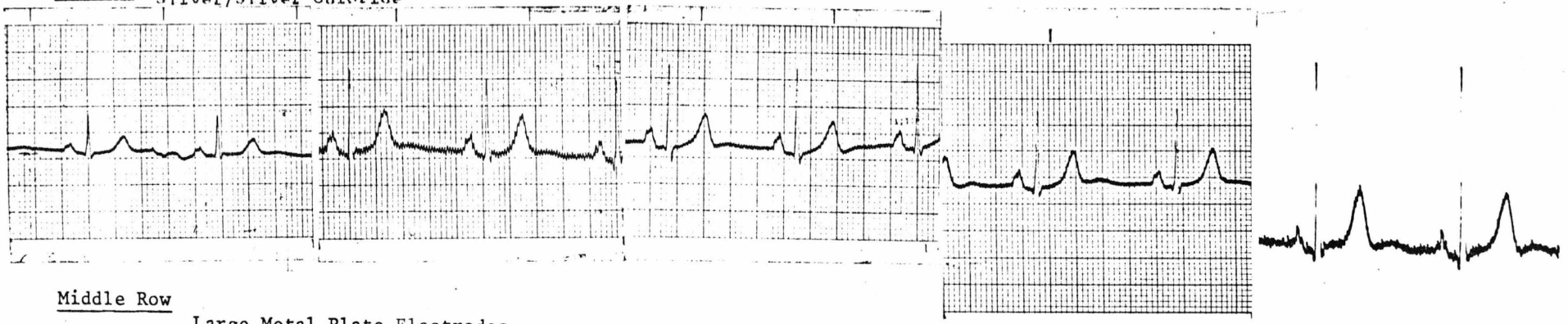
Hewlett Packard

Cambridge Multi Channel Analyser

FIG. 4

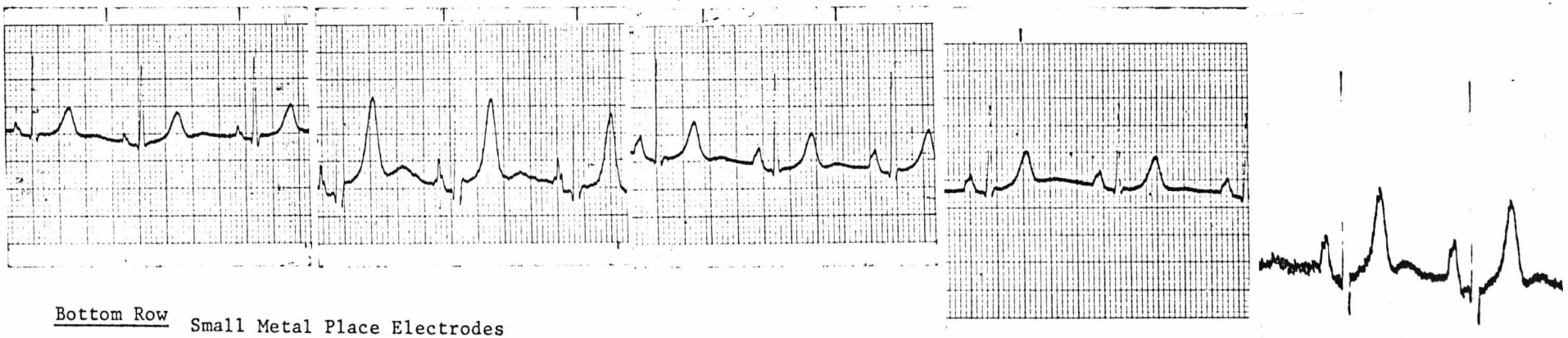
Lead III

Top Row Silver/Silver Chloride

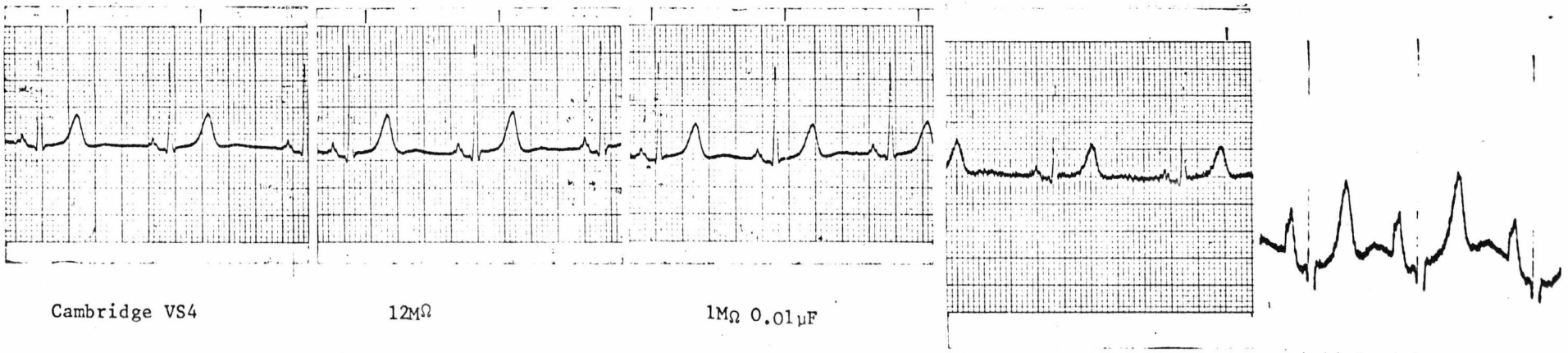


Middle Row

Large Metal Plate Electrodes



Bottom Row Small Metal Place Electrodes



Cambridge VS4 12MΩ 1MΩ 0.01μF

Hewlett Packard

Cambridge Multi Channel Analyser

large metal plate electrodes the P waves are normal - except for the Cambridge M.C.A. Other instances can be seen of variations between electrodes in any one machine and between machines for any one electrode.

This leads to the conclusion that matching of electrodes and amplifiers is important, and cardiac departments ought to look more carefully at this and have a policy of one machine per electrode type - or just use one electrode type alone. Certainly charts such as those of the *Figs. (2)* to *(4)* should be available in each ECG department as references, if the previous policy is difficult to implement.

8.3 Further Research Work

There is some further work that can be carried out upon the electronics of the ambulance telemetry system, but most of the effort should be directed towards its implementation and evaluation. The equipment will have to be decided upon and the training programmes for the ambulance men arranged. In the assessment of the unit, it will be important not just to examine the figures for patients with infarction that reached the hospital alive, but to find out the effect upon the overall mortality rates. Another point is to investigate how satisfactory medical staff find it, having to make diagnoses from an ECG and reported symptoms alone. If it is found that it cannot be done well, then that leaves no alternative but to employ a doctor and nurse in the vehicle. On the electronics side, the decoder could be modified a little, by the improvement of the pre-amplifiers and increasing the rate of roll-off of the final filter, to remove any 2KHz signal from the ECG. The deviation

of the tone in the encoder could be reduced to ±100Hz instead of ±1KHz which would improve the signal-to-noise ratio of the output.

For the ward telemetry, the usefulness of the devices must be evaluated, especially in terms of how the ward staff react to them. The long term effects of under the skin electrodes must be evaluated to see how comfortable they are (or not) and to investigate ways of minimising any problems of infection of reaction that occur. Many more experiments to confirm their better offset stability when used with low impedances could be performed.

Much work remains to be done determining the theoretical and practical aspects of low input impedance recording, especially with respect to the electrodes. The effect of the skin as a semi-permeable membrane upon the discharge of the concentration cell formed by the electrodes should be determined, and it would also be useful to learn more about the quantities of charge that are involved in establishing the equilibrium of the electrode/electrolyte interface. This could be done for several types of surface electrodes and under the skin electrodes, since this has an effect upon the time of discharge of the offset potential and the values of R_{in} that are needed. The effect of low input impedances on defibrillator discharge and the recovery time of the signal should also be investigated on real patients.

The properties of surface electrodes and the variation of the resistive and capacitative parts of the electrode impedance with frequency can be determined, and then used to find the effect of this upon the frequency spectrum of the signal when a low input impedance is used. From this and the previous

work it should be possible to determine an optimum value of R_{in} for different types of electrodes.

One important piece of work to be done is to establish some standards for the types of electrodes used in routine monitoring. The variations in the ECGs recorded from the different combinations of electrodes and machines indicate strongly the need for standardisation. The American Heart Association has set standards for the bandwidth of the amplifiers used, but this work has shown that unless the relation between electrode and input is accounted for, then these standards are undermined.

The work undertaken in this thesis is but a very small part of all that could be done in patient monitoring and even then has only examined the collection of signals and has not begun to touch upon their analysis. But the work has shown that however the signals are analysed or whatever the reaction or opinion of the clinician to the signals, (for an expansion of this very interesting theme see the Appendix) they must still be carefully collected. It is not only the development of complex electronic processors and analysers and displays that is important, but it is also at the very beginning of the process, at the electrode, where significant advances in patient monitoring can still be made.

REFERENCES

(1) M. CAMERON et al "Follow-up of emergency ambulance calls in Nottingham: implications for coronary ambulance service" 1975. British Medical Journal, 15th Feb., p. 384-386.

(2) P.N. YU "Pre-hospital care of acute myocardial infarction" 1972. Circulation, Jan., Vol. 45, p. 189-204.

(3) J. SCURR "Private Communication" 1976. E.C.G. Dept., Kent and Canterbury Hospital.

(4) L.A. GEDDES "Electrodes and the measurement of bioelectric events" 1972. Wiley Interscience.

(5) D. LEWES "Multipoint electrocardiography without skin preparation" 1966. World Medical Electronics, Vol. 4, p. 240-245.

APPENDIX

THE WIDER ASPECTS OF THE MEASUREMENT AND ANALYSIS OF CLINICAL EVENTS

The thesis has been concerned with the measurement of the electrocardiogram at three periods during the course of the illness of a cardiac patient. Until now, nothing has been said about the general reaction of the medical staff to electronic machines and their results - nor about the possibilities of alternative forms of analysis of collected data. It is the purpose of this appendix to discuss some of these wider issues and to show that clinical measurement involves more than just measuring parameters and displaying the results to the clinicians.

Contrary to expectations, presenting information to them is not as easy as it would seem. Giving them measurements, and accurate ones too, of important parameters is relatively straightforward but getting them accepted is another matter entirely. A Consultant has told me that, often, he notes the heart rate reading from the ECG machine connected to a patient, and then observes the patient to see what he thinks the heart rate ought to be. If there is a difference in the values then out comes his watch, and a rate reading is taken from the patient's pulse. Others have told me of blood gas analyses sent back if they do not match up to the doctor's expectations, blood pressures re-recorded and other similar circumstances.

Whilst this is quite upsetting to the bio-engineer who spent time developing the transducer and associated circuitry, there is, hopefully, more to this phenomenon that just a

reactionary approach to instrumentation upon the behalf of the clinician.

Throughout the latters' training they have been taught to observe their patients, to note the details of colour, perspiration, dilation or constriction of the pupils and many, many other things. Hippocrates(5th Century B.C.) the 'father of medicine' used and taught this approach and so it has continued. Thus, from all these observations and their experience they are trained to have opinions on the patients' condition and so they tend to use electronic measurements to confirm their diagnoses, rather than produce a diagnosis from measurements.

The bio-engineer must learn to appreciate this and it is perhaps, not such a bad approach after all. A colleague[1] has told me of an instance from his experience when too much reliance was placed on machines and measurements. For many years a large chemical company used trained personnel to interpret mass spectrograph analyses of their products until a computerised system was introduced. In certain instances rather stupid results were output from the computer, but were believed because they did come from a computer! Later on they were questioned and re-examined by the trained personnel who agreed that a silly mistake had been made. A much greater error was made in assuming that whatever issued from the computer was right and so nobody gave the results the check they needed.

Perhaps the critical approach of the clinician will avoid this problem occurring in medical practice. It is better to have doubtful results questioned than just accepted.

The preceding comments are cautionary and are not meant to imply that there is an absolute distrust of measurement in

medicine and ceaseless arguments with the results. On the contrary many machines are used and trusted by clinicians, thankful for the assistance, and much information is obtained that would otherwise be unavailable to them e.g., information on the foetus during pregnancy and childbirth, the condition of the heart valves as they operate in situ, and values of blood flow velocity and stroke volume can be obtained.

The healthy criticism of measurements must become even more marked when dealing with machines that make clinical decisions - simple or otherwise. There have appeared upon the market two examples of complex, expensive cardiac diagnostic machines. One is a bedside monitor, which can recognise one of several types of arrythmia, and then when one occurs, the ECG for several seconds before and after the event, is output on paper strip (£3,000). The second is a complete three lead (vector leads) ECG routine analyser, costing £20,000 at least and designed to cope with the routine work of a cardiac department. When I spoke to the respective sales representative neither of them could claim large scale sales though both were optimistic, blaming the lack of money in the Health Service. It is my opinion, however, that the cause may not only be this, but may lie elsewhere.

Enough problems have been found by clinicians with simple rate alarms connected to ECG monitors - so much so, in fact, that many of them are turned off, and hardly used[2]. Their suspicion of the usefulness of such a complex system as the bedside monitor, then, is not hard to understand. The cost is also prohibitive as it is a case of one machine per bed (at the time of writing). When patients are critically ill, then staff supervision is necessary. I am sure that no one will

trust a machine to do the job. When the crises are past and a machine can be left to do the job, then, I believe £3,000 a bed is too expensive for non-critical monitoring.

The bigger system may suffer from similar problems - who will trust completely the output of the system without checking? If it has to be checked is the machine then necessary? Some clinicians disagree with the diagnostic criteria used in the machine - and what of the patient? Often the latter seek the re-assurance that a clinician gives by just listening to his patient and prefer human contact when they are ill. Will they regard the computer as a vital part of their treatment or look upon it as a diagnostic vending machine - a complicated medical juke box? These problems I believe to be real and must be investigated by the producers of diagnostic machines with as much respect as the technical problems of measuring a signal and then classifying it. Even in periods of economic prosperity, I have my doubts as to whether hospitals should invest such large sums, on equipment as it is at the present time.

One point that is made by the supporters of diagnostic equipment is that the removal of the human element means more accurate first time diagnoses[3]. The bedside monitor was tested against the ward staff in an intensive care unit and was found to detect 99% of clinical alarms to the 48% detected by the staff during the same period. This apparent discrepancy is countered by the studies of Professor D. Taylor[4] who states that an element of preprocessing occurs by the ward staff, and not all of the 50% difference in detection rates were just errors, many, he believes, had been noticed but taken together with other clinical signs they had been ignored. The number of real emergencies missed was much less and he doubted

if any patients were actually lost. He also found from a study of acute surgical cases, although the diagnoses were right only 40% of the time, the correct treatments were given in 85% of the cases (due to the overlap of treatments between illnesses). This helps to mitigate the claim made by automatic diagnosis engineers that machines make more reliable diagnosis than doctors - since it is more important the treatment be right than the diagnosis.

Professor Taylor also mentioned some other interesting points about pre-processing of patient data which has a bearing on the earlier discussion on measurements. A machine was used to continuously monitor different patients, and a nurse was required to make her own recordings of the patient at the usual, regular intervals. Unbeknown to her, another monitor was recording the output from the machine. In the case of a blood pressure measurement, although the true readings were approximately 80 mmHg the nurse did not like the look of the patient, and so actually recorded only 60 mmHg and this was over a reasonable period of time throughout her duty - not just an isolated instance. In another case, the nurse noticed a deteriorating trend in a patient's condition, and so the readings she was recording indicated this trend - but it was considerable time later before the machine itself responded and the true readings changed.

These examples (and there are others, I may add, which are not so complementary - such as the student nurse who only recorded a patient's temperature as 98.4^{o}F over several hours), all illustrate that the problems of patient monitoring do not only lie in building instruments to measure parameters a, b, or c. The whole problem includes not only the measurements but the clinicians' opinion of both the measurement and the patient

taken together, and also the patients response as well.

It is this many-sided approach to measurement that must be realised by the engineer - who must learn to live with it and appreciate its finer points as well. It is also this wide variety of factors involved in clinical decision making, that I believe stands in the way of the automated diagnosis machine - to take an ECG and analyse it is not enough. Unless these problems are faced, automation faces a meteor-like career - glorious albeit brief. Perhaps it is already over - "Medicine" said one of the Canterbury Consultants "is definitely labour intensive and not machine intensive".

REFERENCES

(1) T.J. STONHAM "Personal Communication" 1976. Electronic Laboratories, University of Kent.

(2) M. THOMAS et al "Analysis of 150 patients with acute myocardial infarction admitted to an intensive care and study unit" 1968. British Medical Journal, Vol. 1, p. 787.

(3) N.J. VETTER & D.G. JULIAN "Comparison of arrythmia computer and conventional monitoring in coronary care unit" 1975. The Lancet, May 24th, p. 1151.

(4) D.E.M. TAYLOR "Decision making in intensive care" 1975. Session 16, 15th International Conference of the Biological Engineering Society, Edinburgh.