STUDY OF THE MATHEMATICAL BASIS TOWARDS THE ANALYSIS OF CARDIAC SIGNALS

BY

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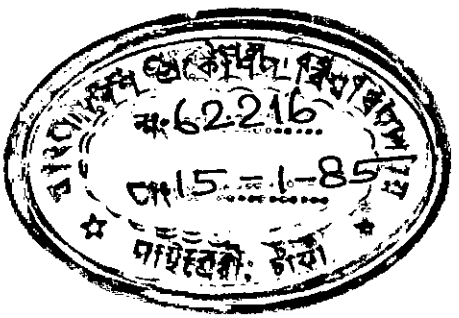
A THESIS

SUBMITTED TO THE DEPARTMENT OF ELECTRICAL AND ELECTRONIC ENGINEERING IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

OF

MASTER OF SCIENCE IN ENGINEERING



DEPARTMENT OF ELECTRICAL AND ELECTRONIC ENGINEERING,
BANGLADESH UNIVERSITY OF ENGINEERING AND TECHNOLOGY,
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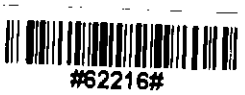
JANUARY 1985



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ACKNOWLEDGEMENT

It is a great pleasure on the part of the author to acknowledge his profound gratitude to his supervisor, Dr. Shamsuddin Ahmed, Professor and Dean of the Faculty of Electrical and Electronic Engineering, Bangladesh University of Engineering and Technology (BUET) for his support, advice, valuable guidance and this constant encouragement through out the progress of this work.

The author also wishes to express his thanks and deep sense of gratitude to Dr. A. M. Zahoorul Huq, Supernumerary Professor of Electrical and Electronic Engineering department for his valuable suggestion and keen interest in this work.

The author also expresses his sincere gratitude to Dr. S. F. Rahman, Professor and Head, Department of Electrical and Electronic Engineering, BUET, for providing facilities in the department and various stages of the work.

Grateful acknowledgement is made to Dr. M.K.Alam and Dr. M. A. Matin, Associate Professors of Electrical and Electronic Engineering Department for their kind help during the work.

The author wishes to thank BUET authority for awarding him a Teaching Assistantship during his study in BUET.

Lastly, thanks are due to all who helped directly or indirectly at different stages of the work.

AUTHOR

ABSTRACT

The nature and origin of the electrical activity of the heart could not be understood fully due to the fact that the underlying principle of bioelectric phenomenon related with cardiac (heart) activity is a very complex one. The complexity arises due to many factors. The body in which heart is situated is not a simple homogeneous medium, rather inhomogeneity arises in different parts of the body. The potential recorded on the body surface due to cardiac activity and its interpretation known as ECG plays an important role in clinical diagonsis. This diagonsis depends on different pathologic condition and on large volume of statistical data.

The ultimate goal of the study of electrocardiography is to know the conditions of the heart under different circumstances. Keeping this in view many mathematical models that may represent the electrical activity of the heart were developed. In this work an attempt has been made to study the various mathematical models considered so far. Since a knowledge of electrophysiology is a basic need for the understanding of the electrical activity of biological system, the electrical activity of different types of cells (especially cardiac muscles and cells) is reviewed.

Among the mathematical model of electrical activity of the heart the most simple one is the dipole model. But experimental evidence shows that this model is a crude one. So if a proper correction is made to dipole model by introducing quadrupole and octopole terms, then it is observed that this model to some extent resembles the actual characteristic of the heart. Another proposed model is the multiple dipole model. But the fact is that none of these models developed is an exact one and these models are subjected to various limitations.

An experimental investigation has been carried out in order to study the effect of blood flow constraint on electrocardiographic records. It is seen from the experimental data that there are noticeable changes namely in heart beats, QRS complex amplitude and QT intervals. To make these data useful as help to clinical diagonsis large volume of data for different individuals at different pathologic condition would be needed.

CONTENTS

LIST OF SYMBOLS:

A^{o}	=	Angstrom
μ	=	micron.
mEq/	=	milli equivalent per liter.
Na^{+}	=	Sodium.
k^{+}	=	Potassium.
Cl^{-}	=	Chlorine.
Ca^{+}	=	Calcium.
C	=	Concentration of ions.
ϕ	=	Surface Potential.
ψ	=	Scalar function.
$\bar{p}$	=	dipole moment.
$\bar{J}_i$	=	Impressed current density/dipole moment per unit Volume.
I_v	=	Volume current density.
$\bar{E}$	=	Electric field.
$\bar{A}$	=	Vectormagnetic Potential.
$\bar{k}$	=	Surface current density.
δ	=	delta function.
σ	=	Conductivity.
ρ	=	Charge density.
μ	=	Permeability.
ϵ	=	Permittivity.
ρ	=	Resistivity.

(x, y, z) = Rectangular cordinates.

(ξ, η, ζ) = Co-ordinates for source location.

(R, θ, ϕ) = Spherical Co-ordinates.

α = angle.

$\bar{\rho}$ = radial distance vector.

Ω = Solid angle.

∇ = Operator del.

Chapter-1

Introduction:

Electrocardiography is concerned with the measurement and interpretation of the time variation of potential at the surface of the body due to the electrical activity of the heart. At any instant the system consists of some distribution of current dipole sources (the active cells in the heart) located in a passive volume conductor (the remaining body tissue). From theoretical stand point the external effect of a source within an arbitrary sphere that contains the heart, including all secondary effects which arise as a consequence of electrical inhomogeneities such as intracavitary blood, can be replaced by equivalent multipole source. This equivalence is with respect to the region external to the arbitrary enclosing sphare. The present electrocardiographic practice is based on a dipole representation. The dipole is capable of yielding good approximation of the body surface potential has been shown by Frank. However, others[5-8] have raised doubts that this simple dipole model is rather inaccurate. Taccardi, Horan & Brody and others have shown on a qualitative basis that nondipolar potentials do exist . So the cardiac activity can be best represented by multipole expansion. In this case the nondipole terms in the multipole expansion provides a convenient correction to the conventional heart source. In the representation of equivalent cardiac generator by summation of multipole source, the surface potential distribution is considered as the sum of contributions from individual multipole fields.

The dipole is the first term in the sum and can be measured by orthogonal lead system. The quadrupole is the next term in the series. If the surface potential distribution is approximated by a dipole, then approximation is enhanced by the addition of quadrupole.

A knowledge of electrical activity in individual cell is the very basis towards the understanding of the complicated phenomenon of the electrical activity of the heart. Keeping this in view a description of the cell and its membrane, development of membrane potential, action potential and their propagation through different types of muscle, is given in Chapter 2.

Chapter 3 describes the physiology of cardiac (heart) muscle and the mechanical activity of the heart controlled by the electrical activities of the different parts of the heart.

In Chapter 4, the recordings of the body surface potential (known as electrocardiograms) and its nature of variation with time and process of such recording by attaching different leads on body, is described.

In Chapter 5, mathematical analysis of electrical activity of heart, is reviewed considering the heart as an electrical source placed in homogenous medium. The case of inhomogeneity is also considered. The drawbacks of dipole

representation of heart and the multipole representation as a correction to dipole representation is also discussed.

Chapter 6, contains a discussion on the experimental results of the effect of blood flow constraint on electrocardiographic records.

As a concluding remark of the project in chapter 7, a discussion on the limitations of electrocardiography and the scope of magnetocardiography and the scope for further research in this field is given.

Chapter 2

Cell and its electrical activity

2.1.1 The cell and its function :

A cell may be defined as "a unit of biological activity delimited by a semipermeable membrane and capable of self reproduction in a medium free of other living system."

Organisation of the cell:

A typical cell as seen by the electron microscope is illustrated in Fig 1. Every cell contains a very prominent, Spherical structure, known as nucleus. Another major part of the cell is cytoplasm. The nucleus is separated from the cytoplasm by a nuclear membrane and the cytoplasm is separated from the surrounding fluids by a cell membrane. The different substances that make up the cell are collectively called protoplasm.

Protoplasm:

All living cells are composed of protoplasm, a viscous fluid consisting mostly of water and composed principally of carbon, hydrogen, oxygen and nitrogen, although other elements such as calcium, potassium, sodium and sulfur are also present. The protoplasm of nucleus is termed "nucleoplasm."

The concentration of inorganic compound in protoplasm is very similar to that of the concentration in sea water. There are also many organic compounds. The carbohydrates range from the simple sugars such as glucose and fructose, upto the very

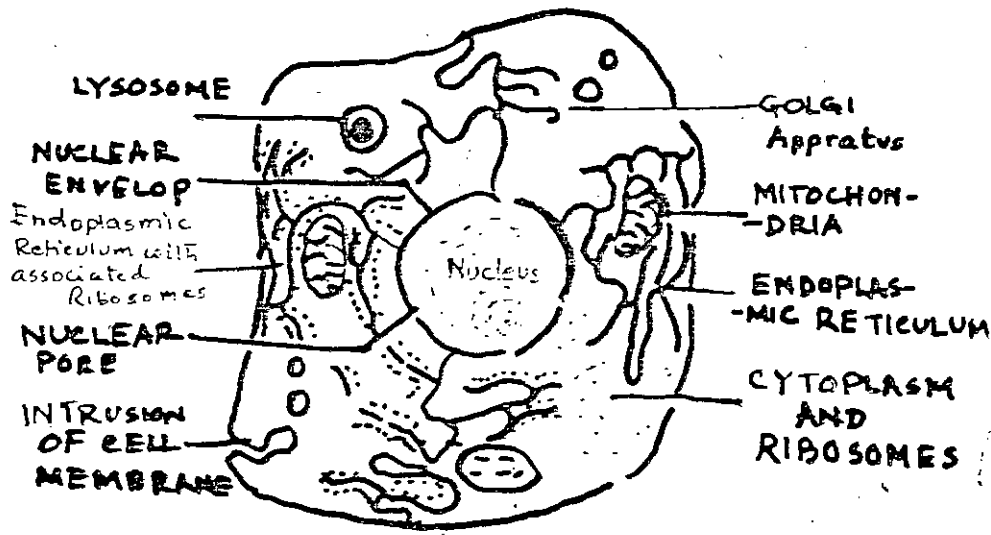


FIG 1

SCHEMATIC REPRESENTATION OF A CELL

complex molecules like glycogen. It also contains different types of amino acids and numerous fatty acids. Protoplasm of most animal cells contains about 80 percent water, 15 percent protein, 3 percent fat, and 1 percent carbohydrate, with electrolytes making up the remaining 1 percent.

2.1.2 Physical structure of the cell:

Besides the fluid and chemicals, cell contains highly organized physical structures called organelles. Some principal organelles of the cell, are the cell membrane, nuclear membrane, endoplasmic reticulum, mitochondria lysosomes, Golgi complex, centrioles, cilia and microtubules.

Cell membrane:

Essentially all physical structures of the cell are lined by a membrane composed primarily of lipids and proteins. The cell membrane is extremely thin and difficult to measure in most cases. The thickness of the membrane is approximately about 70 to 150A°. Cell membranes appear to be a double layer of fat molecules set between two layers of protein. Pores about 8A° in diameter pierce the membrane.

The nucleus :

Most cells contain a small spherical mass which, because of its somewhat different consistency compared to the rest of the protoplasm, is clearly seen in most preparations. This

mass is termed the nucleus. It is surrounded by its own membrane, the nuclear membrane. If the nucleus is removed from a cell, the cell dies. This indicates that the cell cytoplasm is dependent upon, and regulated by the nucleus.

The most important constituent of nucleus is nucleic acid. There are two nucleic acids : deoxyribonucleic acid (DNA) and ribonucleic acid (RNA). They are vital to cell replication in that they control the amino acid content and arrangement in protein synthesized by the cell.

Endoplasmic reticulum :

The endoplasmic reticulum forms a series of small canals through the cytoplasm. It is thought that various substances pass through these canals in moving from membrane to nucleus. Closely associated with the membranes which line the canals are tiny granuals termed microsomes. Because they contain high concentration of RNA, they are often termed as ribosomes. They are essential for protein synthesis.

Mitochondria:

The mitochondria are seen to be relatively large structures dispersed about the cytoplasm having a diameter of about 0.5 μ and a length of about 1.5 μ . The mitochondria are enveloped by a double membrane. The inner membrane sends branches inward thus forming many connecting compartments. The inside of the innerlayer and outside of the outer layer are covered with tiny granuals which measure not more

than 50 to 60A° and they play a major role in the function of mitochondria.

Cells do many things and for all these activities energy is required. The mitochondria have been termed the "principal power plants of the cell". This energy is liberated in the following way. The many infoldings of the inner membrane form shelves onto which the oxydative enzymes of each cell are attached. In addition the inner cavity of the mitochondrion is filled with a gel matrix containing large quantities of dissolved enzymes that are necessary for extracting energy from nutrients. These enzymes operate in association with the oxidative enzymes on the shelves to cause oxidation of nutrients, there by forming carbon dioxide and water. The liberated energy is used to synthesize a high energy substance called adenosine-triphosphate (ATP). ATP is then transported out of the mitochondrion, and is diffused through out the cell to release its energy whereever it is needed for performing cellular functions.

Golgi Complex:

The Golgi complex (or apparatus) is a network of threads in the cytoplasm of the cell and consists of several large, apparently empty vacuoles surrounded by a membrane. The precise function of this structure remains uncertain. It has been suggested that it is some sort of intracellular pump that regulates the movement of fluids in the cell and the expulsion of secretory

products from the cell. It may well be necessary for the secretion of very large molecules by the cell. Recent evidence indicates that the Golgi region is the site where carbohydrate are linked to protein.

Centrosome:

In some cell the centrosome can be seen as a clear area near the nucleus in which there is a very small granule, the centriole. It is generally seen that the centriole is visualized during cell division.

During cell division the centriole divides. The two products of this division then move away from one another until they lie on either side of the cell with the nucleus between them. The centrimose and the centrioles play an important role in cell division.

Lysosomes:

In many cells, lysosomes can be identified as roughly spherical bodies about a half micron in diameter and they have a fine granular makeup. The granules within lysosomes are approximately 75A^{o} in diameter.

Lysosomes apparently serve at least four functions :

(i) the digestion of large particle that enter the cell,

(ii) the digestion of intracellular substances,

(iii) the digestion of cell itself,

(iv) the digestion of substance external to the cell.

Cilia and Flagella :

Some cells have hairlike processes capable of vibratory or lashing movements. Numerous shot processes are termed cilia. If there is a long taillike structure it is a flagellum. Both cilia and flagella serve to propel the cells through a fluid medium. But there are also ciliated cells comprising stationary tissue, in which instance the cilia serve to clean the surface.

2.1.3 Transport through cell membrane:

The cell membrane is essentially a sheet of lipid material, called lipid matrix and partially covered on each surface by a layer of protein. A cell is surrounded by such a membrane.

The fluid inside cells of the body is called intracellular fluid and is different from that outside the cell called extracellular fluid. The extracellular fluid circulates in the space between the cells and also mixes freely through the capillary walls with the fluid of the blood. It is the extracellular fluid that supplies the cells with nutrients and other substances needed for cellular function. But before these substance is utilized they must be transported through the cell membrane.

(15)

The table 2.1 gives the representative composition of both extracellular and intracellular fluids. It is noted that extracellular fluid contains large quantities of sodium but only small quantities of potassium. Exactly the opposite is true of the intracellular fluid. Similar is the case with other components also. These differences between the components of the intracellular and extracellular fluids are extremely important to the life of the cell.

Substances are transported through the cell membrane by the two major processes, "diffusion" and "active transport". Diffusion means the free movement of substance in a random fashion caused by the normal kinetic motion of matter, where as active transport means movement of substance in chemical combination with carrier substance in the membrane and also movement against an energy gradient such as from low concentration state to a high concentration state, a process that requires chemical energy to cause the movement.

Diffusion:

All the molecules and ions in the body fluids, including both water molecules and dissolved substance are in constant motion, each particle moving its own separate way. The continual movement of molecules among each other in liquids or in gas is called diffusion.

Table 2.1

Chemical compositions of extracellular and intracellular fluids.

	EXTRACELLULAR FLUID	INTRACELLULAR FLUID
Na^+ -- --	142 mEq/L	10 mEq/l
K^+ -- --	5 mEq/l	141 mEq/l
Ca^{++} -- --	5 mEq/l	1 mEq/l
Mg^{++} -- --	3 mEq/l	58 mEq/l
Cl^- -- --	103 mEq/l	4 mEq/l
HCO_3 -- --	28 mEq/l	10 mEq/l
Phosphates --	4 mEq/l	75 mEq/l
SO_4^{--} --	1 mEq/l	2 mEq/l
Glucose --	90 mgm.%	0 to 20 mgm.%
Amino acids --	30 mgm.%	200 mgm.%?
Cholesterol Phosphalipids Neutral far --	0.5 gm.%	2 to 95 gm.%
P_{O_2} -- --	35 m.m.Hg.	20 mm.Hg ?
P_{CO_2} -- --	46 mm.Hg	50 mm.Hg ?
pH -- --	7.4	7.0

The rate of diffusion of a substance from one area to another depends on the following factors.

(1) The greater the concentration difference, the greater is the rate of diffusion,

(2) The less the molecular radius, the greater is the rate of diffusion,

(3) The shorter the distance, the greater is the rate,

(4) The greater the cross-section of the chamber in which diffusion is taking place the greater is the rate of diffusion,

(5) At higher temperature the diffusion rate is higher.

Considering all the factors we can write,

$$\text{Diffusion rate} \propto \frac{(\text{Concentration difference} \times \text{Cross-sectional area} \times \text{Temperature})}{(\text{Molecular radius} \times \text{Distance.})}$$

Net Diffusion Through Cell Membrane & The Factors That Affect it:

A cell membrane is essentially a sheet of lipid material, called lipid matrix, partially covered on each surface by a layer of protein. The fluid on each side of the membrane are believed to penetrate the protein portion of the membrane with ease, but the lipid portion of the membrane is an entirely different type of fluid medium, acting as a limiting boundary between the extracellular and a intracellular fluids.

The two different methods by which the substances can diffuse through the membrane are : (a) by becoming dissolved in the lipid and diffusing through it in the same way that diffusion occurs in water or (b) by diffusing through minute pores that pass directly through the membrane at wide intervals over its surface.

It is noted that the substance that diffuse in one direction can also diffuse in other direction.

It is not the total quantity of substance diffusing in both directions through the cell membrane that is important to the cell but instead the 'net quantity' diffusing either into or out of the cell.

The following factors determine the net diffusion of substance: Permeability of the membrane, concentration difference of the substance across the membrane, the electrical potential difference across the membrane and the pressure difference across the membrane.

a) Permeability: The permeability can be defined as the rate of transport through the membrane for a given concentration difference. The permeability of membrane is not constant under different condition. For instance excess calcium in the extra cellular fluid causes the permeability to decrease and diminished calcium causes considerably increased permeability.

(b) Effect of Concentration Difference:

Let C_1 and C_2 be the concentration of substance in outside and inside of the membrane. The diffusion of substance from outside to inside depends on C_1 and those from inside to outside depends on C_2. If C_1 is greater than there will be a net diffusion from outside to the inside of the cell (Fig. 2.2).So we can write,

$$\text{Net diffusion} \propto P (C_1 - C_2)$$

where P is the permeability of the membrane.

(c) Effect of an Electrical Potential Difference:

If an electrical potential difference is applied across the membrane, ions because of their electrical charges will move through the membrane even though no concentration difference exists to cause their movement. In the fig (2.3) there is equal concentration of negative charges in inside and outside the membrane. If an electrical potential difference is applied with inside positive and outside negative polarity then negative ions from outside will be repelled by its negative polarity of the applied field and at the same time will be attracted towards inside due to the positive polarity of the applied field. After some time the concentration of ions will be greater in inside of the membrane. The developed concentration difference tends to cause the movement of ions towards outside which is opposite to that of due to the

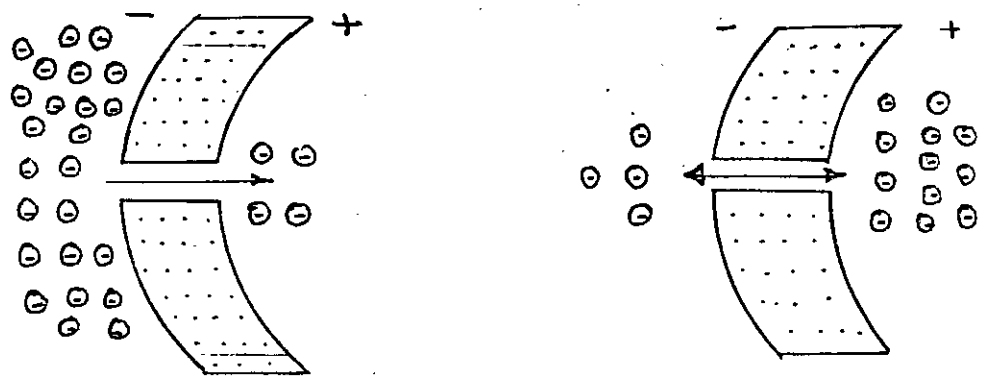


FIG 2.3. ELECTRICAL POTENTIAL DIFFERENCE.

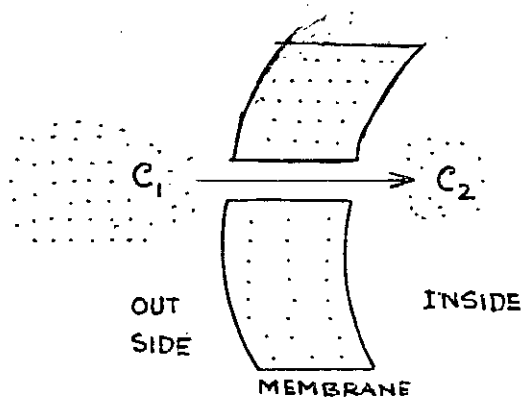


FIG 2.2
CONCENTRATION
DIFFERENCE

electrical potential applied across the field. When the concentration difference rises high enough, the two effect exactly balances each other.

(d) Effect of Pressure Difference:

At times considerable pressure difference develops between two sides of the membrane. This occurs, for instance, at the capillary membrane, which has a pressure approximately 23mm Hg greater inside the capillary than outside. In this case increased amount of energy is available to cause net movement of molecules from the high pressure side towards the lowpressure side thereby causing a net diffusion through the membrane.

Net Movement of Water Across Cell Membrane:

The most abundant substance to diffuse through the cell membrane is water. Usually enough water ordinarily diffuse through cell membrane per second in each direction which is 100 times the volume of the cell itself. Normally the amount that diffuses in two directions is so precisely balanced that not even the slightest net movement of water occurs. Therefore volume of the cell remains constant. However, under certain condidtions, a concentration difference of water can develop across the membrane, just as concentration differences for other substance can also occur. When this happens, net movement of water does occur across the membrane of the cell, causing the cell either to swell or to shrink, depending on the direction

of net movement. This process of net movement of water caused by concentration difference is called "Osmosis".

Active Transport:

From the data given in table 2.1 it is seen that concentration of potassium in extracellular fluid is very low and its concentration in intracellular fluid is very high yet there is a continuous transportation of patassium from outside of the cell towards inside of the cell. Conversely, other substances frequently enter the cell and must be removed even though their concentrations inside are far less than outside. This is true of sodium ions. But it is seen that no substance can diffuse against a concentration gradient or "uphill". To cause movement of substance uphill, energy must be imparted to the substance. When there is movement of molecules through membrane against a concentration gradient (or uphill against an electrical or pressure gradient) the process is called "active transport". Among the different substances that are actively transported through the cell membranes are sodium ions, potassium ions, calcium ions, iron ions, hydrogen ions, Chloride ions, iodide ions, urate ions, several different sugars and amino acids.

Basic Mechanism of Active Transport:

The mechanism of active transport is believed to be similar for all substances and to be dependent on transport by carriers. Fig. 2.4 illustrates the basic mechanism, showing a substance S

entering the outside surface of the membrane where it combines with carrier C. At the inside surface of the membrane, S separates from the carrier and is released to the inside of the cell. C then moves back to the outside to pickup more S.

Here the energy is imparted to the system incourse of the transport, so that transport can occur against a concentration gradient (or against an electrical or pressure gradient). It is supposed that the carrier has a natural affinity for the substance to be transported so that at the outer surface of the membranme the carrier and the substance readily combine. Then the combination of the two diffuse through the membrane to the inner surface. Here an enzyme-catalyzed reaction occurs, utilizing energy from ATP to split the substance away from the carrier. But the released substance, being insoluble in the membrane, can not diffuse backward through the lipid matrix of the membrane. Therefore it is released to the inside of the membrane while the carrier alone diffuse back to the outside surface to transport still another molecule of substance in the inward direction.

Active Transport of Sodium and Potassium:

From the table 2.1 it is seen that the sodium concentration outside the cell is very high in comparison with its concentration inside, and the converse is true for potassium. Sodium and potassium ions are positively charged ions and for this reason they can pass through cell membrane with extreme difficulty whereas negatively charged ions can pass through the cell membrane

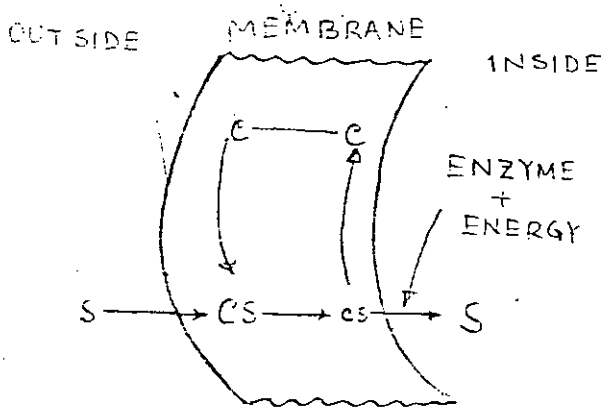
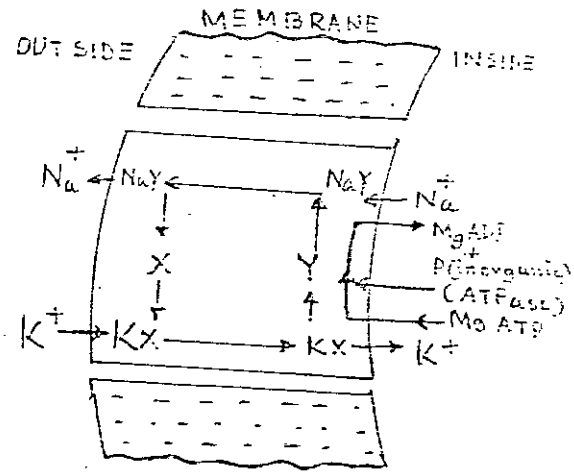


Fig. 2.4. BASIC MECHANISM OF ACTIVE TRANSPORT.

FIG. 2.5. POSTULATED MECHANISM FOR ACTIVE TRANSPORT OF SODIUM AND POTASSIUM.



pores much more easily. So minute quantities of sodium and potassium can diffuse through the pores of the cell. If such diffusion should take place over a long period of time, the concentration of the two ions would eventually become equal inside and outside the cell unless there were some means to remove the sodium from the inside and to transport potassium back in.

Furtunately, a system for active transport of sodium and potassium is present probably in all cells of the body. The mechanism is illustrated in the Fig 2.5 which shows sodium (Na) inside the cell combining with carrier Y at the membrane surface to form a large quantities of the combination Na Y. This then moves to the outersurface where sodium is released and carrier Y changes its chemical composition slightly to become carrier X. This carrier then combines with potassium K to form KX, which moves to the inner surface of the membrane where energy is provided to split K from X under the influence of enzyme ATPase, the energy being derived from Mg-ATP. At the same time carrier X is reconverted into Y, which transports a new sodium ion to the exterior, the cycle continuing indefinitely. The transport mechanism state above is believed to be more effective in transporting sodium than in transporting potassium, usually transporting one to three sodium ions for every one potassium ions. The carrier is probably a lipoprotein and it is likely that this same lipo-protein molecules acts as the enzyme

ATPase to release the energy required for transport. The sodium transport mechanism is called the sodium pump.

2.2 Membrane Potential :

One of the characteristic property of the cell membrane is its ability to maintain an unequal distribution of ions between the inside and outside of the cell. This creates a potential difference between the two areas. Because this potential derives from the membrane characteristic, it is termed the membrane potential.

The two basic means by which membrane potentials can develop are : (1) active transport of ions through the membrane thus creating an imbalance of negative and positive charges on the two sides of the membrane and (2) diffusion of ions through the membrane as a result of a concentration difference between two sides of the membrane, this also creating an imbalance of charges.

2.2.1 Membrane Potentials caused by active transport:

Fig 2.6 illustrate the process how active transport can create a membrane potential. In this figure equal concentrations of anions, which are negatively charged, are present both inside and outside the nerve fiber However the "Sodium pump" has transported some of the positively charged sodium ions to the exterior of the fiber. Thus more negatively charged anions than positively

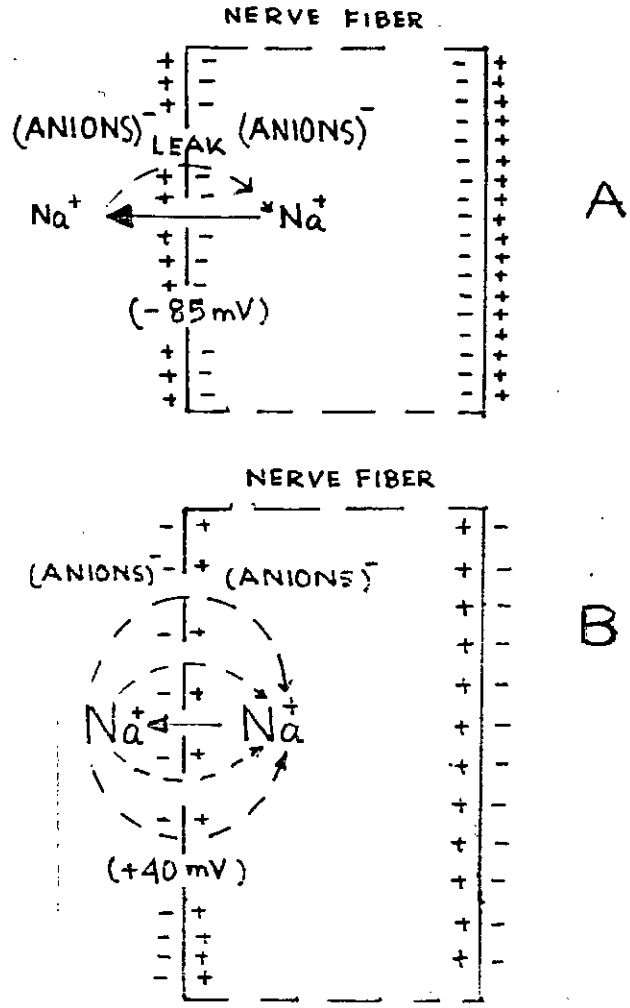


FIGURE 2.6. (A) ESTABLISMENT OF MEMBRANE POTENTIAL AS A RESULT OF ACTIVE TRANSPORT OF SODIUM IONS.

FIG(B) ESTABLISHMENT OF MEMBRANE POTENTIAL OF OPPOSITE POLARITY CAUSED BY RAPID DIFFUSION OF SODIUM IONS FROM AN AREA OF HIGH CONCENTRATION TO A AREA OF LOW CONCENTRATION.

charged sodium ions remain inside the nerve fiber causing negativity on the inside of the fiber. On the other hand, outside the fiber there are more positively charged sodium ions than negatively charged anions, thus causing positivity outside the fiber. A pump such as this, which causes the development of a membrane potential, is called an electrogenic pump.

In most nerve cells and muscle cells potassium is pumped into the cell at the same time that sodium is pumped out;usually however two to five times as much sodium as potassium is pumped.

2.2.2 Membrane Potentials caused by "Diffusion":

Fig 2.6 illustrate the nerve fiber under another condition in which the permeability of membrane to sodium has increased so much that diffusion of sodium through the membrane is now greater in comparison with the transport of sodium by active transport. Since sodium and anion concentrations are great outside the cell and slight inside, the positively charged sodium ions now move rapidly to the inside of the membrane, leaving an excess of negative charge outside, but creating an excess of positive charges inside.

When a concentration difference of ions across a membrane causes diffusion of ions through the membrane, thus creating a membrane potential, the magnitude of the potential is determined by the following equation. At body temperature($38^{o}C$) potential

is determined from the following formula :

$$\text{EMF (in millivolts)} = 61 \log_{10} \frac{\text{Concentration inside}}{\text{Concentration outside}} \quad ..(1)$$

This equation is called Nernst equation.

However, two conditions are necessary for a membrane potential to develop as a result of diffusion : (1) The membrane must be semipermeable, allowing ions of one charge to diffuse through the pores more easily than ions of opposite charge. (2) The concentration of the diffussible ions must be greater on one side of the membrane than on the other side.

When a membrane is permeable to several different ions, the diffusion potential that will develop depends on the following three factors : (1) the polarity of the electrical charge of each ion (2) the permeability of membrane (P) to each ion and (3) the concentration of the respective ions on the two sides of the membrane. Then, for ions of biological interest(namely, potassium, sodium, and chloride), in the intra- and extracellular media the following equation[1] may be used to evaluate the potential.

$$V = \frac{RT}{F} \ln \frac{P_k(C_k)_o + P_{Na}(C_{Na})_o + P_{Cl}(C_{Cl})_i}{P_k(C_k)_i + P_{Na}(C_{Na})_i + P_{Cl}(C_{Cl})_o} \quad .. \quad .. (2).$$

where P is the permeability coefficient and C denotes the concentration of each of the ions inside and outside the membrane.

The term R = kL, is known as gas constant and L is the Avogadros number and F = eL, where F is the Faraday and e is the electronic charge and k is the Boltzmann's constant.

2.2.3 Origin of the cell membrane potential:

In order to explain the origin of the cell membrane potential the following basic facts need to be understood.

(a) The nerve membrane is endowed with a sodium pump and a potassium pump, sodium being pumped to the exterior and potassium to the interrior.

(b) The resting nerve membrane is normally 50 to 100 times as permeable to potassium as to sodium. Therefore potassium diffuses with relative ease through the resting membrane, where as sodium diffuses only with difficulty.

(c) Inside the nerve fibre and large numbers of anions (negatively charged) that cannot diffuse through the nerve membrane at all or that diffuses very poorly. These anions include especially organic phosphate ions, sulphate ions and protein ions.

Now considering the above facts the formation of membrane potential is as follows:

First sodium is pumped to the outside of the fiber, while potassium is pumped to the inside. However, because two to five sodium ions are pumped out of the fiber for every potassium ion that is pumped in, more positive ions are continually pumped

out of the fiber than into it. Since most of the anions inside the fiber are nondiffusible, the negative charges remain inside the nerve fiber so that the inside of the fiber is electronegative, while the outside becomes electropositive. It is illustrated in the fig 2.7 . As progessively more sodium ions are pumped out of the nerve fiber, they begion to diffuse back into the nerve fiber because of the fact that a sodium concentration gradient develops from the outside of the fiber toward the inside and a negative membrane potential develops inside the fiber and attracts the positively charged sodium ions inward. Eventually there comes a point at which inward diffusion equals the outward pumping by the sodium pump. When this occurs, the pump has reached its maximum capability to cause net transfer of sodium ions to the outside. This occurs when sodium ion concentration inside the nerve fiber falls to about 10 mEq/l (in contrast to 142 mEq/l in extracellular fluid) and membrane potential inside the fiber falls to approximately -85 millivolts. Therefore -85 mv is also the resting potential of the nerve membrane.

2.2.4 The Action Potential:

So long as the membrane of the nerve fiber remains completely undisturbed, the membrane potential remains approximately -85 millivolts which is called the resting potential. However, any factor that suddenly increases the permeability of the membrane to sodium is likely to elicit a sequence of rapid changes in membrane potential lasting a minute fraction of a second,

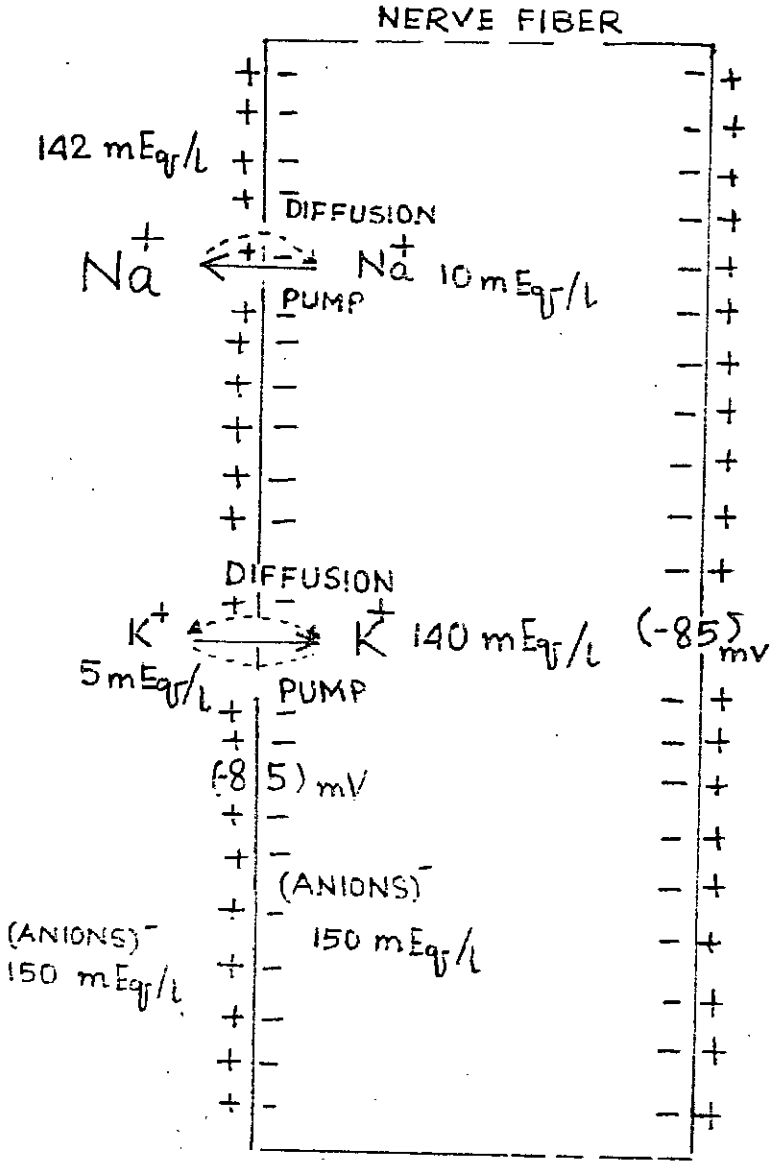


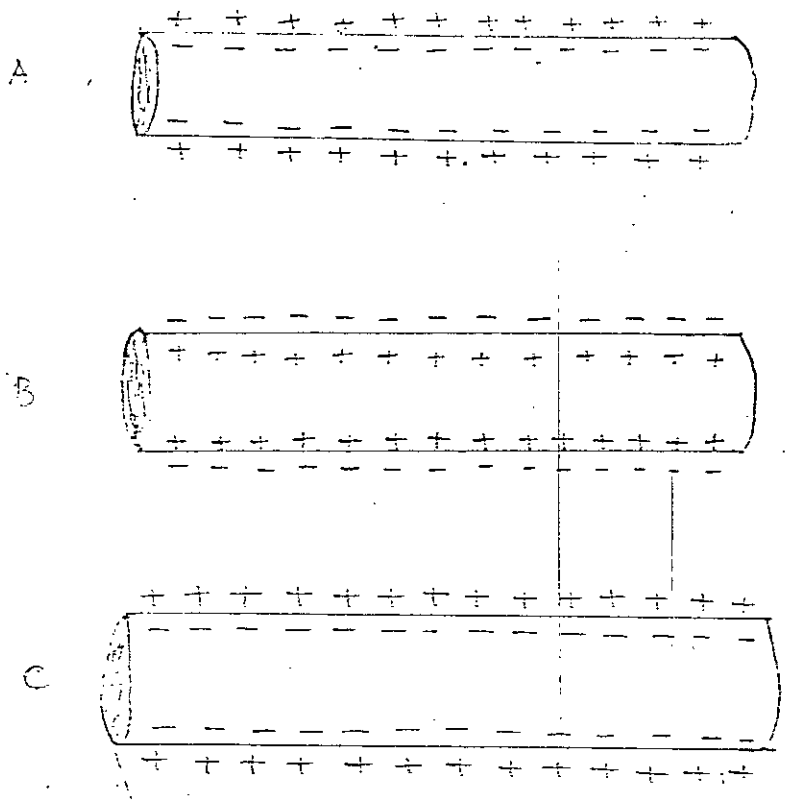
FIGURE 2.7. ESTABLISHMENT OF A MEMRANE POTENTIAL OF -85 mV IN THE NORMAL RESTING NERVE FIBER AND THE DEVELOPMENT OF CONCENTRATION DIFFERENCE OF SODIUM AND POTASSIUM IONS BETWEEN THE TWO SIDES OF THE MEMBRANE.

followed immediately there after by a return of membrane potential to its resting value. This sequence of change in potential is called the "action potential".

The factors that can elicit an action potential are electrical stimulation of membrane, application of chemicals to the membrane to cause increased permeability to sodium, mechanical damage of the membrane, heat, cold or almost any other factor that momentarily disturbs the normal resting state of the membrane.

Depolarization and Repolarization:

The action potential occurs in two separate stage called depolarization and repolarization which may be explained from the figure 2.8. Fig. 2.8a illustrate the resting state of the membrane with negativity inside and posivivity outside. When the permeability of the membrane pores to sodium ions suddenly increases, many of the sodium ions rush to the inside of the fiber, carrying enough positive charges to the inside to cause complete disappearence of the normal resting potential and usually enough positive charges accumulates actually to develop a positive state inside the fiber instead of normal negative state, as illustrate in Fig 2.8b. This positive state inside the fiber is called the reversal potential. This changes in potential is also known as depolarization.



Sequential events during action potential showing:
(A) the normal resting potential, (B) development of reversal of Potential during the depolarization, (C) re-establishment of the normal resting potential during repolarization.

(Fig 2.8)

Almost immediately after depolarization takes place, the pores of the membrane again becomes almost totally impermeable to sodium ions. Because of this the positive reversal potential inside the fiber disappears, and the normal resting membrane potential returns. This is called repolarization. (Fig 2.8c)

The action potential is caused by a sequence of changes in the membrane permeability for sodium and potassium. The first event is a tremendous increase in sodium permeability. Because of the very high concentration of sodium outside the fiber, sodium ions now diffuse rapidly to the inside, carrying positive charges and changing the inside electrical potential from negativity to positivity.

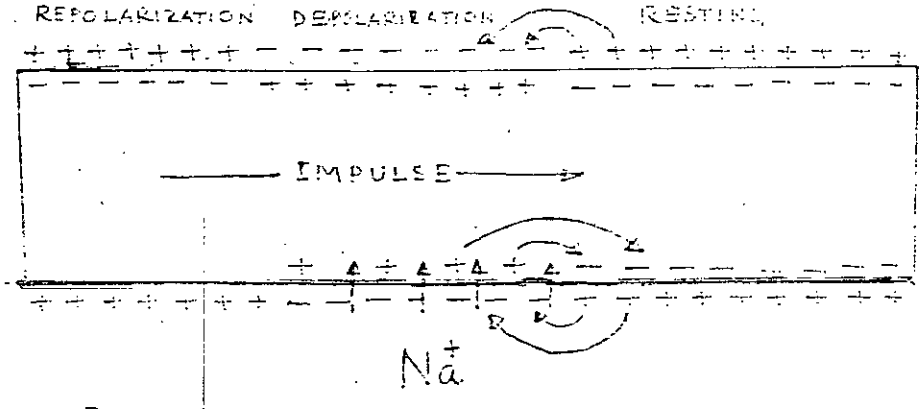
The second event is an increase in potassium permeability coupled with simultaneous decrease of the sodium permeability back to normal. This allows large quantities of positively charged potassium ions to diffuse out of the fiber, a diffusion which returns the inside membrane potential back to its negative resting level.

The third even is a decrease of the potassium permeability back to normal.

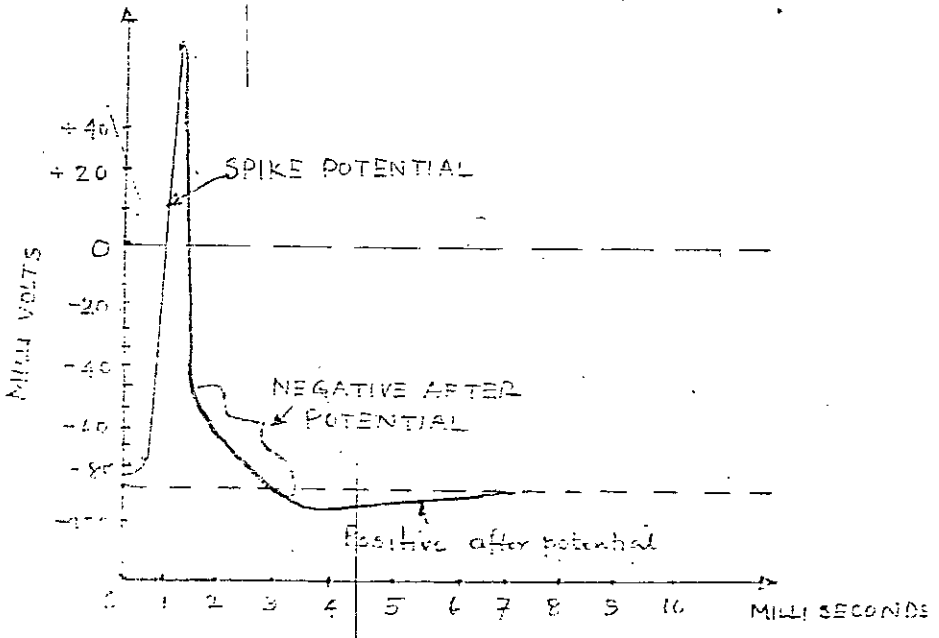
The fourth event is active transport of sodium backout of the nerve fiber and, concurrently, both active transport and diffusion of potassium ions back into the nerve fiber, thus re-establishing the original state of the fiber.

Propagation of action potential:

The concept just outlined emphasizes that to initiate an action potential there must be sufficient alteration of the membrane so as to permit the inward diffusion of sodium. If this alteration is adequate, a sequence is started which produces a brief reversal in the membrane potential. The outside of the cell becomes negative in relation to the inside. But the entire cell does not participate in these changes simultaneously. The changes first occur at the point of stimulation. In that area the outside of the membrane becomes negative while contiguous areas of the membrane are still positive (Fig 2.9). The positive charges are therefore attracted to the negative ones. This area of the negative charges is said to be a sink into which the positive charges flow. The removal of positive charges reduces the potential difference of the two sides of the membrane at that point. That is the resting potential at that point is less negative; therefore, permeability increases and sodium quickly moves in. This part of the membrane then undergoes polarity reversal and the outside becomes negative. The sequence is now reday to be repeated. Accordingly there results a progressive series of action potentials which move in the both direction along the neurons from the point of stimulation. The action potential so generated constitute what is termed the impulse.



PROPAGATION OF AN IMPULSE
(Fig 2.9)



An idealized action potential, showing the initial spike followed by a negative after potential and a positive after potential.

Fig 2.10

Direction of Propagation:

It is now obvious that an excitable membrane has no single direction of propagation, but that the impulse can travel in both directions away from the stimulus and even along all the branches of a nerve fiber-until the entire membrane has become depolarized.

The All-or-Nothing Principle:

Once an action potential has been elicited at any point on the membrane of a normal fiber, the depolarization process will travel over the entire membrane. This is called the all or nothing principle and it applies to all normal excitable tissues. Occasionally when a fiber is in an abnormal state the impulse will reach a point on the membrane at which the action potential does not generate sufficient voltage to stimulate the adjacent area of the membrane when this occurs the spread of depolarization stops. Therefore, for normal propagation of an impulse to occur, the ratio of action potential to the 'threshold' for excitation, called safety factor must be greater than unity.

Propagation of Repolarization:

Repolarization normally occurs first at the point of original stimulus and then progressively along the membrane, moving in the same direction that depolarization had previously spread.

The Spike Potential and the After potential:

The figure (2.10) illustrates an action potential with a much slower time scale.

The Spike Potential:

The initial very large change in membrane potential shown in figure is called the spike potential. The duration of this spike potential is about 0.4 millisecond. The spike potential is also known as nerve impulse.

The negative after-potential:

At the termination of the spike potential, the membrane potential fails to return all the way to its resting level for another few millisconds. This is called negative after potential.

The Positive after-potential:

Once the membrane potential has turned to its resting value, it becomes a little more negative than its normal resting value; this excess negativity is called the positive after potential.

Plateau in the Action Potential:

In some instances the excitable membrane does not repolarize immediately after depolarization, but, instead, the potential remains on a Plateau near the spike sometimes for many

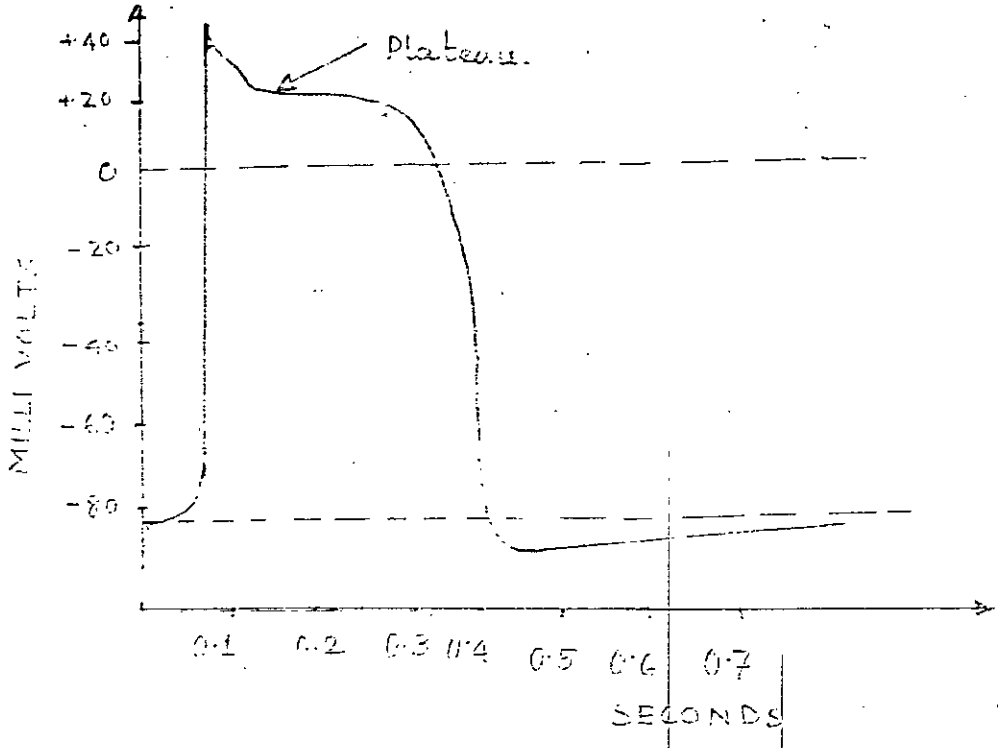
milliseconds before repolarization begins. Such plateau is illustrated in the fig. 2.11.

Special Aspect of Impulse Transmission in nerve:

So far we have discussed about the propagation of action potential is valid for unmyelinated nerve fiber. But if there is myelin the situation is some what different. The myeline acts as insulator but this insulation is broken at the nodes of Ranvier. Accordingly the positive charges flow from one node over the myelin to the next node when that node becomes negative. Because the charge leap from node to node, this form of propagation is termed saltatory propagation, saltatory means dancing.(Fig. 2.12).

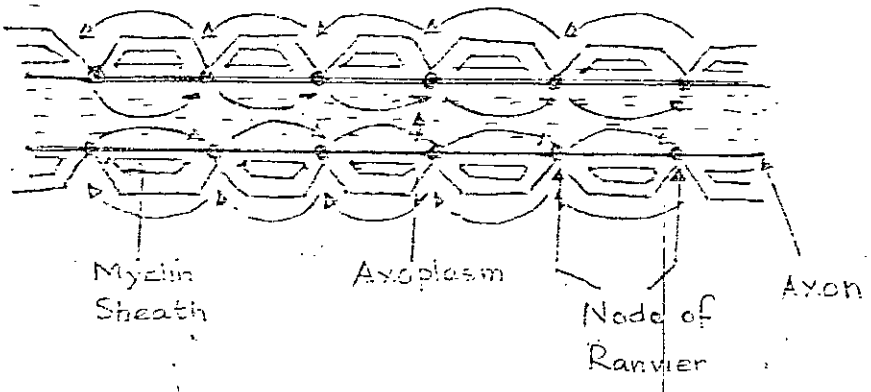
Excitability Threshold:

During the period of time required for the upswing of the spike potential, the cell will not respond to another stimulus no matter how intense. This is an "absolute refractory period". The duration is of about 0.4mSec in mammalian nerve cell. It is followed by "relative refractory period" during which an above threshold stimulus will evoke a response. There is then a sudden reversal of excitability. During the period of negative after-potential the excitability threshold is lower than normal so that, at this time, the nerve cell may be more easily activated. Another reversal occurs during the positive after-potential period in which the excitability threshold is



An action potential from a purkinje fiber of the heart showing "plateau".

Fig 2.11



Saltatory conduction along a myelinated axon.

Fig · 2·12

once agains higher than normal.

The absolute refractory period limits the number of impulses that can be propagated per unit time, that is, the frequency of response. If the nerve cell has an absolute refractory period of 0.4 mSec then the maximum impulse frequency can not exceed 2500/Sec.

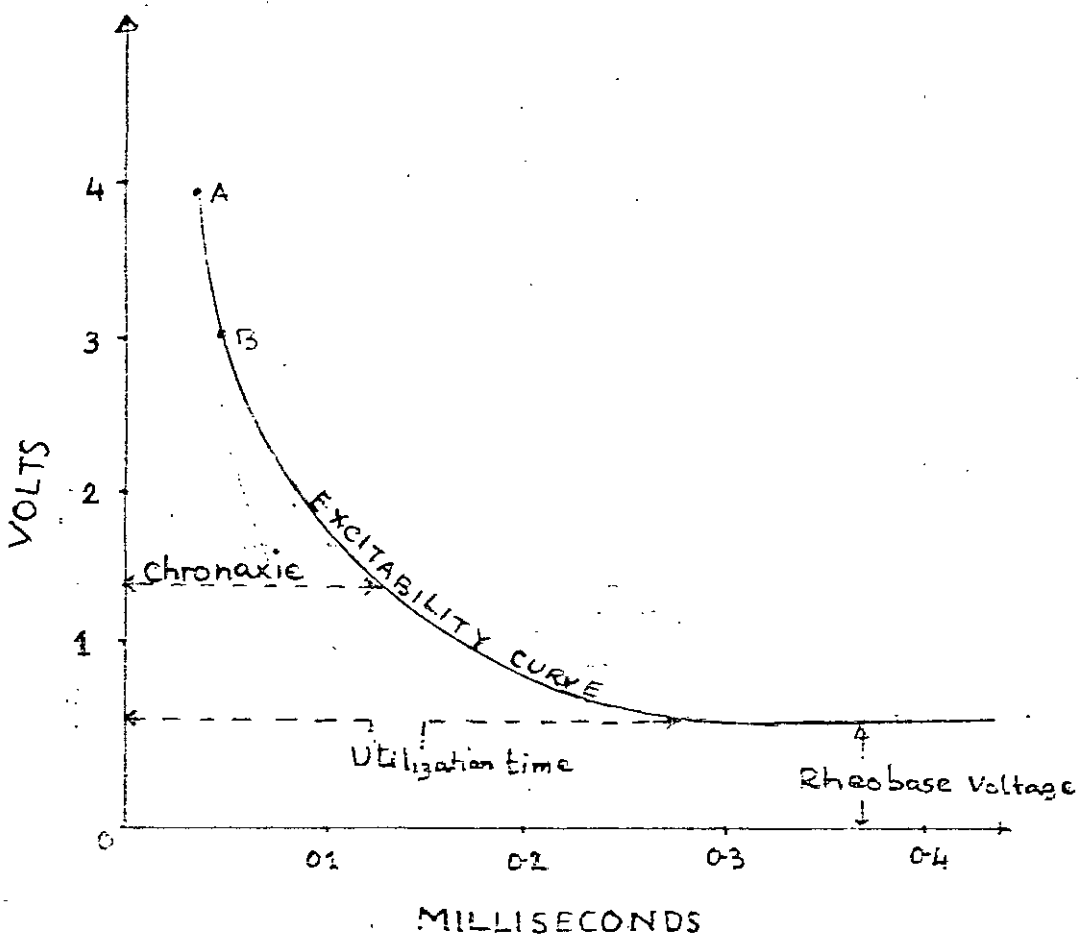
Velocity of Propagation:

The velocity at which a nerve cell propagates an impulse is a function of its diameter. The fiber of greatest diameter have been found to propagate impulses at a velocity of up to 150 m/Sec. Very small mammalian nerve cells have a propagation velocity of less than 1m/sec. In mammalian cardiac muscle the impulse propagates a approximately 0.5m/Sec.

Chronaxie:

The chronaxie is a measure of the irritability of the cell. Irritability means the ability to respond to a change in the environment. Typical responses are the propagation of an impulse in a nerve, contraction of a muscle, or secretion of a gland. The chronaxie can thus be used to compare the responsiveness of various cells and it proves more satisfactory to use action potential as an indication of response.

In order to calculate the chronaxie, a excitability curve is constructed for a nerve fiber as shown in the figure 2.13.



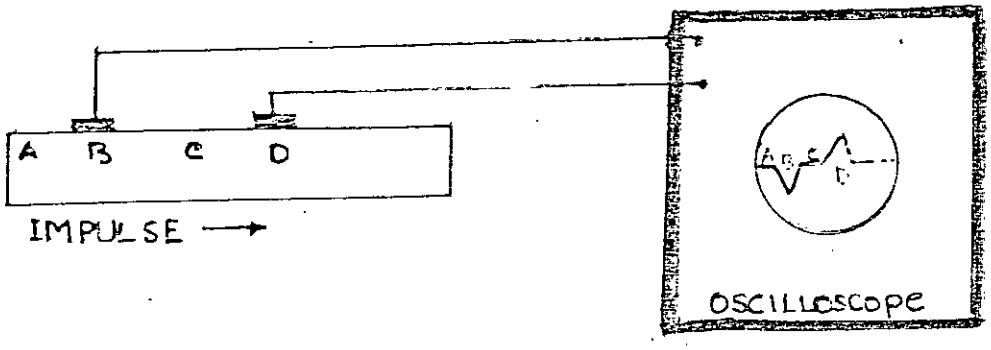
Excitability Curve of a nerve fiber.

Fig 2·13.

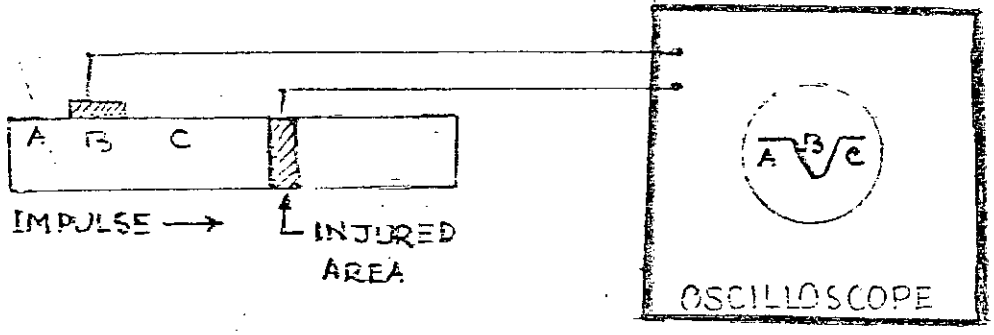
To obtain the curve a high voltage stimulus (4 Volt, in this instance) is first applied to the fiber and the minimum duration of stimulus required to excite the fiber is found. The voltage and minimal time are plotted as point A. Then a stimulus of 3 volt is applied and the minimal time required is again determined , the results are plotted as point B. The same is repleated at 2 volts. 1 volts, 0.5 volts and so forth until the least voltage possible at which the membrane is stimulated, has been reached. On connection of these points, the excitability curve is determined. It can be seen that the weaker the stimulus, the longer it must act on the cell in order to evoke response. The least possible voltage at which the fiber will fire is called the "rheobase" and the time required for this least voltage to stimulate the fiber is called the "utilization time". Then, if the voltage is increased to twice the rheobase voltage the time required to stimulate the fiber is the chronaxie.

Diphasic and Monophasic Action Potential:

If two recording electrodes are placed upon the surface of a nerve cell (as shown in the fig. 2.14) which is then stimulated to propagate an impulse, the resulting recording will be a diphasic action potential. That is to say, there is a deflection in one direction when the area under the one electrode is depolarized, and then there is a deflection in the opposite direction when the area under the 2nd electrode becomes depolarized. By the time the depolarization under the second



A diphasic action potential recording.



A monophasic action potential recording.

RECORDING OF ACTION POTENTIAL

Fig 2.14

electrode occurs, the area under the first is repolarized, resulting in a deflection in the opposite direction. However, when the impulse is between the electrodes, both areas have the same polarity and thus there is no deflection. It is often desired to eliminate the second deflection and the potential so recorded is known as monophasie action potential. This can be achieved in at least 3 ways : (1) With one electrode inside and the other on the outside of the membrane, a single deflection from the resting position will result when the membrane is depolarized; (2) With the two electrodes on the surface of the nerve and an area between the two electrodes blocked by an anesthetic or by injury and(3) With one of the electrodes on an injured area.

2.2.5 Types of Muscle and Their Action Potential:

There are three types of muscle such as skeletal muscle, smooth muscle and cardiac muscle. A muscle basically consists of numerous individual fiber (cell) that are arranged parallel to one another. In higher animal forms, the ability to change position is solely the function skeletal muscle. But muscle is also vital for other purpose. Cardiac muscle serves to pump the blood through the circulatory system. Smooth muscle is essential to bladder contaction, to the regulation of the size of the pupil of the eye, to activity of the gastrointestinal tract, as well as to many visceral function.

Skeletal Muscle:

Skeletal muscle is generally excited by direct stimulation; it rarely contracts spontaneonsly as smooth and cardiac muscle do. In the intact organism excitation of skeletal muscle is under the control of the nervous system. In man, for example, hundreds of nerve fibers innervate each muscle. Usually the nerve fiber arborize into many branches, each of which terminates on an individual muscle fiber. All of the muscle fibers thus controlled by a single neuron (nerve cell)constitute one motor unit. There is no protoplasmic continuity between the neuron and the muscle fiber. There remains a potential space between the two cell membranes called the myoneural junction.

When the impulse propagated by the neuron reaches the end of the motor neuron, acctylcholine is liberated which alters the end-plate potential so as to give rise to an action potential. The fig (2.15) shows the typical response of skeletal muscle. Two significant event occurs: there is an action potential, and the muscle cantracts. It is to be noted that the action potential occurs first and only about 2mSec after the muscle is stimulated does it begin to contract. This delay is termed the "Latent Period".

Cardiac Muscle:

The most characteristic aspect of cardiac muscle is that it possesses inherent rhythmicity. A detail discussion is

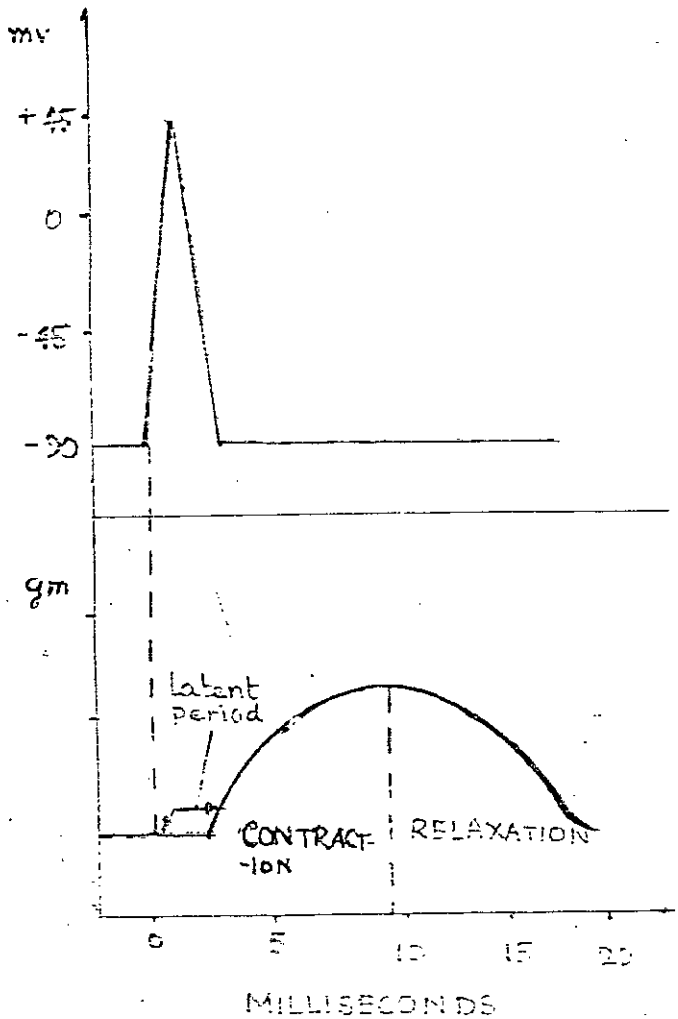


Fig-2.15 Response of a skeletal muscle to a single stimulus was applied at zero time. The resting potential is -90 mv.

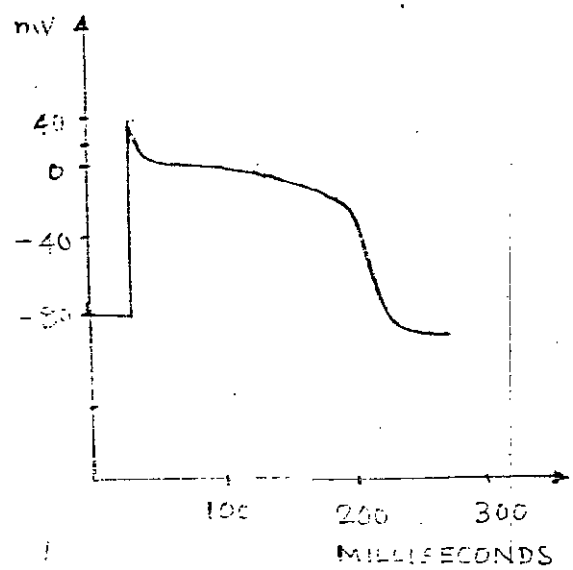


Fig-2.16
Cardiac muscle action potential

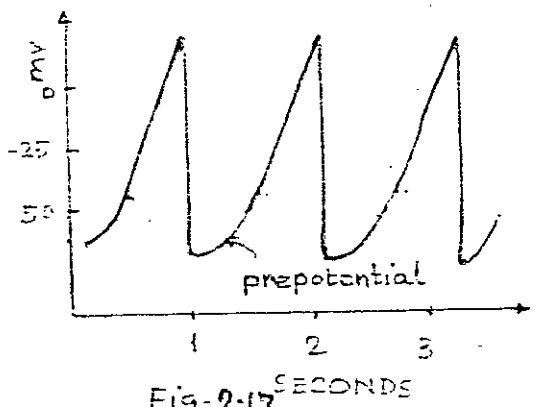


Fig-2.17 Smooth muscle action potential.

Action Potential in different types of muscle.

presented in next chapter.

The action potential associated with the contraction of cardiac muscle differs considerably from those recorded from nerve and skeletal muscle. In figure 2.16. the action potential recorded from cardiac muscle is seen to be very prolonged. During activation there is a typical rapid upswing of the spike, an over shoot, then the process of repolarization begins.

Smooth Muscle:

The physiology of the smooth muscle is very inadequately understood. The action potential associated with contraction of smooth muscle varies from moment to moment and muscle to muscle. In some smooth muscle the action potential resembles that of a skeletal muscle in that there is rapid repolarization. In others, the action potential more nearly resembles that of a cardiac muscle. In addition resting potential also varies greatly. Quite often a progressive change in resting potential is noted between contractions. In figure (2.17) it is to be noted that followng repolarization there is a slow depolarization which is culminated by the onset of the action potential. This preliminary slow depolarization is termed as "prepotential".

Smooth muscle contracts spontaneously. Prepotentials appear to be characteristic of all muscle that contracts spontaneously.

For example, a specific area of the heart has this property; the part which initiates the beat. Accordingly, it is referred to as the "pacemaker". If action-potentials are recorded from this area, typical prepotentials are seen to be present, where as action potentials from other areas of the heart donot show them.

Chapter 3

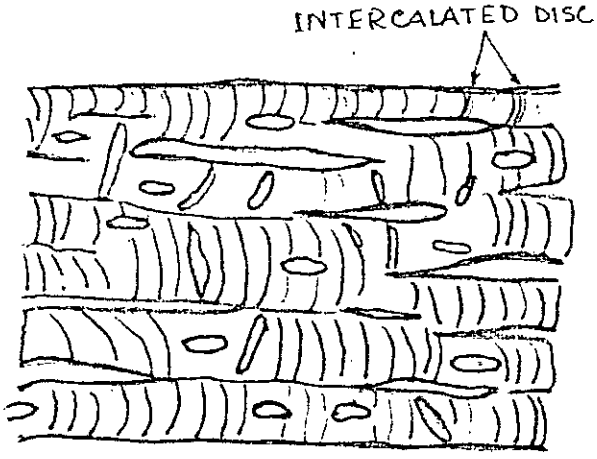
HEART MUSCLE; HEART AS A PUMP

The heart is a pulsatile, four chamber pump composed of two atria and two ventricles. The atria functions principally as entryways to the ventricles, but they also pump weakly to help move the blood on through the atria into ventricles. The ventricles supply the main force that propels blood through the lungs and through the peripherial circulatory system.

3.1.1 Physiology-of Cardiac Muscle:

The heart is composed of three major types of cardiac muscle. Normally a muscle consists of numerous individual fibers (cells) that are arranged parallel to one another. The types of cardiac muscles are : atrial muscle, ventricular muscle and specialized excitatory and conductive muscle fibers. The atrial and ventricular muscle contract in same manner as skeletal muscle fibers. On the other hand the specialized excitatory and conductive fibers contract only feebly because they contain few contractile fibrils; instead they provide an excitatory system for heart and a transmission system for rapid conduction of impulses through out the heart.

Cardiac muscle is similar structurally, in same ways, to skeletal muscle. For years cardiac muscle was described as being a syncytium. That is protoplasmic continuty was thought to



THE SYNCYTIAL NATURE
OF CARDIAC MUSCLE
Fig - 3.1

exist between the individual fibers. Electron microscopy, however, fails to support this conclusion. Practically all of the fibers are seen to be surrounded by a membrane and seemingly well insulated from contiguous ones. It remains a fact that even though cardiac muscle not be a syncytium, it contracts as one expects a syncytal muscle to contract. For example, in skeletal muscle individual fibers may contract while neighboring ones remain quiescent. In cardiac muscle this does not normally occur. Stimulation of any part of cardiac muscle generally causes all of the fibers to contract. Thus graded response is not possible. In skeletal muscle the individual fibers exhibit all -or- none principle; in cardiac muscle the entire muscle responds in this way.

Characteristic of cardiac muscle is the presence of intercalated discs (Fig. 3.1). These are thickened and tortuous membrane which usually cross the muscle in a stepwise manner. It is thought likely that intracellular current is transferred from one cardiac muscle to another at the intercalated discs. Thus, even though cardiac muscle is not now considered to be a syncytium, it probably acts like one because of ease of the spread of current from cell to cell at the site of the discs.

So the heart may be thought to be composed of two seperate functional syncytiums, the atrial syncytium and ventricular syscytium. These are separated from each other by the fibrous

tissue surrounding the valvular rings, but an action potential can be conducted from atrial syncytium into ventricular syncytium by way of a specialized conductive system, the A-V bundle.

3.1.2 All or Nothing Principle as Applied to Heart:

The stimulation of any single atrial muscle fiber causes the action potential to travel over the entire artrial mass and similarly, stimulation of any single ventricular fiber causes excitation of the entire ventricular muscle mass. If A-V bundle is intact, the action potential passes also from the atria to the ventricles, but because the cardiac muscle fibers are interconnected with each other, the all-or none-principle applies to the entire functional syncytium of the heart rather than to single fiber.

3.1.3 Action Potential in Cardiac Muscle:

The resting membrane potential of a normal cardiac muscle is approximately-80 to-85 mV and approximately -90 to -100 millivolt in the specialized conductive fibers, the Purkinje fibers.

The action potential recorded in the cardiac muscle, shown in Fig.3.2 is 90 to 105 millivot which means that the membrane potential rises from its normally very negative value to a slightly positive value of about +20 mV. Because of its change of potential from negative to positive, the positive portion is called the reversal potential.

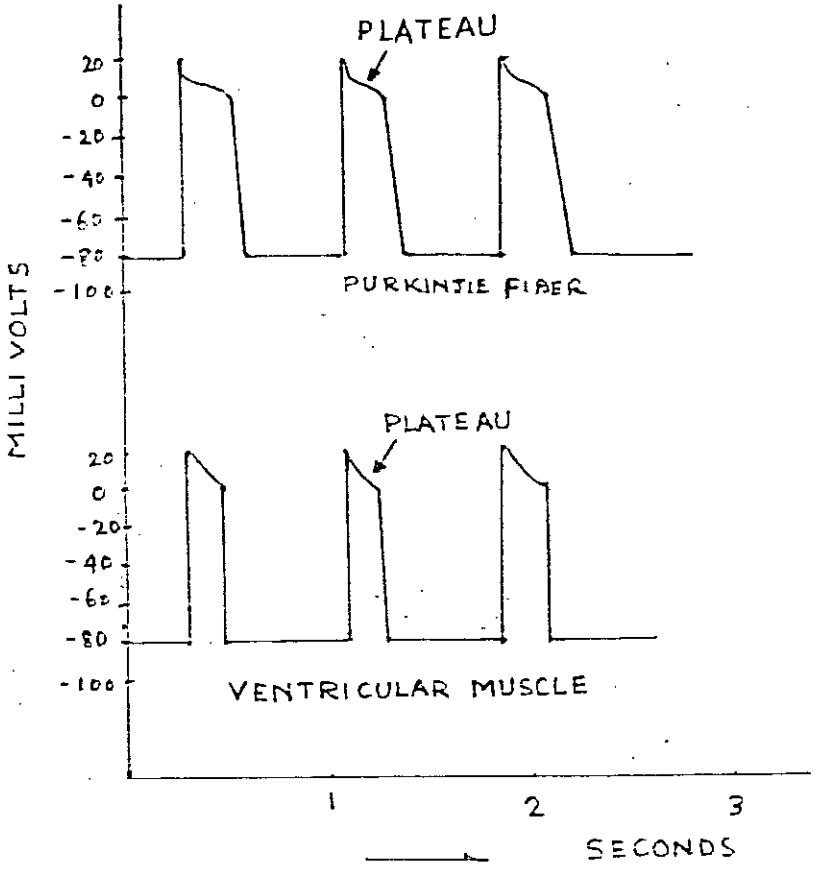


FIG 3.2

RHYTHMIC ACTION POTENTIAL FROM PURKINJIE FIBER AND VENTRICULAR MUSCLE

Cardiac muscle has a peculiar type of action potential. After initial spike the membrane remains depolarized for 0.15 to 0.3 Seconds exhibiting a plateau, followed at the end of the plateau by abrupt repolarization. The presence of this plateau in the action potential causes the action potential to last 20 to 50 times as long in cardiac muscle as in skeletal muscle and causes a correspondingly increased period of contraction.

3.1.4 Contraction of Cardiac Muscle:

When the action potential travels over the muscle, it also causes flow of action potential to the interior of the fiber by the way of T tubules. This T tubules action potentials in turn, are believe to cause release of calcium ions which diffuses rapidly into the myo-fibrils and there catalyzed the chemical reactions that promote sliding of actin and myosin filaments along each other : thus inturn produces the muscle contraction. Immediately after the action potential is over the calcium ions are transported back into the longitudinal tubules of the sarcoplasmic reticulum, so that within few milliseconds the density of the released calcium ions around the myofibrils falls below that needed to maintain contraction, as a result, the muscle relaxes.

3.1.5 The Cardiac Cycle:

The period from the end of one heart contraction to the end of the next is called the cardiac cycle. The cardiac cycle

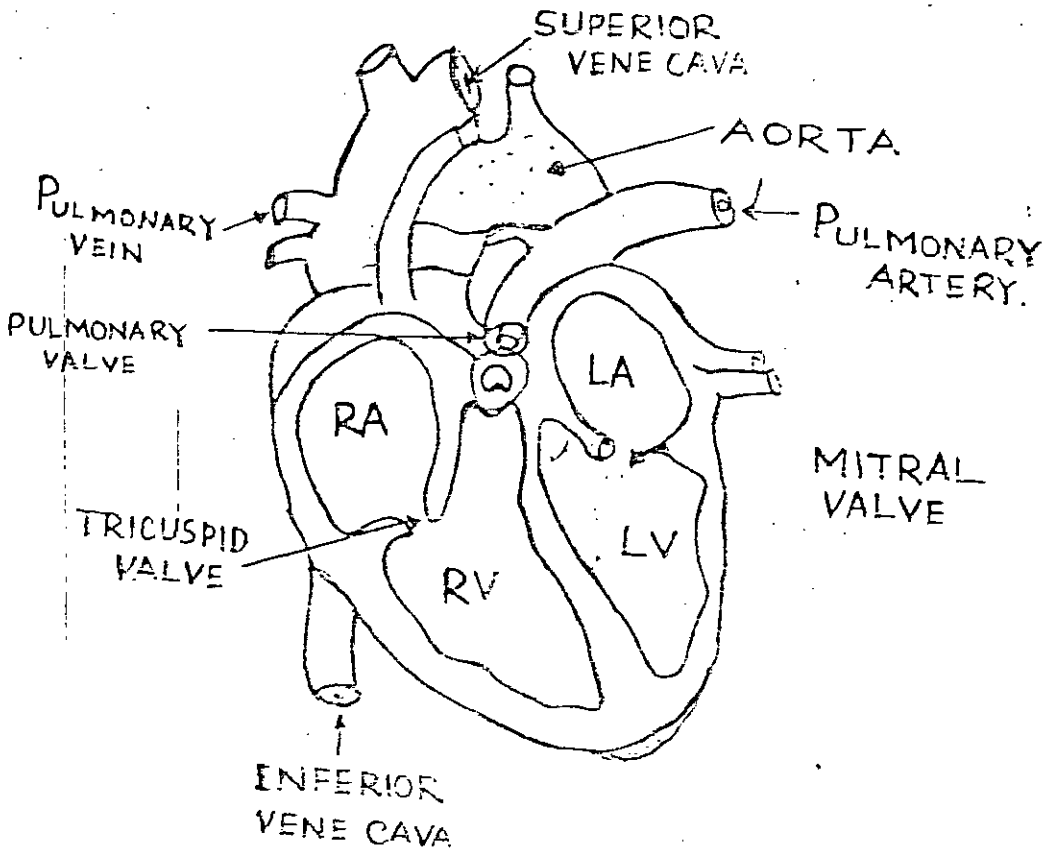


FIG. 3.3. CROSS-SECTION OF HUMAN HEART.

consists of a period of relaxation called diastole followed by a period of contraction called systole.

3.2 Rhythmic Excitation of the Heart:

3.2.1 Mechanical Activity of the Heart:

Figure(3.3) illustrates the major anatomical parts of interest of the human heart. Blood is returned to the heart through the inferior and superior venae cavae where it enters the right atria (RA). The blood then flows into the right ventricle (RV) from which it is forced into the lungs, oxygenated, and then returned to the left atria (LA). From there, it enters the left ventricle (LV), where-upon the fresh blood is then forced back into the system through the aorta.

The physical activity of the heart is rhythemic and consists of the following recognized intervals:(1)

1. Diastasis: This corresponds to the end of the main pressure pulse in the system. The atrioventricular valves (tricuspid & mitral valves) have been open for sometime and all chembers have filled with blood. This condition may be thought of as the initial or resting state of the heart.

2. Atrial Contraction: Diastasis ends with the beginning of atrial contraction. This causes only a relatively slight rise in intra-atrial pressure. The ventricular volume and pressure increases as a result of the entrance of blood from the atria.

3. Ventricular Isometric contraction: The first phase for ventricular contraction corresponds to an increase in pressure but not in volume. The increase in pressure closes the AV valves, hence preventing the initial change in volume. At the outset of ventricular contraction, the pressure in the discharge vessel of the right ventricle (pulmonary artery) is 7 mm Hg, while that in the aorta (the artery carrying blood from left ventricle) is 80 mm. The aortic and pulmonary valves remain closed until a sufficient pressure is built up.

4. Ventricular Ejection: When the ventricular pressure builds up to a value which exceeds that in the aorta, the aortic valve opens and the left ventricle rapidly discharges blood through the aorta into the system. A peak pressure of about 125 mm Hg. is reached. After the major portion of ejection, the ventricular muscle fibers are shortened and can no longer contract forcefully. The ventricular and aortic pressures begins to drop.

5. Isometric Relaxation: When ventricular pressure falls sufficiently the aortic and pulmonary pressures exceed that within the chembers and the aortic and pulmonary valves close. At this point ejection ceases. As the ventricle continues to relax, the pressure falls still further; but while the pressure exceeds that in the atria no exit is provided for tapped blood, so that the relaxation is characterized as isometric.

6. Ventricular Filling: When the ventricular pressure drops below that of atria, the AV valves opens and rapid ventricular filling begins. This phase is followed by a slowing down of the filling as the ventricle reaches a maximum diastolic size. This is the period of diastasis and this is the completion of one cycle followed by the next cycle upon the initiation of atrial systole.

3.2.2 Stroke Volume Output of the Heart:

The stroke volume output of the heart is the quantity of blood pumped from each ventricle with each heart beat. Normally it is about 70 ml, but under conditions compatible with life it can decrease to as little as few ml. per beat and can increase to about 140 ml. per beat in the normal heart and to over 200 ml. per beat in persons with very large heart, such as in some athletes.

3.2.3 Work output of the Heart:

The work output of the heart is the amount of energy that the heart converts to work while pumping blood into the arteries. This is in two forms; First; by far the major proportion is used to move the blood from the low pressure veins to high pressure arteries. This is potential energy of pressure. Second, a minor proportion of energy is used to accelarate the blood to its velocity of ejection through the aortic and pulmonary valves. This is Kinetic energy of blood flow.

Energy Expended to create Potential Energy of Pressure:

The work performed by the left ventricle to raise the pressure of the blood during each heart beat is equal to "stroke volume output" X (left ventricular mean ejection pressure minus left atrial pressure). Like wise the work performed by the right ventricle to raise the pressure of the blood is equal to (stroke volume output") X(right ventricular mean ejection Pressure minus right atrial pressure). When pressure is expressed in dyne per square centimeter and stroke volume output in milliliters, the work output is in ergs. Right ventricular output is usually about one seventh the work out put of the left ventricle because of the difference in systolic pressure against which the two ventricle must pump.

The kinetic Energy of blood flow : The work output of each ventricle required to create kinetic energy of blood flow is proportional to the mass of the blood ejected times, the square of the velocity of ejection. That is,

$$\text{Kinetic Energy} = \frac{mv^2}{2}$$

when the mass is expressed in grams of blood ejected and the velocity in centrimeter per second the work output is in ergs.

Ordinarily, the work output of the left ventricle required to create Kinetic energy of blood flow is about 1 percent of the total workoutput of the ventricle,. Most of this energy is required

to cause the rapid accelaration of blood during first quarter of systole.

3.2.4 Electrical Activity of the Heart:

The heart is endowed with a special system (a) for generating rhythmical impulses to cause rhythmical contraction of the heart muscle and for (b) conducting these impulses throughout the heart (Fig. 3.4). Major share of the ills of the heart is based on abnormalities of this special excitatory and conductive system.

The adult human heart normally contracts at a rhythmic rate of about 72 beats per minute. The figure shows the special excitatory and conductive system of the heart that controls these cardiac contractions. The figure shows,

(a) the SA node in which the normal rhythmic self excitatory impulses is generated.

(b) the AV node in which the impulse from the atria is delayed before passing into the ventricle.

(c) the AV bundle, which conducts the impulse from atria into the ventricle.

(d) the left bundles and right bundles of Purkinje fibers which conduct the cardiac impulse to all parts of the ventricles.

The mechanical activity described before is accompanied by electrical activity in the heart. This activity begins in the sino-atrial (S-A)node, which is consequently known as the pace maker. Cells in this region have the characteristic that the

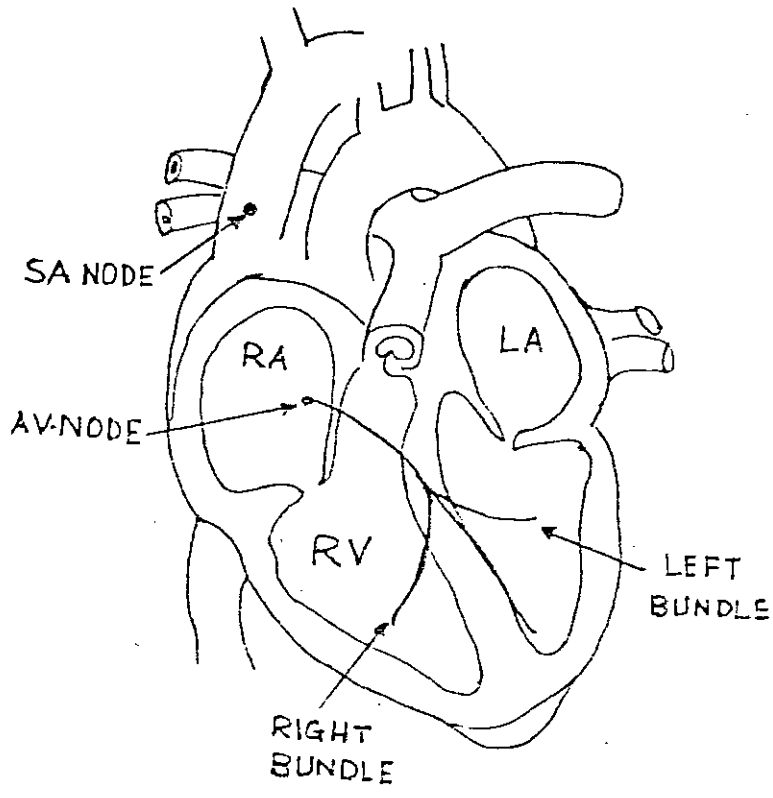


FIG. 3.4 SCHEMATIC DIAGRAM SHOWING SPECIALIZED EXCITATORY AND CONDUCTIVE SYSTEM OF HEART.

resting potential is not maintained but continually diminishes until the cell 'fires'. Upon recovery, the process is repeated so that a periodic excitation results.

As noted heart rate is basically determined by the pacemaker activity in S.A. node, which operates in a sense, as a free running multivibrator. The rate, however, is modified by the competing effects of the parasympathetic and sympathetic nerves. The effect of the vagus nerve is one of the slowing the heart, while the sympthetic nerves increase the rate. Cutting both nerves to heart results in an increased rate, so that vagal effects appear to be dominant under resting conditions. It should be noted that the cardiac output depends on both heart rate and stroke volume, so that an increase in heart rate, for example, does not necessarily results in an increased output of heart.

The activity initiated by the SA node spreads through the muscle of the atria at about 1m/Sec. For the human, about 80 mSec is required for the complete activation of the atria. Toward the end of this period, the electrical activity reaches the AV node, which is the sole mascular connection between atria and ventricles. The propagation in the AV node is very slow, about 0.1 m/sec. upon passage through this node, the excitation travels at about 2 m/Sec. through the specialized right and left bundles and then through the arborized Purkinje fibers. This system serves to initiate electrical activity in the ventricular musculature. An illustration of the system is given in schematic diagram.

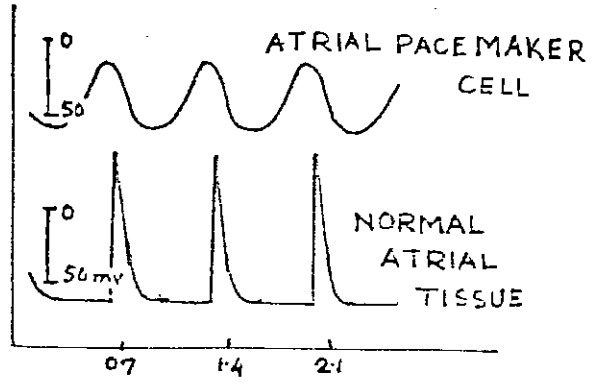


FIG 3.5 ACTIVITY OF A SINGLE ATRIAL PACEMAKER AND A A SINGAL CELL OF ATRIAL TISSUE.

The heart muscle (myocardium) behaves in many ways as if it were a single cell. Excitations spreads at about 0.5 m/Sec. and causes mechanical contraction in an efficient synchronous way. Cardiac muscle is divided into its many elements by vertical and horizontal double membranes. The latter are actually irregular tongulike process. These boundaries they from between muscle "domains" are known as interealted disks, and constitute the basis for discarding the concept of the syncytum in the anatomical sense.

Studies of heart excitation have been carried out in an effort to elucidate the pathways of activation. In general these have involved insertion of the bipolar electrodes into the beating heart of a dog and measuring the difference of potential. The result in general monophasic wave. This is interpreted as arising from the passage of a wave (double layer) of activity, and the time of passage at the midpoint of the electrode pair is the time when the signal reaches its peak. Through measurement at many points throughtout the heart and with ECG (Electrocardiogram) as a time reference, one can establish the location of the activation wave fronts at successive instants of time. It is seen that the activation pattern is fairly complex even for the normal heart.

Chapter -4

Electrocardiogram

4.1. The Normal Electrocardiogram:

As the depolarization wave or Cardiac impulse passes through the heart, electrical currents spread into the tissues surrounding the heart and a small portion of these spreads all the way to the surface of the body. If the electrodes are placed on the body on opposite sides of the heart, the electrical potential generated by the heart can be recorded, the recording is known as an electrocardiogram. A normal electro-cardiogram is illustrated in fig. (4.1).

4.2 Characteristic of Normal Electrocardiogram:

The naormal electrocardiogram is composed of a P wave, a QRS complex and a T wave. The QRS complex is actually three separate waves, the Q wave, the R wave, and the S wave illustrated in fig.(4.2).

The remarks concerning the tracing are:-(16)

1. The P wave corresponds to atrial activity.
2. The QRS (Complex) is the result of ventricular activity.
3. The T wave corresponds to ventricular repolarization. Atrial repolarization is masked by the QRS.
4. PR interval is a measure of AV conduction time, and disorders of the conduction are related to this interval.
5. The base line is established by the TP segment of the wave. The relative level of ST segment is an important diagonstic measure. Normal ST segments are at the base line, while

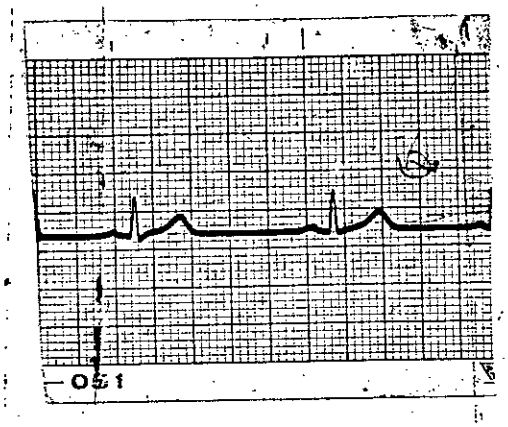


FIG. 4·1 NORMAL ELECTROCARDIOGRAM

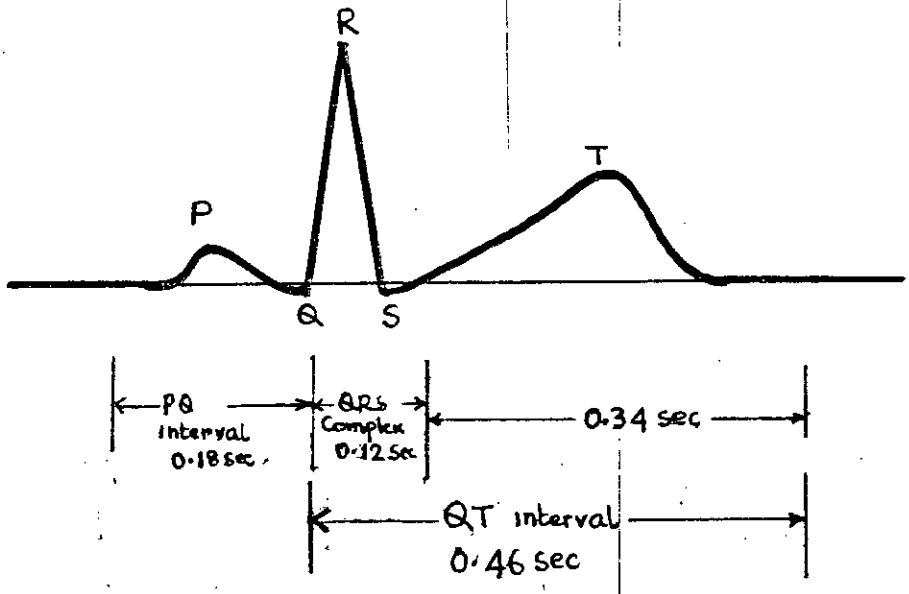


FIG. 4·2. SIGNIFICANT FEATURES OF STANDARD ELECTROCARDIOGRAM.

coronary insufficiency results in a depressed segment.

6. The QT interval gives the total duration of the ventricular systole. It should be less than half the preceeding RR interval.

7. The T wave is caused by the currents generated as the ventricles recover from the state of depolarization.

4.3. Voltage and time calibration of the Electrocardiogram:

All the recordings of electrocardiograms are made with appropriate calibration lines on recording paper. As illustrated in fig. (4.1). The calibration lines are so arranged that 10 small divisions in the vertical direction in the standard electrocardiogram represents 1 millivolt, with positivity in the upward direction and negativity in the down ward direction. Each inch or 2.5 cm in horizontal direction is 1 second, and each inch inturn is broken into 5 segments by dark vertical lines; The interval between each dark vertical lines represents 0.2 seconds. These intervals are then broken into five smaller intervals by thin lines and each of these represents 0.04 second.

Normal voltages in the Electrocardiogram:

The voltages of the waves in the normal electrocardiogram depend on the manner in which electrodes are applied to the surface of the body. When one electrode is placed directly over the heart and the second electrode is placed else where on the body, the voltage of the QRS complex may be as much as 3 to 4 millivolts

Even this voltage is very small in comparison with monophasic action potential 120mv. recorded directly from heart muscle. When the electrocardiograms are recorded from electrodes on the two arms or one arm and one leg, the voltage of QRS complex is approximately 1 mV from the top of R wave to the bottom of S wave, the voltage of the P wave between 0.1 and 0.3 mV; and that of the T wave between 0.2 and 0.3 mV.

The P-Q or P-R and Q-T Interval:

The duration of time between the beginning of the P wave a and beginning of the QRS wave is the interval between the contraction of atrium and the beginning of the contraction of the ventricle. This period of time is called PQ interval. The normal PQ interval is approximately 0.18 Second. This interval is sometimes also called the P-R interval because Q wave is frequently absent.

Contraction of ventricles lasts essentially between the beginning of the Q wave and the end of the Twave. This interval of time is called the QT interval and oridinarily is approximately 0.46 Seconds.

Rate of the Heart as determined from Electrocardiogram:

The rate of heart-beat can be determined easily from electrocardiogram. The time interval between two successive beats is the reciprocal of the heart rate.

Usually pen recorder and Oscilloscope may be used for recording the electrocardiogram.

4.4 Electrocardigraphic Leads:

Usually different electrodes are placed on different point on the body surface for the recording of electrocardiograms. This arrangement of electrodes for recording or electrocardiogram are called electrocardiographic leads. The different types of leads are discussed below.

The Three Standard limb leads:

In earliest work in electrocardiography Einthoven developed a system of lead (electrode) placement at the extremities of the body on the assumption that this would enhance the validity of the so called idipole heart model. This system of leads are called standard or limbleads. This system constitutes the most common climical system.

We define the 'Standard' leads, V_I, V_{II}, V_{III} as follows in (fig. 4.3a.)

$$V_I = V_{ab} \quad \text{(lead 1)} \qquad \ldots (1)$$

$$V_{II} = V_{cb} \quad \text{(lead 2)} \qquad \ldots (2)$$

$$V_{III} = V_{ca} \quad \text{(lead 3)} \qquad \ldots (3)$$

In the above 'a' corresponds to the wrist of the arm (LA), b to the wrist of the right arm (RA) and to the lower portion of the left leg.

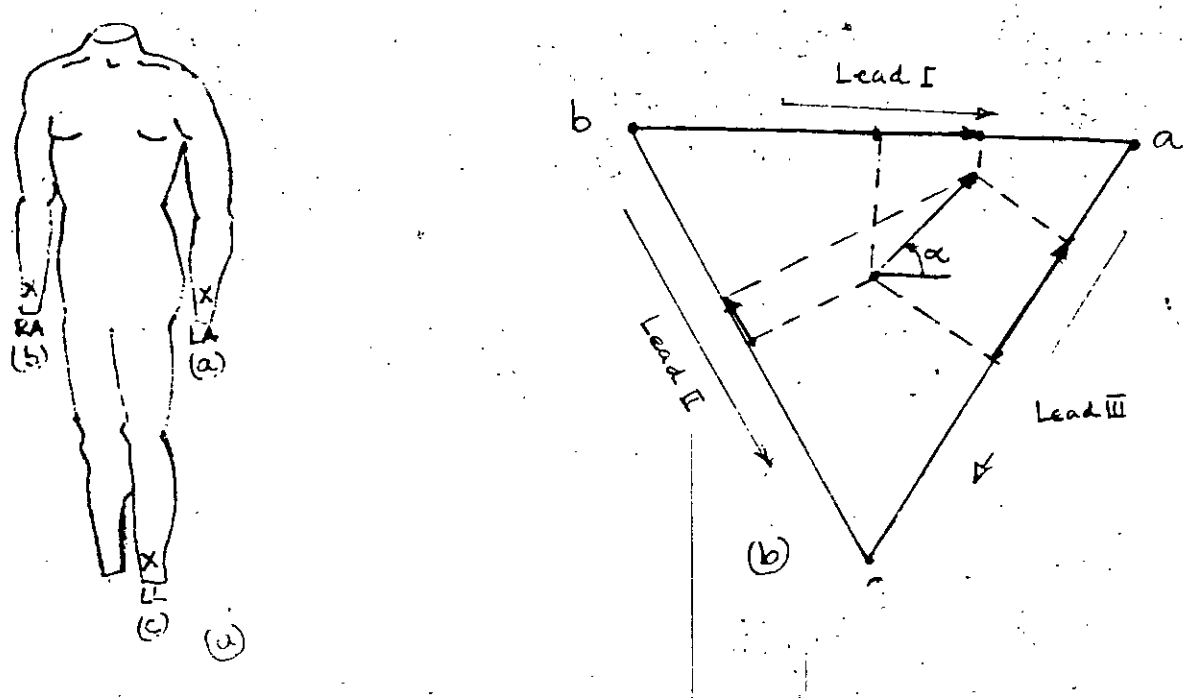
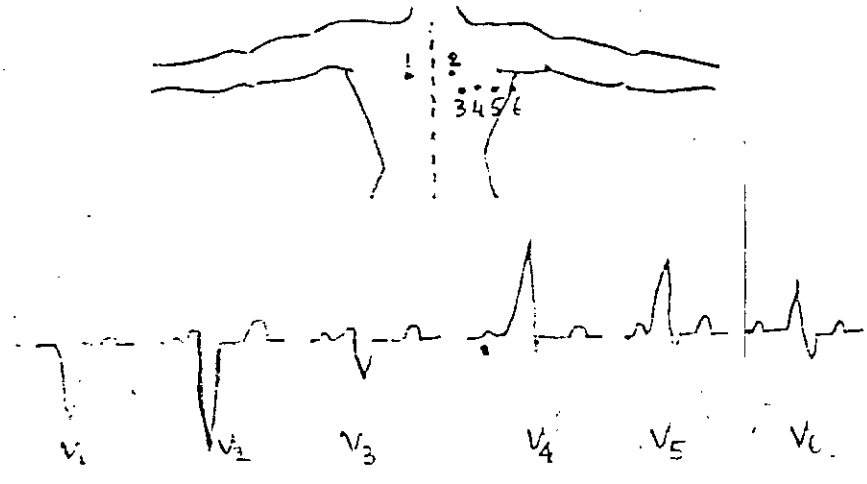


Fig 4·3(a) Standard leads of Einthoven. Each lead is located by an x. Fig 4·3(b) Einthoven triangle. Positive leads are represented by directed segments as shown for leads I, II and III.



Fig·6·4: Precordial leads. Location and typical records.

In the equation (1) to (3) double subscript notation is used for representation of voltage and is read as $V_{ab} = \phi_{(a)} - \phi_{(b)}$ where $\phi_{(a)}$ and $\phi_{(b)}$ represents the potential of a and that of (b) respectively. So V_{ab} represents the potential of (a) minus potential of (b).

From Kirchoff's Voltage Law we have

$$V_{ca} + V_{ab} = V_{cb}$$

hence,

$$V_{III} + V_{I} = V_{II} \quad .. \qquad ..(4).$$

The equation (4) can be represented by the following "Vector" relations in the Einthoven equilateral triangle illustrated in the Fig.(4.3b). The origin is the centre of the triangle, while the origin for each lead is the projection on the corresponding side. Positive directions are taken as indicated on the figure and corresponds to a reversal of the double-subscript notation (by convention). If the lead voltages are plotted along the sides of the triangle, each from its respective origion, then projections of their termini define the unique vector $\bar{V}$ as illustrated in the figure.

It can be shown that all the three projections meet in a common point. To do this let us assume the existence of the resultant vector $\bar{V}$. As a consequence, the sealar leads must be,

$$\text{Lead II} = V_{II} = -V\,\text{Cos}(120^{\circ}-\alpha) = \frac{V}{2}\text{Cos}\alpha - \frac{\sqrt{3}}{2}V\,\text{Sin}\alpha \quad ..(5).$$

Lead III $= V_{III} = -V \sin(\alpha + 30^\circ) = -\left(\frac{V}{2}\cos\alpha + \frac{\sqrt{3}}{2}\sin\alpha\right)$ .....(6).

Lead I $= V_I = V \cos\alpha$ .. .. ..(7).

we see that; $V_{III} + V_I = \frac{V}{2}\cos\alpha - \frac{\sqrt{3}}{2} V \sin\alpha = V_{II}$.

Hence, construction satisfies the relation (4).

From the figure it is seen that the vector V contains all the information of the three separate lead components. Its direction plotted at characteristic instants in the cardiac cycle has been found to have diagnostic significance. If positive is measured clockwise from the horizontal (the usual ECG convention) then it is called the electrical axis. In normal ECG $0 < \alpha < 90^\circ$. In the above figure α would be described as negative, a condition referred to as left axis deviation. For $\alpha > 90^\circ$, the condition is right axis deviation.

The electrical axis noted above is actually calculated from the effective lead voltages V_1, V_2, V_3. The later are determined as follows:

$$V_1 = Q_1 + R_1 + S_1$$

$$V_2 = Q_2 + R_2 + S_2$$

$$V_3 = Q_3 + R_3 + S_3$$

where Q_i, R_i and S_i are taken as peak values, they are not necessarily simultaneous. Thus the Einthoven construction may

not be unambiguous.

Precordial Leads:

An important reference lead has been arbitrarily established by connecting the 'limb lead' (that is RA, LA, and LL) together, each through a 5000-ohm resistance. The reference formed is known as the Wilson central terminal. If with respect to this the "exploring electrode" is placed over the chest at each of six standard locations, the precordial leads V_1, V_2, V_3, V_4, V_5 and V_6 are established. Fig. (4.4) shows a normal record for each precordial (chest) lead and also indicates the Chest position for each lead. In this system the reference is well defined and therefore, consistant results from patient to patient can be obtained.

In addition to the precordial leads, augmented unipolar limb leads are used where two limb leads are connected through 5000 ohms each to form a reference and the relative potential of the remaining limb lead is measured. If the positive terminal is the right arms, the lead is aV_R. The left arm(with respect to RA and LL tied through 5Kohms each) is lead aV_L. Similarly, the left leg gives aV_F.

The three standard leads plus $V_1, V_2, V_3, V_4, V_5, V_6$ and aV_R aV_L and Av_F constitute the standard 12-lead ECG.

Chapter 5

Mathematical Analysis of heart as a bioelectric source

From the previous discussion, the mechanism of nerve propagation can be described as resulting from the depolarization effect of action currents on the surrounding resting membrane. Through this mechanism the site of depolarization is capable of spreading contiguously untill all parts of an excitable fiber, and in some cases adjacent fibers, have been activated.

While the current density arising from membrane activity is strongest in the immediate vicinity of the active region, there will be current every where in the surrounding region. This region is called volume conductor and designate there by the aggregate of passive tissue which supports current from active sources. It is desirable to relate the measured potential difference that characterise a current flow field to the sources of that field.

5.1 Quasi-Static Formulation:

In order to describe mathematically, the potential field or current flow field in a volume conductor, due to the distributed time varying bioelectric sources we shall apply the concept of electromagnetic theory. Due to the electrical properties of physiological system, a simplified mathematical model of the source can be obtained with the assumption that the time-

varying bioelectric source can be considered as if steady state conditions exists at any instant of time.

We start with general formulation for the electric field in an infinite volume conductor due to an applied (impressed) current density $\bar{J}_i$, whose temporal behavior is harmonic at an angular frequency ω. The medium is assumed to be linear, homogenous, isotropic and characterized by physical parameters μ, σ and ϵ .

With an applied (impressed) current density $\bar{J}_i$ we can write the Helmotz equation as

$$\nabla^2 A + K^2 \bar{A} = - \mu \bar{J}_i \qquad (5.1.1)$$

where $\bar{A}$ is the vector magnetic potential and K is the propagation constant. In order to evaluate $\bar{E}$ and $\bar{H}$ it is advantageous to calculate first vector magnetic potential $\bar{A}$ and scalar potential $\emptyset$.

The vector potential $\bar{A}$ and scalar potential $\emptyset$ are given by[1]

$$\bar{A}(x', y', z') = \frac{\mu}{4\pi} \int_v \frac{\bar{J}_i(x,y,z)e^{-jkR}}{R} dv \qquad (5.1.2)$$

$$\emptyset(x',y',z') = \frac{1}{4\pi(\sigma + jw\epsilon)} \int_v \frac{I_v(x,y,z)e^{-jkR}}{R} dv \qquad (5.1.3)$$

where

$$R = \sqrt{(x - x')^2 + (y - y')^2 + (z - z')^2} \quad \ldots \quad (5.1.4).$$

The unprimed variables referring to the source point, the primed variables to the field point and $\nabla . \bar{J}_i = - I_v$ The last expression constitute the definition for volume current density I_v.

We also have from the wave equation

$$\nabla^2 \bar{E} = \mu \epsilon \frac{\partial^2 \bar{E}}{\partial t^2} + \mu \sigma \frac{\partial \bar{E}}{\partial t} \quad \ldots \quad (5.1.5).$$

with harmonic time variation

$$\nabla^2 \bar{E} = - \mu \epsilon E w^2 \bar{E} + jw \mu \epsilon \bar{E} = (-\mu \epsilon \omega^2 + jw \mu \sigma) \bar{E}$$

$$\nabla^2 \bar{E} = -K^2 \bar{E}$$

where $K^2 = \mu \epsilon w^2 - jw \mu \sigma$

$$\text{or } k^2 = w^2 \mu \left(\epsilon + \frac{\sigma}{jw} \right)$$

$$= \frac{\omega^2 \mu \sigma}{jw} \left(1 + \frac{jw\epsilon}{\sigma} \right)$$

$$= - jw \mu \sigma_c \quad \ldots \quad \ldots \quad (5.1.6)$$

Here $\sigma_c = \sigma \left(1 + \frac{jw\epsilon}{\sigma} \right)$ is a complex conductivity that includes pure conductive effects, dielectric losses and dielectric displacement.

Finally the electric field is found from vector and scalar potentials $\bar{A}$ and $\emptyset$ by

$$\bar{E} = jw\bar{A} - \nabla\emptyset . \qquad .. \quad (5.1.7).$$

The quantities $\bar{A}$, $\bar{E}$, $\emptyset$, I_v, σ_c and $\bar{J}_i$ are all complex and are functions of spatial co-ordinates and w. To obtain physical quantities it should be multiplied by e^{jwt} and considering only real or imaginary parts.

The usual implication of quasi-stationary involves the following assumptions, which simplify the above equations, for the bioelectric-materials, source and the surrounding medium.

Electrical properties of biological materials:

The value of the conductivity σ for biological material is chosen to be 0.2 mho/m which represents a mean value (Table-5.1)[1]. The highest component frequency of significance in bioelectric system is of the order of 1KHz. This probably relates the rise time for action potential in the order of 1 msec. So 1KHz is chosen as the maximum value of frequency in any computations regarding bioelectric phenomena. The complex conductivity $\sigma\left(1 + \frac{jw\epsilon}{\sigma}\right)$ should also be considered. The term $\frac{jw\epsilon}{\sigma}$ is the ratio of capacitive to resistive current should be considered. The maximum value of distance R corresponds to an over all dimension of typical physiological system; for simplicity Rmax = 1 meter.

Table-1

Tissue	Mean Conductivity mho/m.
Blood	0.67
Lung	0.05
Liver	0.14
Fat	0.04
Human trunk	0.21

Table-2

Average ratio of Capacitive to resistive current for various frequencies and body tissue.

	10 Hz	100 Hz	1KHz	10KHz
Lung	0.15	0.025	0.05	0.14
Fatty tissue	-	0.01	0.03	0.15
Liver	0.2	0.035	0.06	0.20
Heart Muscle.	0.10	0.04	0.15	0.32

Capacitive Effect:

The nature of the conductive medium is described by its conductivity and dielectric permittivity. The mathematical expression in eq. (5.1.3), where the Co-efficient $(\sigma + j\omega\epsilon)$ can be written as $\sigma(1 + \frac{j\omega\epsilon}{\sigma})$ and the conductivity viewed as a complex phasor quanity. In quasi-static approximation, a purely resistive medium is required. The quantity $(1 + \frac{j\omega\epsilon}{\sigma})$ will be real so long as $|\frac{j\omega\epsilon}{\sigma}| << 1$. Consultation of Table (5.2) revels that this condition is fairly satisfied. So for bioelectric system the conductivity may be considered as real.

Propagation Effect:

The time required for the changes at the source to "propagate" to a field point is represented by the phase delay e^{-jkR} in (5.1.2) and (5.1.3).

Since $$e^{-jkR} = 1 - jkR + \frac{(kR)^2}{2!} - \frac{(kR)^3}{3!} + \ldots \qquad -(5.1.8)$$

the propagation effect can be ignored if $|kR| << 1$, since e^{-jkR} is then approximately unity. The numerical value of k Rmax, even for $(1 + \frac{j\omega\epsilon}{\sigma})$ equals as high as 2 at frequency of 1KHz and with Rmax equals to 1meter gives.

$$kRmax = \frac{(1-j)}{\sqrt{2}}\sqrt{2\pi \times 1000 \times 4 \times \pi \times 10^{-7} \times 0.2}$$

$$= 0.0397\,(1-j)$$

Thus the magnitude of e^{-jkR} is unity to within a 4 percent error, while the phase-angle error of 0.0397 rad (2.3°) is clearly negligible.

Inductive Effect:

The inductive effect corresponds to the component of electric field that arises from magnetic induction. This is given, mathematically, in (5.1.7) by the term $jw\bar{A}$. We wish to compare the importance of this term relative to that expressed by $\nabla\phi$. We shall do this by considering the specific case of differential current element source and assume that if $|w\bar{A}| \ll |\nabla\phi|$, then the distributed source such might arise in electrophysiology and which are the superposition of such elements would also satisfy the in-equality.

Thus, for a source element $\bar{J}_i\, dV$ we have, from (5.1.2)

$$\bar{A} = \frac{\mu}{4\pi} \frac{\bar{J}_i dV}{R} \quad .. \qquad .. \qquad (5.1.9)$$

Since $\bar{A}$ and ϕ in (5.1.2) & (5.1.3) must satisfy the Lorentz equation

$$\nabla' . A = -jw\epsilon \left(1 + \frac{\sigma}{jw\epsilon} \right) \mu\phi \quad .. \qquad .. \quad , (5.1.10)$$

where $\nabla' \equiv \bar{a}_x \frac{\partial}{\partial x'} + \bar{a}_y \frac{\partial}{\partial y'} + \bar{a}_z \frac{\partial}{\partial z'}$ we have

$$\phi = \frac{\mu}{j\, 4\pi w \epsilon_c \mu} \nabla' . \frac{(\bar{J}_i dV)}{R} \quad .. \qquad .. \quad (5.1.11)$$

$$= - \frac{dV}{j4\pi w \epsilon_c} \bar{J}_i . \nabla \left(\frac{1}{R} \right) = \frac{dV}{j4\pi w \epsilon_c} \frac{\bar{J}_i . \bar{a}_R}{R^2}$$

.....(5.1.12)

and $\epsilon_c = \epsilon \left(1 + \frac{\sigma}{jw\epsilon}\right)$.

The unprimed del operator is defined by

$$\nabla = \bar{a}_x \frac{\partial}{\partial x} + \bar{a}_y \frac{\partial}{\partial y} + \bar{a}_z \frac{\partial}{\partial z}$$

operates on source co-ordinate only. The following identity is also used in deriving (5.1.12) .

$$\nabla \left(\frac{1}{R}\right) = - \nabla' \left(\frac{1}{R}\right) .$$

If we let z axis coincide with the direction of $\bar{J}_i$, then

$$\phi = \frac{\bar{J}_i \, dV \, \cos\theta}{j4 \, w \epsilon_c \, R^2} \quad .. \quad .. \quad (5.1.13)$$

and $$|\nabla \phi| = \frac{\bar{J}_i \, dV}{4\pi w \epsilon_c R^3} \quad .. \quad .. \quad (5.1.14)$$

The ratio of interest is then $\left|\frac{w\bar{A}}{\nabla\phi}\right| = |w^2 \mu \epsilon_c \bar{R}|$

$$= |kR|^2 \quad .. \quad (5.1.15)$$

Thus inductive effect to be negligible $|kR|^2 \ll 1$ must be satisfied. So long as propagation effect is ignored this condition is automatically satisfied.

Boundary Conditions:

Since the total current (conduction plus displacement) is solinoidal, the normal component at the interface between two media must be continuous. The boundary condition between

different tissues is given by

$$\sigma_1 \left(1 + \frac{jw\epsilon_1}{\sigma_1} \right) E_{1n} = \sigma_2 \left(1 + \frac{jw\epsilon_2}{\sigma_2} \right) E_{2n} \quad -(5.1.16)$$

If we neglect the displacement current at the boundary, (5.1.16) is reduced to

$$\sigma_1 E_{1n} = \sigma_2 E_{2n} \quad .. \quad .. \quad (5.1.17)$$

where σ_1 and σ_2 are the conductvities of regions 1 and 2 respectively and E_{1n} and E_{2n} the respective normal component of electric fields.

For electrocardiography, the space which surrounds the human body has conductivity $\sigma_2 = 0$ which results in $E_{1n} = 0$.

The meaning of the above criterion may be clarified by noting that boundary conditions serve to take into account secondary surface sources (at the boundary). The field in the conductive medium can be thought of as containing a component which arises from the primary sources $\bar{E}_o$ and a portion due to the aforementioned secondary source $\bar{E}_s$; the total field in the external medium is designated $\bar{E}_e$. Application of (5.1.16) gives,

$$\sigma_1 \left(1 + \frac{jw\epsilon_1}{\sigma_1} \right) E_{on} + \sigma_1 \left(1 + \frac{jw\epsilon_1}{\sigma_1} \right) E_{sn} = jw\epsilon_2 E_{en} \quad \ldots (5.1.18)$$

Now E_{on}, E_{sn} and E_{en} are in the same order of magnitude, so that if $|jw\epsilon_2/\sigma_1| \ll 1$, then

$$\sigma_1 \left(1 + \frac{jw\epsilon_1}{\sigma_1}\right) E_{on} = - \sigma_1 \left(1 + \frac{jw\epsilon_1}{\sigma_1}\right) E_{sn} \quad .. \qquad (5.1.19)$$

i.e. the total normal current due to the applied field is equal and opposite that due to the secondary field. Equation (5.1.19) is equivalent to the requirement

$$\left(E_o + E_s \right)_n = 0 \quad .. \qquad .. \qquad (5.1.20)$$

which corresponds to the stationary condition ($E_{in}=0$).

Now utilizing the above simplication the expression for scalar potential is,

$$\phi = \frac{1}{4\pi\sigma} \int_v \frac{I_v (x, y, z)}{R} dV \quad .. \qquad (5.1.21)$$

$$\bar{E} = - \nabla \phi \quad .. \qquad (5.1.22)$$

The total current $\bar{J}$ is the sum of the source current $\bar{J}_i$ and the conduction current $\sigma\bar{E}$; consequently

$$\bar{J} = \bar{J}_i + \sigma\bar{E} \quad .. \qquad (5.1.23)$$

Since $\bar{J}$ is solenoidal

$$\nabla \cdot \bar{J} = 0 = \nabla \cdot \bar{J}_i + \nabla \cdot \sigma\bar{E} \quad .. \qquad (5.1.24)$$

and for homogeneous media we have, utilizing (5.1.22) and (5.1.23),

$$\nabla \cdot \bar{J}_i = \sigma \nabla^2 \phi. \quad .. \qquad (5.1.25)$$

Taking the Laplacian of (5.1.21) we get.

$$\nabla^2 \phi = - \frac{I_v}{\sigma} \quad .. \qquad .. (5.1.26)$$

where $I_v = - \nabla \cdot \bar{J}_i$.

The equation (5.1.26) represents the conventional, basic, quasi-static mathematical formulation with (5.1.16) as boundary conditions. Although homogeneous condition have been assumed in this analysis, the results are readily generalized to a region consisting of phase inhomogeneities.

In the material that follows we shall utilize Possion's equation as stated in (5.1.26) and electric field is derived from (5.1.22). Furthermore from (5.1.25) and (5.1.26) the volume source density is also expressed interms of the impressed current by

$$\nabla \cdot \bar{J}_i = - I_v \quad .. \qquad .. \quad (5.1.27).$$

and finally the current flow field will be related to the potential field by virtue of (5.1.23).

5.2 Dipole Representation of Electrical Activity in Heart as a bioelectric source:

It is shown in previous discussion that the total instantaneous current density consists of ohmic and non-ohmic components. With this decomposition the total current density $\bar{J}$ is given by

$$\bar{J} = \sigma \bar{E} + \bar{J}_i \quad .. \qquad (5.2.1).$$

where $\bar{J}_i$ is the non-ohmic part. The term $\sigma \bar{E}$ is a differential expression of ohm's law and evaluates the current density that arises in a pasiive conductive medium of conductivity σ because of the presence of an electric field $\bar{E}$. The electric field and the current associated with it ($\sigma\bar{E}$), are a response to the primary sources represented by $\bar{J}_i$. This latter component designates the "impressed" current density and is a consequence of the conversion of energy to an electric form.

In Nerst equation[11], within membrane the current density is described as a sum of conduction current and diffusion current. The diffusion current is a contribution to the impressed current density and results from conversion of chemical energy into electrical form. For cardiac muscle, the capacitive displacement current, in addition to the different ionic contributions to the impressed current, may not be neglected and and both diffusion current and capacitive displacement current constitute the basic impressed current.

In equation (5. .1) $\bar{J}$, since it includes all possible contributions, must be solenoidal. Consequently we obtained earlier in (5.1.24).

$$\bar{J} = 0 = (\sigma\bar{E}) + \bar{J}_i \quad , \quad .. \quad (5.2.2.)$$

If we consider a homogeneous region of conductivity σ in which impressed current exists then equation (5.1.25) can be written

as

$$\nabla^2 \phi = \frac{\nabla \cdot \bar{J}_i}{\sigma} \quad .. \qquad .. \qquad (5.2.3)$$

and is an expression for Poisson's equation. The role of $\bar{J}_i$ as a source function is clearly seen in (5.2.3) which is a partial differential equation for ϕ with $(\nabla \cdot \bar{J}_i)/\sigma$ as a nonhomogeneous(source) term,

In electrostatic fields, the potential due to the charge density $\rho(x, y, z)$ in a uniform dielectric medium of permittivity ϵ is

$$\phi(x', y', z') = \frac{1}{4\pi\epsilon} \int_V \frac{\rho \, dV}{r} \quad .. \qquad (5.2.4)$$

where (x', y', z') locates the field point and

$$r = \sqrt{(x-x')^2 + (y-y')^2 + (z-z')^2} \quad .. \qquad (5.2.5)$$

is the distance from the source point $(x\ y\ z)$ to the field point. But $\phi(x, y, z)$ also satisfies the Poinson's equation, namely

$$\nabla^2 \phi = - \frac{\rho}{\epsilon} \quad .. \qquad .. (5.2.6)$$

If (5.2.6) is compared with (5.2.3), it can be noted that these equations are interchangeable if ϵ is replaced by σ, and ρ by $-\nabla \bar{J}_i$. If this duality is applied to (5.2.4), then we can obtain

$$\phi(x', y', z') = \frac{1}{4\pi\sigma} \int_V - \frac{\nabla \cdot \bar{J}_i}{r} \, dV \quad .. \quad (5.2.7)$$

as a solution to (5.2.3). We note from equation (5.2.7) as well as duality of $-\nabla\cdot\bar{J}_i$ with ρ , that $(-\nabla\cdot\bar{J}_i)$ plays the role of a divergence-type source density. Utilizing the designation of (5.1.27), we have

$$-\nabla\cdot\bar{J}_i = I_v \qquad .(5.2.8)$$

where I_v has the dimensions of current per unit volume. With this notation

$$\phi\ (x', y', z') = \frac{1}{4\pi\sigma}\int \frac{I_v}{r}\,dV \qquad ..(5.2.9)$$

The equation (5.2.9) is a solution to (5.2.3) can be demonestrated by considering a point source δ , where $\delta = 0$ except at the origin, where it is infinite, but $\int_v \delta\,dV = 1$. Let ϕ_o be the solution to the following partial differential equation:

$$\nabla^2\phi_0 = \frac{\delta}{\sigma} \qquad .. (5.2.10).$$

Because of the spherical symmetry, ϕ_o depends only on r and writting the Laplaccian in spherieal co-ordinates, we get

$$\frac{1}{r^2}\ \frac{d}{dr}\ \left(r^2\ \frac{d\phi_o}{dr}\right) = \frac{\delta}{\sigma} \qquad ..(5.2.11).$$

If we integrate with respect to r , then

$$r^2\frac{d\phi_o}{dr} = \frac{1}{\sigma}\int \delta r^2 dr$$

$$= \frac{1}{4\pi\sigma}\int 4\pi r^2\,dr = \frac{1}{4\pi\sigma}\int \delta\,dV$$

$$= \frac{1}{4\pi\sigma} \qquad .. (5.2.12).$$

A second integration yields

$$\phi_o = - \frac{1}{4\pi\sigma r} \qquad .. \quad (5.2.13)$$

In the notation used in above equation, we have $r = \left[x'^2 + y'^2 + z'^2\right]^{\frac{1}{2}}$ and (x', y', z') is the point at which ϕ_o is evaluated, the delta source being at (0, 0, 0,).

Now for a source ($\nabla \cdot \bar{J}_i / \sigma$), we can consider it the superposition of delta sources typified by $(\nabla \cdot \bar{J}_i / \sigma)dV$ at (x, y, z) whose contribution to the potential field $d\phi_o(x', y', z')$ at (x', y', z') is given by [using (5.2.13) with origin at (x, y, z) and field point at (x', y', z')]

$$d\phi_o (x',y',z') = - \frac{(\nabla \bar{J}_i \,)dV}{4\pi \sigma r} \qquad .. \quad (5.2.14)$$

and $r = \sqrt{(x'-x)^2 + (y'-y)^2 + (z'-z)^2}$. Integration of(5.2.14) gives (5.2.7) as required.

It often proves convenient to represent the bioelectric sources as volume source density function I_v. On this basis, the potential field is found as a solution to Poisson's equation

$$\nabla^2 \phi = - \frac{I_v}{\sigma} \qquad .. \quad (5.2.15)$$

On the other hand, representation of the sources as an impressed current density $\bar{J}_i$ permits an identification with physical processes in the membrane. Still another interpretation

of $\bar{J}_i$ is obtained through the vector identity:

$$\nabla \cdot \left(\frac{\bar{J}_i}{r}\right) = \frac{1}{r} \nabla \cdot \bar{J}_i + \bar{J}_i \cdot \nabla \left(\frac{1}{r}\right) \qquad .. (5.2.16)$$

If the integral of both sides of (5.2.16) is taken, which extends throughout the total volume in which sources are located, and if the divergence theorem is used, then

$$\int_s \frac{\bar{J}_i \, d\bar{s}}{r} = 0 = \int_v \frac{\nabla \cdot \bar{J}_i}{r} \, dV + \int_v \bar{J}_i \cdot \nabla \left(\frac{1}{r}\right) dV \qquad ..(5.2.17)$$

The surface integral evaluates to zero since $\bar{J}_i = 0$ over the bounding surface which lies beyond all sources. Consequently,

$$\int_v - \frac{\nabla \cdot \bar{J}_i}{r} \, dv = \int_v \bar{J}_i \cdot \nabla \left(\frac{1}{r}\right) dv \qquad ..(5.2.18)$$

where $\nabla \left(\frac{1}{r}\right) = - \left(\frac{1}{r^2}\right) \bar{a}_r$ and $\bar{a}_r$ is a unit vector from the field to the source point. Substituting (5.2.18) to (5.2.7) gives

$$\phi (x', y', z') = \frac{1}{4\pi\sigma} \int_v J_i \, dV \cdot \nabla \left(\frac{1}{r}\right) \qquad (5.2.19)$$

But we know that the potential field of a dipole source of strength $\bar{P}$ is

$$\phi(P) = \frac{1}{4\pi\sigma} \bar{P} \cdot \nabla \left(\frac{1}{r}\right) \qquad .. (5.2.20)$$

If (5.2.20) is compared with (5.2.19) then $\bar{J}_i \, dV$ may be interpreted as elemental dipole source. Put in another way $\bar{J}_i$ has the dimensions of a dipole moment per unit volume. Since the isolated point source with fields as given by (5.2.13) can not

exist in electrophysiological systems, the characterization of a bioelectric source as a dipole distribution is a natural one.(1)

5.3.1. Centric Dipole Model of Heart:

There is abundent evidence that the depolarization (electrical activity) of action potential spreads uniformly through the myocardium in a wavelike manner(at least in the outer two thirds of the wall). The width of the wavefront encompasses five or more cardiac cells. The actual source in the wave width can be replaced by an averaged dipole moment per unit volume designated by $\bar{J}_i$ and is averaged from the physical source entities in the same way as the mathematical charge density is related to a physical cloud of point charges.

If the source density $\bar{J}_i$ is uniform over a surface normal to the direction of propagation, which is usually assumed, then the field setup by the activation wave can be obtained from the following analysis. So a simple approximation that will represent the electrical activity of heart is to simply find the vector summation of all dipole moments $\bar{J}_i dV$. That is, we form the vector $\bar{P} = \int_V \bar{J}_i dV$ and let this single dipole represent the heart electrically and it can be designated as a equivalent heart vector.

Surface Potential Due to the Effective Dipole(Heart) Source:

One of the important ECG problem is to obtain the surface potential due to the effective dipole(heart) source. The following technique[3] can be used to compute the surface potential due to the internal current source in three dimensional, finite and homogenous media. The effects of inhomogeneitics is also considered in subsequent discussion.

Problems in homogeneous Media:

The relationship between current density source $I_v(-\nabla\cdot\bar{J}_i)$ and ϕ, the potential is given by Poisson's equation $\nabla^2\phi = \frac{I_v}{\sigma}$, where σ is the electrical conductivity. In an infinite homogeneous medium of conductivity σ, a solution to the Poisson's equation is

$$\phi(x) = \frac{1}{4\pi\sigma}\int \frac{I_v(x')}{r(x,x')}dv' \qquad ..(5.3.1)$$

where the integration is over all the space.(Fig. 5,3.a)

To find the relationship within an arbitray surface S, We can use Green's threrem:

$$\int(\phi\nabla^2\psi - \psi\nabla^2\phi)\,dv' = \int(\phi\nabla\psi - \psi\nabla\phi)\,ds \qquad (5.3.2)$$

where ϕ and ψ are scalar functions of position. The two functions are chosen as ψ the function $\frac{1}{r}$ and ϕ as the electric potential. Substituting $\frac{1}{r}$ for ψ in (5.3.2) and integrating gives

$$\phi(x) = \frac{1}{4\pi\sigma}\int \frac{I_v(x')}{r}\,dv' + \frac{1}{4\pi}\int\left(\phi\frac{\bar{r}}{r^3} + \frac{\nabla\phi}{r}\right)\cdot d\bar{S} \qquad (5.3.3)$$

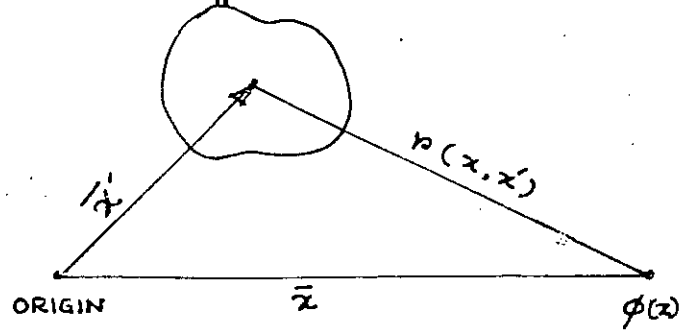


FIGURE 5.3a. POTENTIALS IN AN INFINITE MEDIUM.

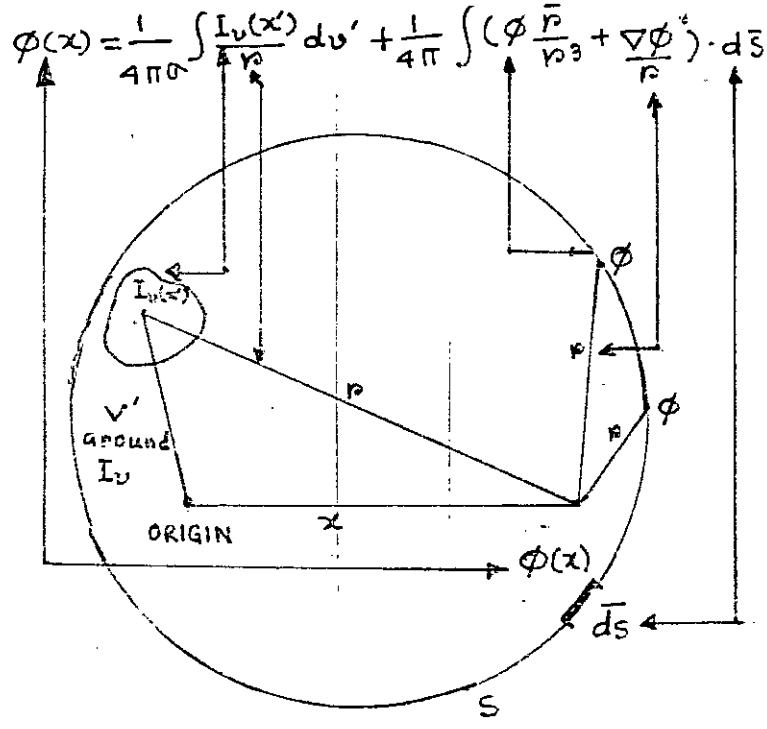


FIG. 5.3b. THE POTENTIAL ON AND IN A CLOSED SURFACE 'S'. A DISTRIBUTION SOURCE AND SINKS $I_v(x')$ IS IN THE INTERIOR REGION V'. $\bar{x}'$ IS A VECTOR TO AN ARBITRARY POINT IN THE INTERIOR, AND $\phi(x)$ IS THE POTENTIAL AT x. ϕ AND $\nabla\phi$ ARE THE POTENTIAL AND POTENTIAL GRADIENT AT A POINT ON THE SURFACE. $\bar{r}$ EXTENDS FROM x EITHER TO THE CURRENT SOURCES OR TO THE SURFACE.

The potential $\phi(x)$ and its different component is shown in fig. 5.3b. These integrations are now only over the boundary and inside the surfaces S. If the normal component of $\nabla\phi$ is zero on the boundary, a very good approximation in electrocardiography, (5.3.3) becomes

$$\phi(x) = \frac{1}{4\pi\sigma}\int \frac{I_v(x')}{r}\, dv' + \frac{1}{4\pi}\int \phi \frac{\bar{r}}{r^3}\, \bar{ds} \quad \ldots \quad \ldots \quad (3.3.4)$$

However $\frac{\bar{r}}{r^3}\cdot ds$ is identical with a differential element of solid angle $d\Omega$.

That is,

$$\phi(x) = \frac{1}{4\pi\sigma}\int \frac{I_v(x')}{r}\, dv' + \frac{1}{4\pi}\int \phi\, d\Omega \quad \ldots \quad \ldots (5.3.5)$$

This is shown in Fig. (5.3.c)

The expression (5.3.5), could equivalently, be written as:

$$\phi(x) = \frac{1}{4\pi\sigma}\int \frac{I_v(x)}{r}\, dv' + \lim_{n\to\infty} \sum_{j=1}^{N} \frac{1}{4\pi}\, \phi_j\, \Delta\Omega_j \quad \ldots \quad \ldots \quad (5.3.5)$$

Since the summation is over the bounding surface S, each $\phi(x)$ is the potential on the jth segment of that surface, and each $\Delta\Omega_j$ is the increment of solid angle of the jth segment when viewed from the point x .

A finite summation approximates the limit as j approaches infinity. That is

$$\phi(x) \approx \frac{1}{4\pi\sigma}\int \frac{I_v(x')}{r}\, dv' + \sum_{j=1}^{N} \frac{1}{4\pi}\, \phi_j\, \Delta\Omega_j \quad \ldots \quad \ldots (5.3.6)$$

The $\phi(x)$ on the left side is the potential at an artitray point inside the surface, but since $\phi(x)$ can be located at an arbitrary interior point, one may choose an especially favorable location. In particulars we choose the location of $\phi(x)$ inside the i^{th} element and very close to the outer surface of the i^{th} element. However, since $\nabla \phi$ normal to the surface is zero, a point very close to the surface point has the same potential as the surface point, and $\phi(x)$ equals to ϕ_i .

Therefore, with $\phi(x)$ just inside element i,

$$\phi(x) = \phi_i = \frac{1}{4\pi\sigma}\int \frac{I_v}{r} dv' + \sum_{j=1}^{N} \frac{1}{4\pi} \phi_j \Delta\Omega_j \quad \cdots \quad \cdots (5.3.6)$$

The summation is over all n of the surface elements. One term of of the summation is for j equals i. This term is $\frac{1}{4\pi}\phi_i \Delta\Omega_i$. The ϕ_i in this term is same as ϕ_i on the left side of the equation. The $\Delta\Omega_i$ represents the solid angle of the i^{th} element as seen from the observation point at x. Since the observation point is just inside the i^{th} element, the solid angle of the i^{th} element as seen from the observation point is about 2π. That is, there are many surface elements; each is there by approximately flat. From a point just inside the i^{th} element, this element obseures a solid angle of 2π .

Therefore,

$$\phi(x) = \phi_i = \frac{1}{4\pi\sigma}\int \frac{I_v}{r} dv' + \sum_{j=1, j\neq i}^{N} \frac{1}{4\pi} \phi_j \Delta\Omega_j + \frac{2\pi}{4\pi} \phi_i$$

$$\text{or,} \quad \phi_i = 0.5\phi_i + \frac{1}{4\pi\sigma}\int \frac{I_v}{r} dv' + \sum_{j=1, j\neq i}^{N} \frac{1}{4\pi} \phi_j \Delta\Omega_j \quad \cdots \quad \cdots (5.3.7)$$

or $$0.5\phi_i + \sum_{j=1, j\neq i}^{N} \left(-\frac{\Delta\Omega_j}{4\pi}\right)\phi_j = \frac{1}{4\pi\sigma}\int \frac{I_v}{r}\, dv' \qquad \cdots(5.3.7)$$

The equation (5.3.7) directly relates the surface potentials to the internal charges, and by choosing i successively just inside surface element one, two, three and so forth, one generates a series of simultaneous equations. If A_{ij} is $(-\frac{\Delta\Omega_j}{4\pi})$ as observed from a point just inside the ith element (i not equal to j) and A_{ii} is one half, then these equations are the set of equations

$$A_{i1}\phi_1 + A_{i2}\phi_2 + \cdots\cdots\cdots + A_{iN}\phi_N = \frac{1}{4\pi\sigma}\int \frac{I_v}{r}\, dv' \qquad (5.3.8)$$

where r is the distance from an increment of current source density to the ith surface element.

Let

$$B_i = \frac{1}{4\pi\sigma}\int \frac{I_v}{r}\, dv' \qquad \cdots \qquad \cdots \qquad (5.3.9)$$

Then using matrix notation (5.3.8) becomes

$$[A][\phi] = [B] \qquad \cdots \qquad \cdots \qquad (5.3.10)$$

The matrix [A] depends only on the characteristic of the surface while matrix[B]depends on charge inside. In fact B_i is just the solution for an infinite medium for the potential at the element i from the internal charges.

$$\phi(x) = \frac{1}{4\pi\sigma}\int I_v(x')dv' + \frac{1}{4\pi}\int \phi \, d\Omega$$

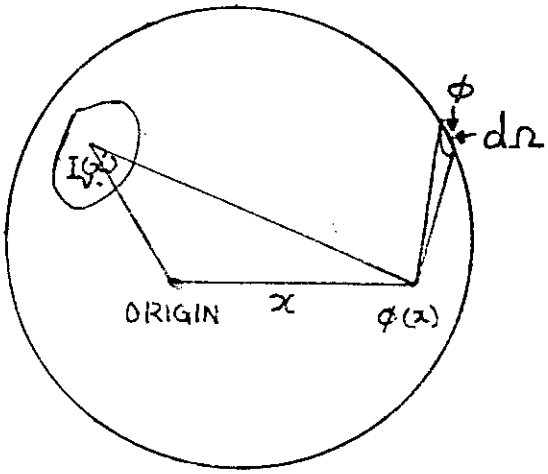


FIG. 5.3c. POTENTIAL INSIDE AND ON A VOLUME.

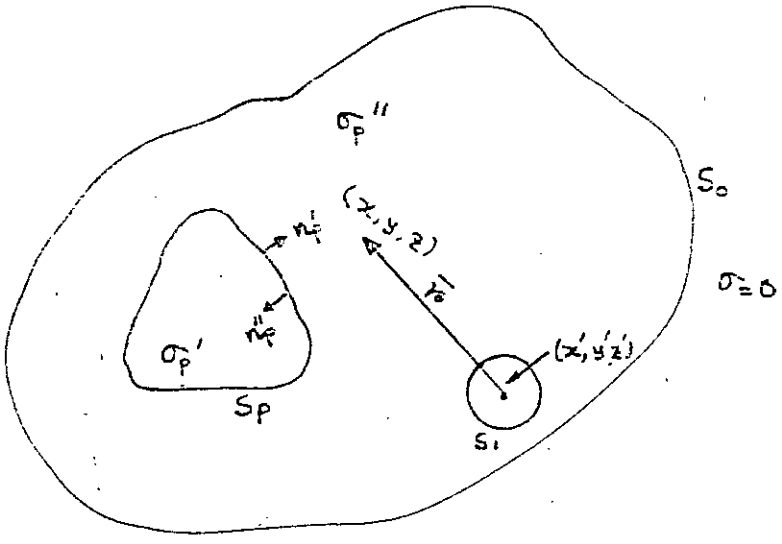


FIG. 5.3d. A VOLUME S_o CONTAINING AN INHOMOGENEOUS REGION S_p.

Problems in Inhomogeneous Media:

If the media contains regions of different conductivity, one must modify the equation (5.3.2) to include the interfaces between these regions. A generalization of equation (5.3.2), to include the effects of regions of different permittivity, can be made as follows:(3)

$$\sum_{p=1}^{q} \int_{S_p} \left[\sigma_p' \left(\psi_p' \frac{\partial \phi_p'}{\partial n_p'} - \phi_p' \frac{\partial \psi_p'}{\partial n_p'} \right) + \sigma_p'' \left(\psi_p'' \frac{\partial \phi_p''}{\partial n_p''} - \phi_p'' \frac{\partial \psi_p''}{\partial n_p''} \right) \right] dS_p$$

$$+ \sum_{j} \int_{S_j} \sigma_j \left(\psi_j \frac{\partial \phi_j}{\partial n_j'} - \phi_j \frac{\partial \psi}{\partial n_j} \right) dS_j = \int_V \left[\psi \nabla \cdot (\sigma \nabla \phi) - \phi \nabla \cdot (\sigma \nabla \psi) \right] dv'$$

... ... (5.3.11)

where n_p' and n_p'' represents the unit normal vectors drawn into the pth boundary that encloses all the regions. This is shown in the fig. (5.3.d)

Let, $\psi = \frac{1}{r}$ and v' go from S_1 to S_2. For simplicity, consider only two regions of different conductivity as shown in figure 5.3.d. Equation (5.3.11) can now be written as

$$\int_{S_p} \left(\sigma_p' \frac{1}{r} \frac{\partial \phi_p'}{\partial n_p'} + \sigma_p'' \frac{1}{r} \frac{\partial \phi_p''}{\partial n_p''} \right) dS_p - \int_{S_p} \left[\sigma_p' \phi_p' \frac{\partial}{\partial n_p'} \left(\frac{1}{r} \right) + \sigma_p'' \phi_p'' \frac{\partial}{\partial n_p''} \left(\frac{1}{r} \right) \right] dS_p$$

$$+ \int_{S_o} \sigma_p'' \left[\frac{1}{r} \frac{\partial \phi_o}{\partial n_o} - \phi_o \frac{\partial}{\partial n_o} \left(\frac{1}{r} \right) \right] dS_o + \int_{S_1} \left[\sigma \frac{1}{r} \frac{\partial \phi_1}{\partial n_1} - \sigma \phi_1 \frac{\partial}{\partial n_1} \left(\frac{1}{r} \right) \right] dS_1$$

$$= \int_V \left[\frac{1}{r} \nabla \cdot (\sigma \nabla \phi) - \phi \nabla \cdot \sigma \nabla \left(\frac{1}{r} \right) \right] dV'$$

... . (5.3.12)

where σ is a variable having either the value σ_p'' or σ_p' and $\bar{r}$ is a vector to the location under consideration.

Furthermore, at the interface between regions of different ent conductivities the normal component of current is continuous and using the following relations:

a) $$\sigma_p' \frac{\partial \phi_p'}{\partial n_p'} = -\sigma_p'' \frac{\partial \phi_p''}{\partial n_p''} \quad \text{in integral 1} \qquad (5.3.13)$$

b) $$\sigma \nabla^2 \phi = -I_v \quad \text{in integral 5} \qquad (5.3.14)$$

[Poisson's equation in inhomogeneous media]

c) $$\phi_p''(r) = \phi_p'(r) \quad \text{in the integral 2} \qquad (5.3.15)$$

and(d)

$$\int_{S_1} \frac{\sigma}{r} \frac{\partial \phi_1}{\partial n_1} dS_1 + \int_{S_1} \frac{\sigma}{r^2} \bar{r}\,\bar{n}_1 \phi_1 \, dS \xrightarrow{\lim r \to 0} -4\pi\sigma\phi(x',y',z')$$

in integral 4 ..(5.3.16)

the equation (5.3.12) may be written

$$\phi(x',y',z') = \frac{1}{4\pi\sigma_p''}\left[\int \frac{I_v}{r} dv' + \int \frac{\sigma_p' - \sigma_p''}{r^2} \phi_p \bar{r}\,\bar{n}_p \, dS_p + \sigma_p'' \int \phi_o \frac{\bar{r}\,\bar{n}_o}{r^2} dS_o\right] \qquad ...(5.3.17)$$

anywhere in σ_p'' region. Just as in the homogeneous case, ϕ (x', y', z') may be located arbitrarily close to the boundary

and, since $\nabla\phi$ is zero on the boundary, ϕ (x', y', z') arbitrarily close to the boundary is equal to ϕ_o on the boundary. Replacing the S_o surface integral by N finite area elements and the S_p integral by M area increments (5.3.17) may be rewritten as

$$0.5\phi_i + \sum_{j=1, j\neq i}^{N} \left(-\frac{\Delta\Omega_j}{4\pi}\right)\phi_j - \sum_{K=1}^{M} \frac{\sigma_p' - \sigma_p''}{4\pi\sigma_p''} \Delta\Omega_K \phi_K = \frac{1}{4\pi\sigma_p''} \int \frac{\rho}{r}\, dv'$$

... ... (5.3.18)

Equ.(5.3.18) represents N equations but there are N plus M unknowns. M more equations result from repeating the steps used in obtaining (5.3.17) but locating the observation point ϕ (x', y', z') at the $(\sigma_p' - \sigma_p'')$ surface interface.

The result is

$$\phi(x', y', z') = \frac{1}{4\pi\sigma_p''}\left[\int \frac{I_v}{r}\, dv' + \int \frac{\sigma_p - \sigma_p''}{r^2} \phi_p\, \bar{r}\cdot\bar{n}_p \cdot ds_p + \sigma_p'' \int \phi_o \frac{\bar{r}\cdot\bar{n}}{r^2}\, ds_o\right]$$

at $\sigma_p' - \sigma_p''$ interface.

...(5.3.19)

Replacing the integrals in (5.3.19) by the same area increments used to obtain (5.3.18) gives the needed M equations.

$$\left[1 + \tfrac{1}{2}\left(\frac{\sigma_p' - \sigma_p''}{\sigma_p''}\right)\right]\phi_i + \sum_{j=1}^{N} -\frac{\Delta\Omega_j}{4\pi}\phi_j - \sum_{K=1,\, K\neq i}^{M} \frac{\sigma_p' - \sigma_p''}{\sigma_p'' r^2} \Delta\Omega_K \phi_K = \frac{1}{4\pi\sigma_p''} \int \frac{I_v}{r}\, dv'$$

5.3.2 Potential Due to Centric Dipole Model:

For an idealized torso that is represented as a uniform conducting sphere, and with a heart model that consists of central dipole (Fig. 5.3.e), the surface potential can readily computed. We have for potential ϕ ,

$$\nabla^2 \phi = 0 \qquad \ldots\ldots \qquad ..(5.3.2.1)$$

$$\left.\frac{\partial \phi}{\partial r}\right|_{r=R} = 0 \qquad ..(5.3.2.2)$$

Solution to (5.3.2.1) in spherical co-ordinates with $\frac{\partial}{\partial \phi} = 0$ due the symmetry, are of the form

$$\phi = \sum_n \left[a_n r^n P_n(\cos\theta) + b_n r^{-(n+1)} P_n(\cos\theta) \right. \qquad ..(5.3.2.3)$$

Since the potential near the origin must vary as $\frac{1}{r^2}$, we choose n=1. [Other terms in positive powers of r are not excluded on this ground, but one can confirm, when applying (5.3.2.2), that since $b_n = 0 \quad (n \neq 1)$ it is necessary that $a_n = 0$] . Consequently, since $P_1 (\text{Cos}\theta) = \text{Cos}\theta$,

$$\phi = ar\cos\theta + \frac{b}{r^2}\cos\theta \qquad .. \qquad .. (5.3.2.4)$$

$$\left.\frac{\partial \phi}{\partial r}\right|_{r=R} = a\cos\theta - \frac{2b}{R^3}\cos\theta = 0 \qquad .. (5.3.2.5)$$

Hence

$$a = 2b/R^3$$

$$\phi = \frac{b}{R^2}\cos\theta \left[\frac{2r}{R} + \left(\frac{R}{r}\right)^2 \right] \qquad .. (5.3.2.6)$$

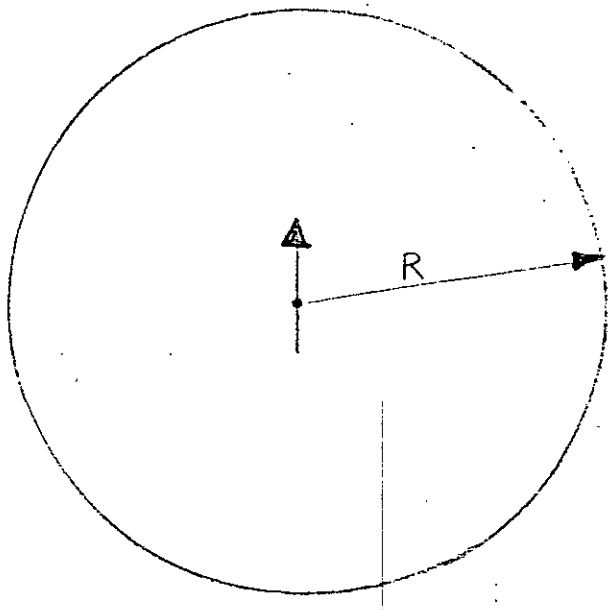


Fig. 5.3e. Centric dipole in a conducting sphere.

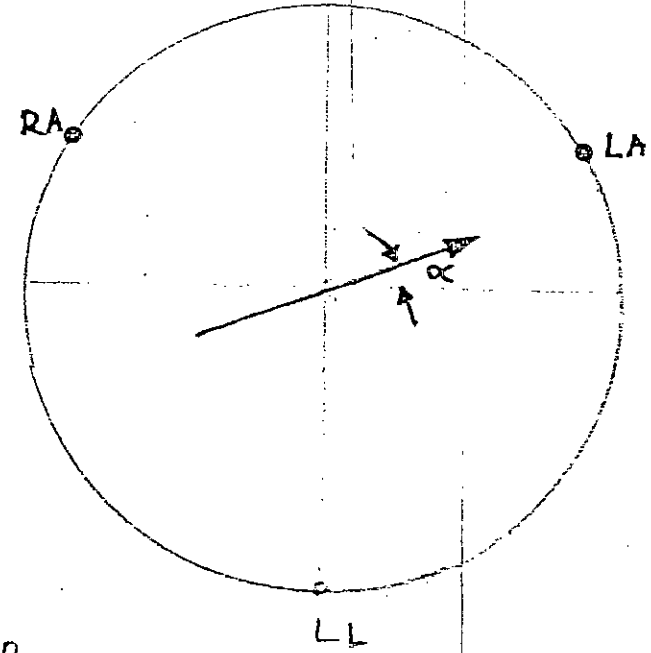


Fig. 5.3.f. Spherical-model simulation of Eindhoven system.

The potential over the surface is $(3b/R^2)\cos\theta$ and this is three times what it would be if the body were infinite in extent.

If we adopt the fiction that the points $\theta = 30^\circ$, $\phi = 0^\circ$; $\theta = 30^\circ$, $\phi = 180^\circ$; $\theta = 180^\circ$ are LA, RA and LL, respectively, then the potential produced by a central dipole of arbitrary orientation and magnitude are precisely those given by the Einthoven triangle. The Einthoven system tacitly assumes that the "heart dipole" lies in the frontal plane, here defined by ($\phi = 0^\circ$, 180°). This can be confirmed by reference to fig. (5.3.f) where the case of unit dipole vector($3b/R^2=1$) at an angle α with the horizontal is described.

Applying (5.3.2.6) to evaluate the potential at the 'limb' locations, we obtain

$$\phi_{LA} = \cos(\alpha - 30^\circ) = \frac{\sqrt{3}}{2}\cos\alpha + \tfrac{1}{2}\sin\alpha \quad ..(5.3.2.7).$$

$$\phi_{RA} = \cos(150^\circ - \alpha) = -\frac{\sqrt{3}}{2}\cos\alpha + \tfrac{1}{2}\sin\alpha \quad ..(5.3.2.8).$$

$$\phi_{LL} = \cos(90^\circ + \alpha) = -\sin\alpha \quad ..(5.3.2.9).$$

For a vector amplitude $V/\sqrt{3}$

$$V_I = V\cos\alpha \quad .. \quad ..(5.3.2.10a)$$

$$V_{II} = \frac{V}{2}\cos\alpha - \frac{\sqrt{3}}{2}V\sin\alpha \quad .. \quad ..(5.3.2.10b)$$

$$V_{III} = -\frac{V}{2}\cos\alpha - \frac{\sqrt{3}}{2}V\sin\alpha \quad .. \quad ..(5.3.2.10c)$$

5.4 Lead-Vector Concept:

One of the most important developments in electrocardiography is the lead vector concept. A relationship between the heart dipole source and the resultant surface potentials can be established by the lead vector concept. The initial assumptions are :

a) Body is heterogeneous, resistive and linear.

b) Electrical activity of heart can be represented by a dipole fixed in position but variable in magnitude and orientation.

c) Actual body shape is to be used.

Let us suppose that heart dipole current source has a vector magnitude $\bar{p}$. For the medium assumed above, the potential at an arbitrary surface point Q, relative to a chosen reference, must be proportional to the magnitude of $\bar{p}$. The proportionality must exist also with respect to the component of $\bar{p}$. If the rectangular components are p_x, p_y, p_z, then the potential ϕ_Q at Q, can be expressed as

$$\phi_Q = c_x p_x + c_y p_y + c_z p_z \quad .. \qquad ..(5.4.1).$$

where c_x, c_y, c_z are proportionality constants which are valid for condition specified. A some-what simpler notation results if c_x, c_y, c_z are considered to be components of a vector c which is known as the "lead vector".

This gives

$$\phi_Q = \bar{c} \cdot \bar{p} \qquad .. \qquad .. \quad (5.4.2)$$

The value of c depends on the location of Q and the equivalent heart dipole, the body shape, the electrical characteristics of the body tissues, etc. If three different surface points Q_i are chosen and their respective values of c_i are determined, then the dipole moment $\bar{p}$ can be found from a specific simultaneous potential measurements at each point. For example we might measure

$$V_I = \bar{A} \cdot \bar{p} \qquad .. \qquad .. \quad (5.4.3a)$$

$$V_{II} = \bar{B} \cdot \bar{p} \qquad .. \qquad .. \quad (5.4.3b)$$

$$V_{III} = \bar{C} \cdot \bar{p} \qquad .. \qquad .. \quad (5.4.3c)$$

$$V_{BR} = \bar{D} \cdot \bar{p} \qquad .. \qquad .. \quad (5.4.3d)$$

where V_{BR} is the back to right arm lead. The value of $\bar{p}$ can be found since (5.4.3) includes three independent linear equations. From the above equations p_x and other compoents can be evaluated. For example the value of p_x can be written with $V_I + V_{III} = V_{II}$ as;

$$p_x = \frac{1}{\Delta} \left[V_I (B_y D_z - B_z D_y) + V_{II}(A_z D_y - A_y D_z) + V_{BR} (A_y B_z - A_z B) \right]$$

where $\Delta = A_x (B_y D_z - B_z D_y) + A_y (B_z D_x - B_x D_z) + A_z (B_x D_y - B_y D_x)$

or $\Delta = \bar{A} \cdot \bar{B} \times \bar{D}$

In current ECG practice, the positive x direction is chosen as right to left, positive z front to back and positive y is top to bottom. The Co-ordinate system is thus an orthogonal right handed system. The frontal plane corresponds to xy, the horizontal plane is xz and saggital plane is yz.

It is possible to determine the co-efficients $\bar{A}, \bar{B}, \bar{C}, \bar{D}$ of equation (5.4.3) from the measurements in an electrolytic tank model of the human torso.

Lead Fields and Reciprocity:

For a given electrode pair on the body surface, we could map the behavior of the lead vector as a function of position of the heart dipole, thus generating a'lead vector field'. In view of the assumption of homogeneity it might be expected that this field would be mathematically 'regular'. In this connection the lead field, as defined by McFee & Johnston, plays an important role in conceptualizing (and computing) lead vectors and also permits a generalization to the lead tensor and compound lead fields. The lead field arises from the general reciprocity theorem which is developed first.

Let us consider an arbitrarily shaped volume conductor V with conductivity σ (x, y, z), where σ is assumed to be a well-behaved function of position. If we further assume

that the medium satisfied ohm's law, then

$$\bar{J} = \sigma \bar{E} \qquad .. \qquad .. (5.4.4).$$

where $\bar{J}$ is the current density and $\bar{E}$ is the electric field intensity. Since the electric field is conservative it may be derived from a scalar potential ϕ so that

$$\bar{E} = - \nabla \phi \qquad .. \qquad .. (5.4.5).$$

and hence

$$\bar{J} = - \sigma \nabla \phi \qquad .. \qquad .. (5.4.6).$$

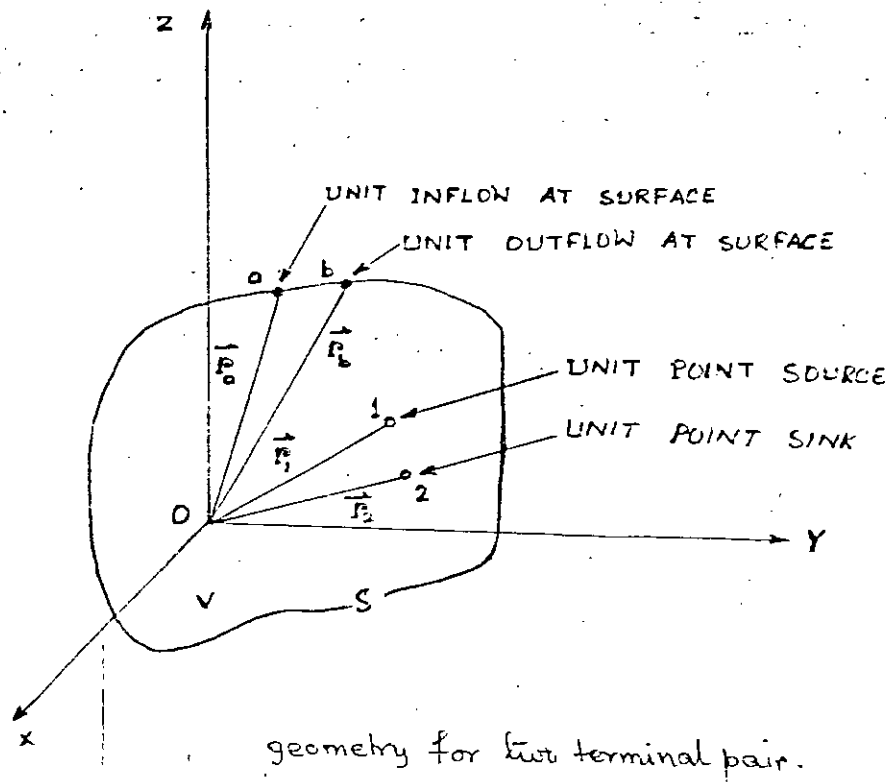
Let us designate the volume sources of current I_v ; then from (5.4.6) we must have

$$-I_v = \nabla \cdot \bar{J}_i = + \nabla \cdot (\sigma \nabla \phi) \qquad .. (5.4.7).$$

Consider an arbitray volume V bounded by the surface S, as illustrated in the figure 5.4.a. Let ϕ_1 and ϕ_2 be any two scalar fields defined in V. Then if σ is the conductivity within V, the following vector identities must be satisfied.

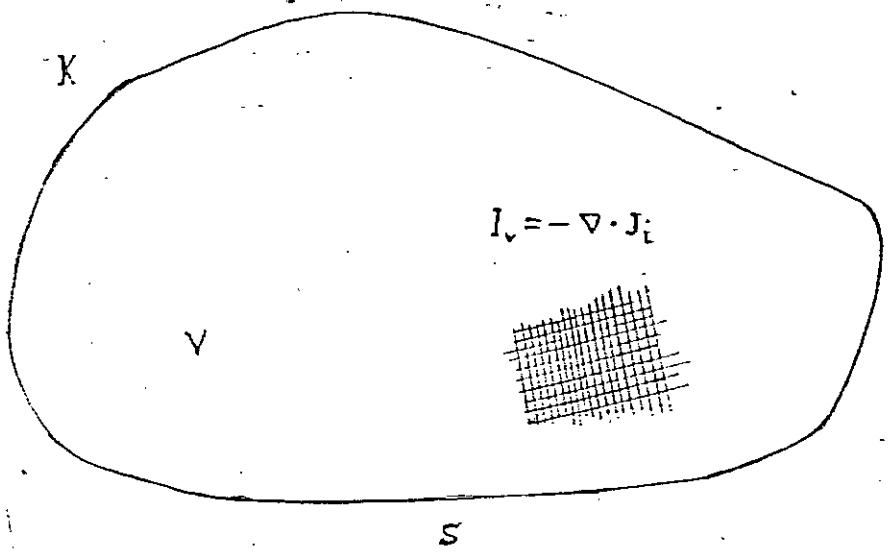
$$\nabla \cdot \phi_1 (\sigma \nabla \phi_2) = \phi_1 \nabla \cdot (\sigma \nabla \phi_2) + \sigma \nabla \phi_1 \cdot \nabla \phi_2 \qquad .. \quad (5.4.8)$$

$$\nabla \cdot \phi_2 (\sigma \nabla \phi_1) = \phi_2 \nabla \cdot (\sigma \nabla \phi_1) + \sigma \nabla \phi_1 \cdot \nabla \phi_2 \qquad .. \quad (5.4.9)$$



geometry for two terminal pair.

FIG - 5.4.b



Torso Volume geometry.

FIG - 5.4a

If we subtract (5.4.9) from (5.4.8) and integrate term by term over the volume V, and use the divergence theorem, we get

$$\int_S \phi_1 (\sigma \nabla \phi_2) \cdot d\bar{s} - \int_S \phi_2 (\sigma \nabla \phi_1) \cdot d\bar{s}$$
$$= \int_V \left[\phi_1 \nabla \cdot (\sigma \nabla \cdot \phi_2) - \phi_2 \nabla \cdot (\sigma \nabla \phi_1) \right] dv$$

... ... (5.4.10).

Now let us assume that ϕ_1 is the scalar potential in V due to the sources within V specified by $I_v = - \nabla \cdot \bar{J}_i$. We shall further assume that ϕ_2 solely arises from currents caused to flow across the surface S; we designate the latter by K (amperes per unit area). We require

$$\int K ds = 0 \qquad (5.4.11).$$

since, the current is solenoidal. The following constraints must then be satisfied by ϕ_1 and ϕ_2 :

$$\nabla \cdot (\sigma \nabla \phi_1) = -I_v \qquad .. \qquad (5.4.12)$$

$$\sigma \nabla \phi_2 \cdot d\bar{s} = K ds \text{ (taking positive } K \text{ as in-flow)..} \qquad (5.4.13)$$

When I_v is the source, no currents crosses the boundary surface; hence

$$\nabla \phi_1 \cdot d\bar{s} = 0 \qquad .. \qquad (5.4.14)$$

For the sources K at the surface, the current must be solenoidal everywhere in V; hence

$$\nabla \cdot (\sigma \nabla \phi_2) = 0 \qquad .. \qquad (5.4.15)$$

Using (5.4.11) to (5.4.15) the equation (5.4.10) becomes

$$\int_S \phi_1 \, K ds = \int_V \phi_2 \, I_v \, dV \qquad .. \qquad (5.4.16)$$

which is the desired form of reciprocity theorem.

Let us consider a specific I_v which consists of a point current source of magnitude Io at $\bar{r}_1$ and a point sink of equal magnitude at $\bar{r}_2$, where $\bar{r}_1$ and $\bar{r}_2$ are the respective position vectors. Thus

$$I_v = \left[\delta_v(\bar{r}_1 - \bar{r}) - \delta_v(\bar{r}_2 - \bar{r}) \right] I_o \qquad (5.4.17)$$

and $\delta_v(\bar{r})$ is a three-dimensional delta function. Consider further that ϕ_2 arises from specific distribution K which is that of a surface electrode a that provides an inflow of a unit current while electrode b has an outflow of a unit current. If the position vector of a is $\bar{r}_a$ and that of b is $\bar{r}_b$, then from very small electrodes, we have

$$K = \delta_s (\bar{r}_a - \bar{r}) - \delta_s(\bar{r}_b - \bar{r}) \qquad (5.4.18)$$

where δ_s is a two-dimensional delta function. If (5.4.17) and (5.4.18) are substituted in (5.4.16), then we get

$$I_o[\phi_2(\bar{r}_1) - \phi_2(\bar{r}_2)] = \phi_1(\bar{r}_a) - \phi_1(\bar{r}_b) \quad \ldots \quad (5.4.19)$$

In words, (5.4.19) signifies that the difference of potential between two surface points a and b [that is the lead voltage $\phi_1(\bar{r}_a) - \phi_1(\bar{r}_b)$] due to a unit source of current(I_o=1)supplied internally between 1 and 2 equals the difference of potential between these same source points (1 and 2) due to a unit current applied between a and b. The geometry for this two terminal pair is shown in fig. 5.4.b.(9)

If the separation of $\bar{r}_1$ and $\bar{r}_2$ approaches zero, the source described by (5.4.17) becomes a dipole with moment $I_o(\bar{r}_1 - \bar{r}_2)$. The value of $\phi_2(\bar{r}_1)$ can be described interms of field at $\bar{r}_2$ by means of Taylor series expansions as follows.

$$\phi_2(\bar{r}_1) = \phi_2(\bar{r}_2) + \nabla\phi_2 \cdot (\bar{r}_1 - \bar{r}_2) + (\text{higher order terms}) \quad (5.4.20)$$

Thus if we let $(\bar{r}_2 - \bar{r}_1)$ approach zero then a dipole moment $I_o(\bar{r}_1 - \bar{r}_2) = \bar{m}_o$ is created and the potential $[\phi_1(\bar{r}_a) - \phi_1(\bar{r}_b)]$ can be written as follows :

$$\phi_2(\bar{r}_1) - \phi_2(\bar{r}_2) = \nabla\phi_2 \cdot (\bar{r}_1 - \bar{r}_2) = \phi_1(\bar{r}_a) - \phi_1(\bar{r}_b) = V_{ab} \quad (5.4.21a)$$

or $$\nabla\phi_2 \cdot \bar{m}_o = V_{ab} \quad (5.4.21b)$$

In (5.4.21), ϕ_2 is the potential due to "reciprocally energizing" the pickup leads ab. The scalar field ϕ_2 has been designated the "lead field" by McFee and Johnston. By comparison with earlier nomenclature we see that ϕ_2 is the lead vector (field), while $\bar{m}_o$ corresponds to the "heart vector" and Vab is the lead voltage. The lead vectors are physically realizable as they are derived as the gradient of scalar field which satisfy Laplace equation.

If we apply the relation $\phi_2(r_1) - \phi_2(r_2) = \phi_1(r_a) - \phi_1(r_b)$, $\phi_2(r)$ can be determined for three independent leads by means of an electrolytic tank. With this information, $\nabla\phi_2$ can be calculated along with measured values of Vab the effective heart vector $\bar{m}_o$ can be found in the fixed dipole electrocardiographic model. Another use would be the determination of the current density due to external active leads. Thus with uniform spheriaal conductor with unit current inflow at $\bar{r}_a$ and outflow at $\bar{r}_b$ we have a potential field ϕ_2 set up which according to (5.4.21.b) causes a current density of

$$\bar{J} = -\sigma\nabla\phi_2 = -\sigma\left[\frac{(V_{ab})_x}{m_x}\bar{a}_x + \frac{(V_{ab})_y}{m_y}\bar{a}_y + \frac{(V_{ab})_z}{m_z}\bar{a}_z\right] \qquad (5.4.22)$$

where m_x, m_y, m_z are the x, y, z components of $\bar{m}_o$ and $(V_{ab})_x$, $(V_{ab})_y$, $(V_{ab})_z$ are the lead voltages by the respective components.

For a volume distribution of dipoles $\bar{J}_i$, then lead fields Vab is given by following relation (assuming $\bar{m}_p$ equals to $\bar{J}_i\ dV$.)

$$Vab = \int_v \nabla \phi_2 \cdot \bar{J}_i\ dV \qquad (5.4.23)$$

By utilizing the lead field concept, we can consider a lead system that is made up of more than two electrodes which are connected together by a suitable resistance network with two terminal output.

The lead field is also useful in indicating the relative contribution to the total lead voltage from each dipole elements of a distribution of sources.

5.5 Orthogonal Lead System:

If we analyse the conventional limb lead it is seen that the expressions for standard leads I and II has the following expression, with the assumption that heart can be modeled by a central dipole,

$$V_I = A_x P_x \qquad .. \qquad .. \qquad (5.5.1).$$

$$V_{II} = B_x P_x + B_y P_y \qquad .. \qquad .. \qquad (5.5.2)$$

The activation process that takes place in heart is three dimensional but the electrocardiographic leads are

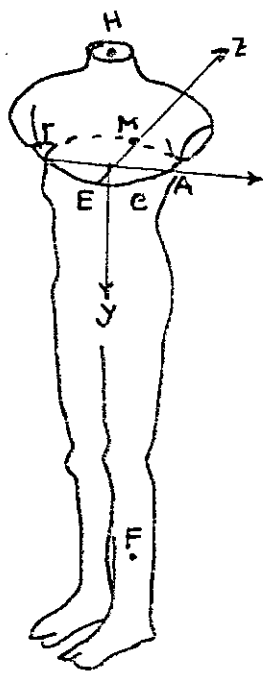
placed on the frontal plane xy plane. Naturally the heart vector components of (5.5.1) and 5.5.2) do not represent the physical situation satisfactorily. In order to over come the short-comings of the standard limb leads another lead system is devised. It uses the concept of lead fields. If we desire to setup three lead fields, each of which is uniform in the heart region and where the gradients are mutually orthogonal, then such a lead system is known as "orthogonal lead system". This system directly measures the x, y, and z components of heart vector. More excactly it measures the linear superposition of the distributed x, y, z components of $\bar{J}_i dV$ as evident from (5.4.23).

In the design of orthogonal lead system a human torso model(a plastic model filled with electrolyte) is utilized so that the lead voltages can be calibrated with respect to active artificial dipole source. Active dipole source, oriented in either the x, y, or z direction are placed at the approximate anatomical centre of heart within torso model. One now seeks a lead which responds to the x dipole but not y or z. This corresponds to the realizing of c_y and c_z components (lead vector) equals to zero. In three dimensional space, this lead vector is then in x direction and yields the x component of heart vector. Similarly, y and z leads are found that respond to the y and z component of heart dipole. If the active dipole is displaced from anatomical centre, the lead system will also give satisfactory result.

Because of the linearity of the system, it is not necessary that the x component be obtained from a single electrode pair. It is possible to combine the potentials at three or more points on the body surface through resistance networks such that across the output terminals the voltage is proportional to p_x and independent of p_y and p_z. Through this mechanism the selection of electrode location is quite satisfactory and it minimizes the sensitivity to imprecise electrode positioning and to body shape. By suitable resistance networks it is also possible to adjust the lead-vector magnitudes to be equal. This gives rise to what is designated as corrected orthogonal lead system. Such a system is shown in figure 5.5.a. is known as Frank lead system(1). The number of electrodes used here is seven rather then a theoretical minimum of four.

In the SVEc III, system fourteen electrodes are used. The translation of dipole should cause no change in lead voltage and both Frank and SVECIII systems performs quite satisfactorily in this respect.

The outputs of the x, y, and z orthogonal leads are independent and in visualizing the three signals, they may be combined "vectorially" to form the hear vector. The tip of the heart vector contains all the informations as the component signals. The three lead signals themselves are referred to as scalar records while their composition into vector constitutes the subject of Vectorcardiography. The trajectory of the vector tip is



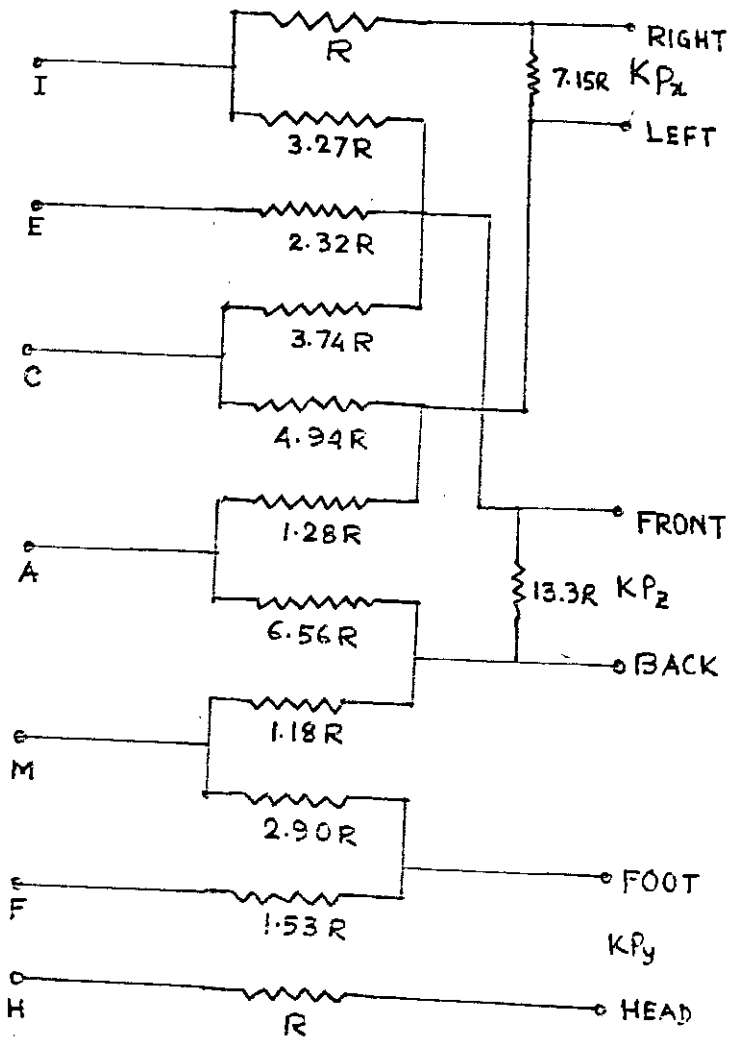


FIG 5.5a.

THE FRANK LEAD SYSTEM WITH ASSOCIATED RESISTOR NETWORK.

referred to as vector loop and since ECG is cyclical, the vector loop is closed.

One interesting aspect of vectorcardiography is that, having determined the heart vector, one can project this on any lead vector, thereby synthesizing other lead outputs.

From a clinical stand point, essentially all clinical information contained in the 12- standard system can also be found in the corrected orthogonal lead system provided lead resolution is used in doubtful cases. The use of lead resolution appears to improve the diagnostic ability of the clinician since the records are displayed in standard form. Even though no information is created by lead resolution, the ability to recognize patterns appears to be enhanced by this operation.

In electrocardiography the main interest is in ventricular activation (QRS) and in vector cardiography it forms a closed loop and lies, in most cases, in a plane in space. The projection puts the vector ECG in a form which is independent of standard x, y, and z axes and rather more closely related to the physical 'axis(of the heart itself). Such projection is known as "polar projection". That is, since the plane of the vector loop should rotate with the heart, the polar ECG projection should be approximately independent of the orientation of (physical) heart in the chest cavity. Since the

physical orientation of the normal healthy heart is a variable, this procedure, at least in principle, removes this factor from the ECG.

On the basis of these ideas, "normalized electrocardiogram" is defined by McFee, by specific procedure of cordinate axes rotation. These are chosen so that deflections in one lead are a maximum, are a minimum in second (this is normal to the plane of the loop) and are equally negative and positive in the third lead. The justification of these technique depends on validity of the dipole model.

5.6 The Dipole Hypothesis:

In the previous discussions about orthogonal lead system, axis rotation and for vectorcardiography we assumed that the heart model can be represented by a dipole fixed in position but free to change its direction and magnitude. There are some evidence that the dipole representation is a satisfactory one.

One of the evidence that dipole representation is satisfactory is seen in the evaluation of resolved leads. If, indeed, arbitray leads are obtainable as linear combinations of only the x, y and z components of an orthogonal lead system, then the dipole basis of cardiac activity is established. Typical computations shows that there is a peak to peak dicrepencies. In a study of 19 normals as a percentage of average of

actual and synthesized signal the discrepencies is about 23 percent with SD of ± 7%.

A comparison of surface potential with true dipole excitation in torso model and equipotentials plot on the surface of the torso shows that the two results are in good agreement during ventricular activation. This comparison was made by Frank. But in recent experiments it is seen that there is a deviation from dipolarity of the heart model with heart disease.

Surface potential maps of human and dogs display a patterns of changing complexity that appear dipolar at certain times during the QRS and nondipolar at other times. A measure of nondipolarity is the appearance of more than one maximum or minimum (or both). All investigations show evidence of multipolarity during some portion of the QRS.

The above study of the dipolarity of heart is a fairly direct one. Another approach, which is some what inferential, is that of "cancellation". To explain this technique we note that the potential is $\phi = \bar{c} \cdot \bar{p}$ and using the linear combination of components we can write

$$
\begin{aligned}
V_1(t) &= C_{11} P_1(t) + C_{12} P_2(t) + C_{13} P_3(t) \\
V_2(t) &= C_{21} P_1(t) + C_{22} P_2(t) + C_{23} P_3(t) \\
V_3(t) &= C_{31} P_1(t) + C_{32} P_2(t) + C_{33} P_3(t)
\end{aligned}
\qquad \ldots \quad \ldots \quad (5.6.1)
$$

where p_1, p_2, p_3 are the three orthogonal components of the assumed heart vector; V_1, V_2, V_3 are the lead voltages at three arbitrary (and assumed independent)leads; and c_{ij} are geometrical constants. Now the above equations can be used to solve for p_1, p_2, p_3 in terms of V_1, V_2 and V_3, giving

$$\begin{aligned} P_1(t) &= \alpha_{11} V_1(t) + \alpha_{12} V_2(t) + \alpha_{13} V_3(t) \\ P_2(t) &= \alpha_{21} V_1(t) + \alpha_{22} V_2(t) + \alpha_{23} V_3(t) \\ P_3(t) &= \alpha_{31} V_1(t) + \alpha_{32} V_2(t) + \alpha_{33} V_3(t) \end{aligned} \qquad (5.6.2)$$

If we now consider an arbitrary fourth lead $V_4(t)$, then it can be written as

$$V_4(t) = c_{41} P_1(t) + c_{42} P_2(t) + c_{43} P_3(t) \qquad (5.6.3)$$

and substituting (5.6.2) for $P_1(t)$ and $p_2(t)$ and $p_3(t)$ in (5.6.3), we have

$$V_4(t) = A_1 V_1(t) + A_2 V_2(t) + A_3 V_3(t) \qquad (5.6.4)$$

where A_1 A_2 and A_3 are geometrical constants and independent of time. Thus a necessary condition for electrical activity of heart to be accounted for by a dipole generator is that any arbitrary lead be a linear contribution of any other 3 linearly independent leads. Equation (5.6.4) is true at any instant of time; it is an identity.

In the "cancellation" experiments three arbitrary leads are led into amplifiers with variable gain, summed, and subtracted from a fourth arbitrary lead. If the difference can be

made essentially zero through an appropriate adjustment of the gain controls, then cancellation has been effected and the results considered a substantiation of the dipole hypothesis. But "cancellation" is shown to be an impercise technique in many experiments and that good cancellation can be effected even in studies where sources are clearly non-dipolar.

Determination of souface potential from current dipoles has already been discussed and it is seen that dipole approximation is fair.

5.7 Multipole Theory:

From the experimental result it is clear that the dipole representation of electrical activity of the heart, is in general, too crude. The actual heart source are specified by a dipole moment density function $\bar{J}_i$. This function has the dimnsion of dipole moment per unit volume and is space and time dependent. A multipole theory is applied to such arbitrary source distribution. In this case, nondipole terms in a multipole expansion provides a convenient additive correction to the conventional heart source model represented by dipole. In addition it provides a useful mechanism for evaluating general properties of electrocardiographic source.

Let us consider a infinite region of uniform conductivity σ which contains an arbitrary distribution of current source $\bar{J}_i$. The current source density ($I_v = -\nabla\cdot\bar{J}_i$) is located at (ξ, η, ζ). The contribution to the potential at an arbitrary point p (x, y, z) due to the current source $I_v\, dV$ is given by

$$d\phi = \frac{1}{4\pi\sigma}\,\frac{I_v\, dv}{R} \qquad (5.7.1)$$

The geometry is illustrated in the figure 5.7.a.

$$d\phi = \frac{1}{4\pi\sigma}\,\frac{I_v\, dv}{\sqrt{(x-\xi)^2+(y-\eta)^2+(z-\zeta)^2}} \qquad (5.7.1)$$

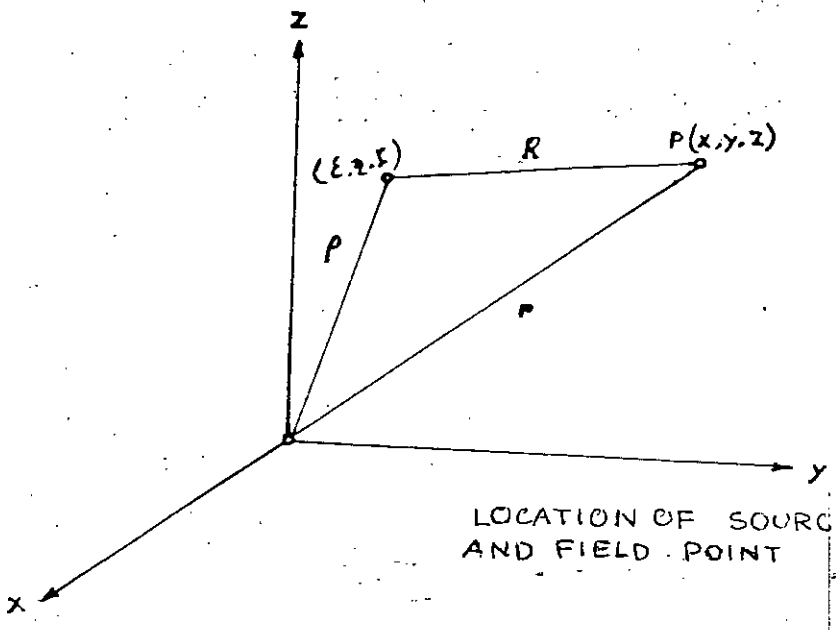
The denominator of (5.7.1) can be expanded about the origin in the powers of ξ, η, ζ by the Taylors series:

$$\frac{1}{\left[(x-\xi)^2+(y-\eta)^2+(z-\zeta)^2\right]^{1/2}} = \left\{\left[\frac{1}{r}\right] - \left[\xi\frac{\partial}{\partial x}\left(\frac{1}{r}\right) + \eta\frac{\partial}{\partial y}\left(\frac{1}{r}\right) + \zeta\frac{\partial}{\partial z}\left(\frac{1}{r}\right)\right]\right.$$
$$+\frac{1}{2}\left[\xi^2\frac{\partial^2}{\partial x^2}\left(\frac{1}{r}\right) + \eta^2\frac{\partial^2}{\partial y^2}\left(\frac{1}{r}\right) + \zeta^2\frac{\partial^2}{\partial z^2}\left(\frac{1}{r}\right) + 2\xi\eta\frac{\partial^2}{\partial x\partial y}\left(\frac{1}{r}\right)\right.$$
$$\left.\left. + 2\eta\zeta\frac{\partial^2}{\partial y\partial z}\left(\frac{1}{r}\right) + 2\zeta\xi\frac{\partial^2}{\partial z\partial x}\left(\frac{1}{r}\right)\right] \quad \cdots\cdots\right\}$$

$\cdots\quad\cdots$ (5.7.2)

The series will converge, provided that $r > a$, where a is the radius of the sphere which encloses all the volume source.

Now putting (5.7.2) in (5.7.1) and integrating with respect to (ξ, η, ζ), through out the entire volume source



LOCATION OF SOURCE POINT AND FIELD POINT

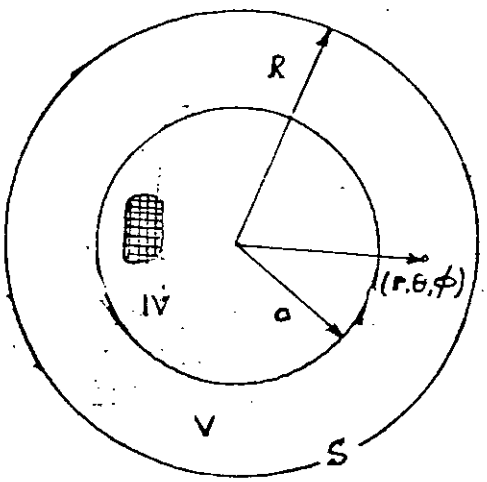


Fig 5.7a

GEOMETRY FOR MULTIPOLE EXAMPLE

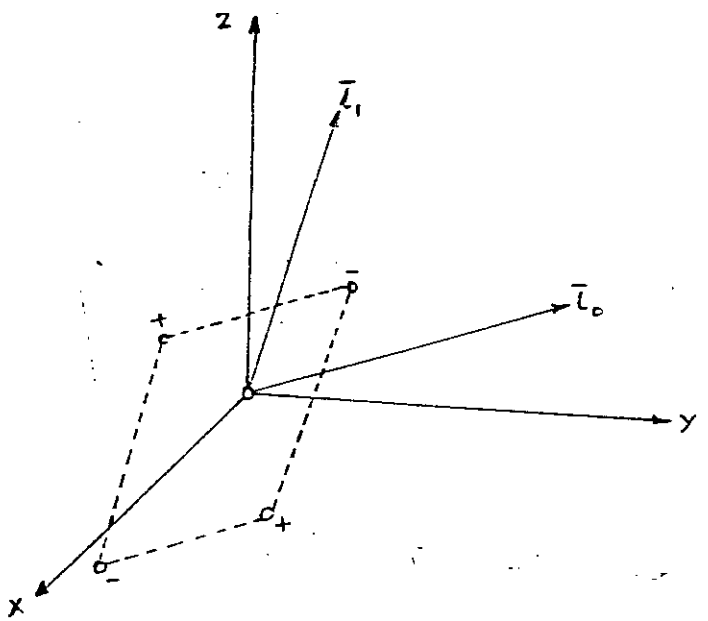


Fig - 5-7-b.
GENERALIZED QUADRUPOLE

distributions yields the total potential as the sum of terms each of which corresponds to the terms of (5.7.2).

$$\text{Thus} \quad \phi_p = \frac{1}{4\pi\sigma} \int \frac{I_v \, dv}{R} \qquad \cdots \qquad (5.7.3)$$

$$\text{or} \quad \phi_p = \sum_{n=0}^{\infty} \phi_n \qquad \cdots \qquad (5.7.4)$$

where ϕ_n is the total contribution from the terms whose radial dependence is $r^{-(n+1)}$. The first term in (5.7.4) is ϕ_o and is given by

$$\phi_o = \frac{I^{(o)}}{4\pi\sigma r} \qquad (5.7.5)$$

where $I^{(o)} = \int I_v dV$ and is the net algebraic sum of all the current source. This term is zero since there is no net source due to the electrophysiological generators.

The next term of (5.7.4) is dominant when the net charge is zero and is given by

$$\phi_1 = -\frac{1}{4\pi\sigma} I^{(1)} \cdot \nabla\left(\frac{1}{r}\right) \qquad \cdots \qquad (5.7.6)$$

where $I_x^{(1)} = \int I_v \xi \, dv \; ; \; I_y^{(1)} = \int I_v \eta \, dv \; ; \; I_z^{(1)} = \int I_v \zeta \, dv \quad \cdots (5.7.7)$

That is $I^{(1)}$ is a vector dipole moment whose rectangular components are given in (5.7.7). From (5.7.6) and (5.7.7) we can say that the potential due to any distribution whose net charge is zero may be approximated at large distances by the potential of a dipole located at the origin whose components are given above.

In similar way the partial potential ϕ_2 arises from a "quadrupole"

$$\phi_2 = \frac{1}{4\pi\sigma}\left[\frac{I_{xx}^{(2)}}{2}\frac{\partial^2}{\partial x^2}\left(\frac{1}{r}\right) + I_{xy}\frac{\partial^2}{\partial x\partial y}\left(\frac{1}{r}\right) + \frac{I_{yy}^{(2)}}{2}\frac{\partial^2}{\partial y^2}\left(\frac{1}{r}\right) + I_{yz}^{(2)}\frac{\partial^2}{\partial y\partial z}\left(\frac{1}{r}\right) + \frac{I_{zz}^{(2)}}{2}\frac{\partial^2}{\partial z^2}\left(\frac{1}{r}\right) + I_{zx}^{(2)}\frac{\partial^2}{\partial x\partial z}\left(\frac{1}{r}\right)\right] \quad (5.7.8)$$

The components of the quadrupole are

$$I_{xx}^{(2)} = \int I_v \xi^2 dv \;;\; I_{yy}^{(2)} = \int I_v \eta^2 dv \;;\; I_{zz}^{(2)} = \int I_v \zeta^2 dv$$
$$I_{xy}^{(2)} = \int I_v \xi\eta\, dv \;;\; I_{yz}^{(2)} = \int I_v \eta\zeta dv \;;\; I_{zx}^{(2)} = \int I_v \zeta\xi dv \quad (5.7.9)$$

and are known as the components of a tensor of second rank and ϕ_2 is the quadrupole term,

The multipole terms of higher order determined from subsequent terms of the expansion (5.7.3) involves successively higher inverse power of r as noted.

Let us derive the expression for potential of general multipole of nth order having moment of $I^{(n)}$.

The potential due to a single positive point current source is given by [zero order having moment $I^{(o)}$]

$$\phi_o = \frac{1}{4\pi\sigma r} \text{ (assuming source at origin)} \quad (5.7.10)$$

Then for a positive and negative source which are displaced by an infinitesimal amount in the arbitrary $\bar{\ell}_o$ direction, the potential is given by

$$\phi_1 = -\frac{1}{4\pi\sigma} I^{(1)} \frac{\partial}{\partial l_0}\left(\frac{1}{r}\right) \qquad (5.7.11)$$

such a source is a dipole whose axis is $\bar{l}_0$.

Now if two dipoles with opposite orientations are displaced infinitesimally in the direction $\bar{l}_1$ then

$$\phi_2 = \frac{1}{4\pi\sigma} \frac{I^{(2)}}{2!} \frac{\partial^2}{\partial l_0 \partial l_1}\left(\frac{1}{r}\right) \qquad (5.7.12)$$

Then by induction the potential of a general multipole of nth order is given by

$$\phi_n = \frac{1}{4\pi\sigma} \frac{(-1)^n}{n!} I^{(n)} \frac{\partial^n}{\partial l_0 \partial l_1 \cdots \partial l_{n-1}}\left(\frac{1}{r}\right) \qquad (5.7.13)$$

For a quadrupole source, the geometry is illustrated in fig. 5.7.b.

The magnitude of a multipole of order (n+1), $I^{(n+1)}$ is defined interms of nth order multipole by the relationship[2]

$$I^{(n+1)} = (n+1) I^{(n)} l_n \qquad (5.7.14)$$

where $l_n \to 0$ and $I^{(n)} \to \infty$, such that their product is finite. It can be noted that for n = o the conventional definition for the dipole magnitude results.

The volume integral of (5.7.3) with the substitution of (5.7.4) can be interpreted as the contribution to the total field from an equivalent multipole component source The finite

component of each multipole source produces field which is identical with actual sources for $r > a$. The terms of equation (5.7.8) each of which can be treated as quadrupole source.

The quadrupole terms in (5.7.9) contains six undetermined parameters. It is seen that one term is redundent. For this let us consider a single dipole source; $I^{(1)}$, for which expression for ϕ_1 reduces to

$$\phi_1 = -\frac{1}{4\pi\sigma} I^{(1)} \nabla\cdot\left(\frac{1}{r}\right)$$

$$\phi_1 = -\frac{1}{4\pi\sigma} I^{(1)} \left[\alpha_0 \frac{\partial}{\partial x}\left(\frac{1}{r}\right) + \beta_0 \frac{\partial}{\partial y}\left(\frac{1}{r}\right) + \gamma_0 \frac{\partial}{\partial z}\left(\frac{1}{r}\right)\right] \qquad (5.7.15)$$

In (5.7.15) α_0 , β_0 , γ_0 are direction cosines of $\bar{I}_1^{(1)} = I^{(1)}\bar{a}_{l_0}$ where $\bar{a}_{l_0} = \alpha_0 \bar{a}_x + \beta_0 \bar{a}_y + \gamma_0 \bar{a}_z$. Carrying out the differentiation of (5.7.15) gives

$$\phi_1 = \frac{1}{4\pi\sigma} \frac{I^{(1)}}{r^3} (\alpha_0 x + \beta_0 y + \gamma_0 z) \qquad (5.7.16)$$

If we put $z = r\cos\theta$, $y = r\sin\theta \sin\phi$, $x = r\sin\theta \cos\phi$, then (5.7.16) results in

$$\phi_1 = \frac{1}{4\pi\sigma} \frac{I^{(1)}}{r^2} \left[(\alpha_0 \cos\phi + \beta_0 \sin\phi) \sin\theta + \gamma_0 \cos\theta\right] \qquad (5.7.17)$$

or $$\phi_1 = \frac{1}{4\pi\sigma} \frac{I^{(1)}}{r^2} \left[(\alpha_0 \cos\phi + \beta_0 \sin\phi) P_1^1(\cos\theta) + \gamma_0 P_1^0(\cos\theta)\right] \qquad (5.7.18)$$

where the function p_n^m $(\cos\theta)$ are the associated Legendre Polynomial defined by

$$P_n^m(\cos\theta) = \sin^m\theta \frac{d^m}{d(\cos\theta)^m} P_n(\cos\theta) \qquad (5.7.19)$$

Between four constants $I^{(1)}$, α_o, β_o and γ_o in (5.7.18) there exists one relation $\alpha_o^2 + \beta^2 + \gamma_o = 1$ and there remains three arbitrary constants to be determined.

Similarly for quadrupole term we obrain the equation with characteristic $\bar{l}_o/l_o = \alpha_o \bar{a}_x + \beta_o \bar{a}_y + \gamma_o \bar{a}_z$ and $\bar{l}_1/l_1 = \alpha_1 \bar{a}_x + \beta \bar{a}_y + \gamma_1 \bar{a}_z$ and magnitude $I^{(2)}$,

$$\phi_2 = \frac{1}{4\pi\sigma} \frac{I^{(2)}}{r^3} \left[a_{20} P_2^0(\cos\theta) + (a_{21}\cos\phi + b_{21}\sin\phi) P_2^1(\cos\theta) + (a_{22}\cos 2\phi + b_{22}\sin 2\phi) P_2^2(\cos\theta) \right]. \qquad (5.7.20)$$

There are only five constants to be determined in equation (5.7.20).

In genral if the sources are confined to the region $r < r_o$, then for $r > r_o$,

$$\nabla^2\phi = 0 .$$

The following are the general expressions for the potential ϕ in equivalent series expansion;

$$4\pi\sigma\phi = \sum_{n=0}^{\infty} \sum_{m=0}^{n} \frac{1}{r^{n+1}} (a_{nm}\cos m\varphi + b_{nm}\sin m\varphi) P_n^m(\cos\theta)$$

--- --- (5.7.21)

$$4\pi\sigma\phi = \sum_{n=0}^{\infty} I^{(n)} \frac{(-1)^n}{n!} \frac{\partial^n}{\partial l_{n1}\partial l_{n2}\cdots\partial l_{nn}}\left(\frac{1}{r}\right) \quad \cdots \quad (5.7.22)$$

$$= \sum_{n=0}^{\infty}\sum_{l=0}^{n}\sum_{k=0}^{n-l} \frac{(-1)^n}{l!\,k!\,(n-l-k)!} C_{nlk} \frac{\partial^n}{\partial x^l \partial y^k \partial z^{n-l-k}}\left(\frac{1}{r}\right) \quad \cdots\cdots (5.7.23)$$

In the general expressions for multipole expansions P_n^m is an associated Legendre Polynomial, and

$$\frac{\partial}{\partial l_{ni}} = \alpha_{ni}\frac{\partial}{\partial x} + \beta_{ni}\frac{\partial}{\partial y} + \gamma_{ni}\frac{\partial}{\partial z} \quad \cdots \quad (5.7.24)$$

where α, β and γ are direction cosine, as stated earlier, which satisfy the relation

$$\alpha_{ni}^2 + \beta_{ni}^2 + \gamma_{ni}^2 = 1 \quad (5.7.25)$$

The n = 0 term constitute a monopole; n = 1 a dipole, n = 2, a quadrupole, etc.

It is to be noted that in representation (5.7.21) b_{no} vanishes and there are (2n+1) parameters for nth order multipole. In (5.7.22) $I^{(n)}$ may be interpreted as the magnitude of the nth order multipole, which has associated with it 3n direction cosines. However, there are n relations (5.7.25) among the direction cosines again leaving 2n+1 independent parameters, Representation (5.7.23) apparently has $\frac{1}{2}(n+1)\cdot(n+2)$ parameters. However, there is redundancy present due to the fact that for $r > 0$

$$\nabla^2\left(\frac{1}{r}\right) = \left(\frac{\partial^2}{\partial x^2} + \frac{\partial^2}{\partial y^2} + \frac{\partial^2}{\partial z^2}\right)\frac{1}{r} = 0 \qquad (5.7.26)$$

and this redundancy reduces the member of independent parameters once more to $2n+1$[8].

The multipole expansion given by (5.7.21) can be written in the following form

$$\phi(p) = \sum_{n=1}^{\infty}\sum_{m=0}^{n}\left[a_{nm}Y_{nm}^{e}(\theta\phi) + b_{nm}Y_{nm}^{o}(\theta,\phi)\right]\frac{1}{r^{n+1}} \qquad (5.7.27)$$

where

$$Y_{nm}^{e} = P_n^m(\cos\theta)\cos m\phi \qquad (5.7.28a)$$

and

$$Y_{nm}^{o} = P_n^m(\cos\theta)\sin m\phi \qquad (5.7.28b)$$

are known as even tessral harmonic (Y_{nm}^{e}) and odd tessral harmonics (Y_{nm}^{o}) respectively.

The co-efficients a_{nm} and b_{nm} can uniquely be determined by a specification of the potential over a closed surface. The potential field of an arbitrary source has the representation (5.7.27) only when the medium is infinite in extent. The effect of a finite spherical boundary, at r=R, generates additional terms which are source free, i.e., solutions of the Laplace equation. The total potential consists of a superposition of

both expressions, and the result is

$$\phi_p = \sum_{n=1}^{\infty} \sum_{m=0}^{n} \left(\frac{a_{nm}}{\rho^{n+1}} + \rho^n a'_{nm} \right) Y_{nm}^{e}(\theta,\phi) + \left(\frac{b_{nm}}{\rho^{n+1}} + \rho^n b'_{nm} \right) Y_{nm}^{o}(\theta\phi) \quad \text{---} \quad (5.7.29)$$

where source free terms are identified by the primed co-efficients.

By virtue of the boundary condition that $\frac{\partial \phi}{\partial \rho} = 0$ at r = R, we require that

$$a'_{nm} = \frac{n+1}{n} \frac{a_{nm}}{R^{2n+1}} \quad ; \quad b'_{nm} = \frac{n+1}{n} \frac{b_{nm}}{R^{2n+1}} \quad \cdots (5.7.30)$$

So that the surface potential is

$$\phi(R,\theta,\phi) = \sum_{n=1}^{\infty} \sum_{m=0}^{n} \frac{2n+1}{n} R^{-(n+1)} \left[a_{nm} Y_{nm}^{e}(\theta\phi) + b_{nm} Y_{nm}^{o}(\theta,\phi) \right] \quad \text{---} \quad (5.7.31)$$

Now if the left hand side is known, then we see that a_{nm} and b_{nm} are readily found by utilizing the orthogonality of the tessral harmonics.

In the electrocardiographic problem the information available is the potential field over the torso surface. It is seen that this is inadequate to determine the underlying

bioelectric sources. However, it is sufficient for the unique determination of multipole coefficients of the spherical-harmonics form given by (5.7.27); the greater the number of coefficients that can be determined, the more the aspects of the source that become known. The limitations in obtaining higher order terms of multipole coefficients lie in the higher order-inverse power of r that is involved and hence, in general, smaller signal to noise ratio at the surface[6]. From a practical stand point the multipole representation permits a quantitative discussion on the nondipolar contribution to the surface potential field. And even if some other physical model is ultimately to be employed, the mathematical formalism of the transformation of the surface data to the multipole representation may be very useful.

The multipole formalism is clearly appropriate for quantitative examination of the dipole hypothesis. For experimental purpose, by measuring the resultant potential field over the surface the multipole co-efficients of cardiac source can be evaluated.

5.8 Inhomogeneitics:

In the previous discussion we assumed that the bio-electric source was surrounded by a uniform homogeneous medium. We shall now consider a distributed source in a nonhomogenous torso composed of regions each of which is assigned a uniform

conductivity. This model will permit taking some account of the conductivity and shape of lungs, fat, muscle, bone, blood etc.

Each region is considered to have uniform conductivity and each region is assumed to be isotropic also.

The body surface in Figure 5.8.a is depicted by S_3, and this represents the external shape of the human, dog, or other vertebrate. The volume V_o is spherical and is chosen so that it completely enclose the heart; S_o is the bounding surface. The surface S_1 and S_2 represent the arbitrary shapes of internal media of differing conductivity such as the lungs, Spinal column, etc.

Now at any instant of time, the primary source consists of current generators due to electrophysiological activation of the myocardium. We shall add to this the induced surface source which arise at discontinuties within So. The latter sources occur because at such discontinuties in conductivity, a surface charge accumulates at the interface in order to satisfy the requirement that the normal component of current density be continuous. The charges so set up become a secondary source for potential field. One can combine this source with the primary current source since the expression for scalar potential due to each is of the same form.

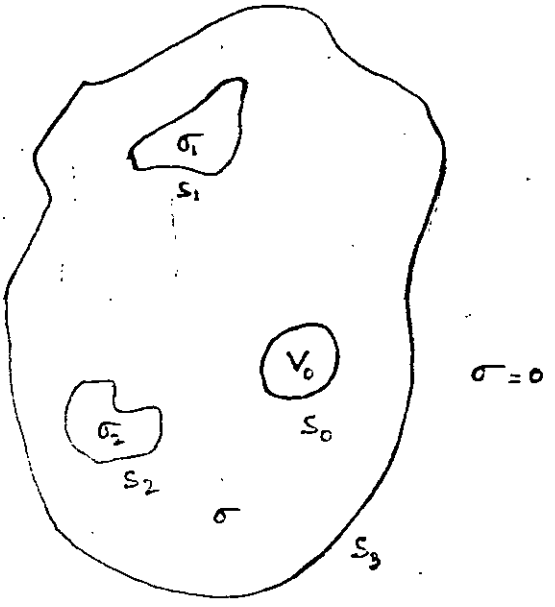


Fig 5.8.1
Volume conductor geometry.

The surface charge density ρ_s at the interface between two media of conductivity σ_a and σ_b in the presence of a normal component of applied field E_n, results in a total normal field of $E_n + \rho_s/2\epsilon$ on one side and $E_n - \rho_s/2\epsilon$ on other side[1]. Applying the continuity of normal current, $\sigma_a(E_n + \rho_s/2\epsilon) = \sigma_b(E_n - \frac{\rho_s}{2\epsilon})$ leads the relation,

$$\rho_s = 2E_n\epsilon\frac{(\sigma_b - \sigma_a)}{(\sigma_b + \sigma_a)} \qquad (5.8.1)$$

If we designate the combined primary and secondary source by a volume source distribution I_v (which includes surface sources as a limiting case) then

$$\nabla \cdot \bar{J} = -I_v \qquad (5.8.2)$$

where $\bar{J}$ is the current density. The major contribution from secondary sources within So is from the surface boundary, the intraventricular blood, which has much higher conductivity than surrounding cardiac tissue.

The potential at any point within or on S_3 can be found from the superposition of contributions from all sources, both primary and secondary. Corresponding to the aforementioned volume source I_v within S_0, induced sources appear on the discontinuity surface S_1, S_2 and S_3, which satisfy (5.8.1) and which are designated ρ_{s_1}, ρ_{s_2}, ρ_{s_3} respectively.

The potential at P_* within S_3 is then

$$\phi_p = \frac{1}{4\pi\sigma}\int_{V_0}\frac{I_v}{R}\,dv + \frac{1}{4\pi\epsilon}\left[\int_{S_1}\frac{\rho_{s1}}{R}\,ds + \int_{S_2}\frac{\rho_{s2}}{R}\,ds + \int_{S_3}\frac{\rho_{s3}}{R}\,ds\right] \quad \ldots \qquad (5.8.3)$$

where R is the distance from the field point p to an element ofsource.

Thus, the electrocardiographic problem of a distributed cardiac source in an inhomogeneous body can be replaced by one in which an equivalent source I_v^e lies in an uniform conductivity σ . It was shown in previous section that the multipole expansion co-efficients can be determined from the measurment of surface potential. Similarly if the geometry of the inhomogeneitics and their conductivities are known, then the multipole expansion of the heart source alone, I_v , can be obtained. In this case the equivalent source I_v^e can be recognized as

$$I_v^e = I_v + \sum_j I_{sj} \qquad (5.8.4)$$

where I_{sj} is a double layer on the jth surface of strength $(\sigma_b - \sigma_a)\phi$. The determination of the co-efficient of multipole expansion due to the secondary source is possible. Several investigations have been conducted to determine the importance of various inhomogeneitics. The effect of an insulating sternum and a lung of four times body resistivity for

two dimensional problem was investigated and result shows an quantitative differences with qualitative similarities.

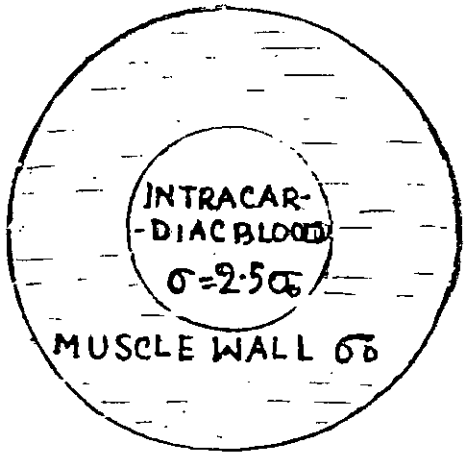
An investigation of the effect of lung and blood inhomogeneitics on the image surface for two dimensional frontal-plane model was performed with dipole as source at different heart position. The result shows an significant asymmetrical effects of inhomogeneitics. But the shape of both bioelectric source distribution and volume conductor deviate so greatly from the two dimensionality that a quantitative generalization is not possible with inhomogeneity taking into consideration. Some studies of the effect of inhomogeneitics, on lead vector, utilizing three dimensional torso models show relatively little perturbation. Some additional evidence arises from the studies where electrophysiological data were used to compute the potential distribution at the surface of torso model and the result shows that inhomogeneitics do not enter in a major way.

The study of the inverse problem in electrocardiography requirs the inclusion of lung and blood to have physiological significance.

The inhomogeneities, due to the intraventricular blood the conductivity of which is five times body conductivity, is the most influential one. The ventricle is considered to be spherical and surrounded by concentric spherical(heart)muscle as shown in fig. 5.8.b., as an idealized model[1].

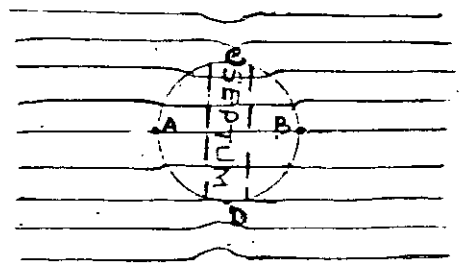
If the intracardiac blood is assumed to be a perfect conductor, then its effect on a dipole source in the muscle wall can be determined by utilizing image theory[4]. Thus, for a radially oriented dipole at the muscle-blood interface the image enhance the actual dipole, with the resultant being double in strengths, On the other hand, the tangential dipoles create an image in the opposiste direction such that cancellation occurs. For a dipole located within the muscle wall its mirror image lies within the blood region, becuase of this spatial separation one speak only approximately of enhancement of radial and reduction of tangential dipole strengths.

The effect of the high conductivity blood can be demonstrated by a lead field approach. For a uniform applied field into which a spherical ventricular blood volume is placed, the resultant flow lines at the boundary will be approximately radial as shown in figure (5.8.c.). Now an orthogonal lead system is designed precisely to set up such a uniform applied lead field in a homogeneous heart and it is seen that the resultant lead vector field is radial. The effect of intracardiac blood is the distortion of lead such that it is sensitive to radial dipoles only. Specifically, for a representation of the heart muscle as a thin concentric spherical layer surrounding the spherical blood, the field can be approximated by that which would exist in the absence of the heart muscle. In particular, a uniform field would exists within blood (Fig. 5.8.c).



IDEALIZED SPHERICAL VENTRICLE WITH CONC-
-ENTRIC SPHERICAL WALL

Fig 5-8 (b)



FLOW LINES FOR A CONDUCTING SPHERE IN A UNIFORM FIELD IN A MEDIUM OF LOWER CONDUCTIVITY.

FIG 5·8(c)

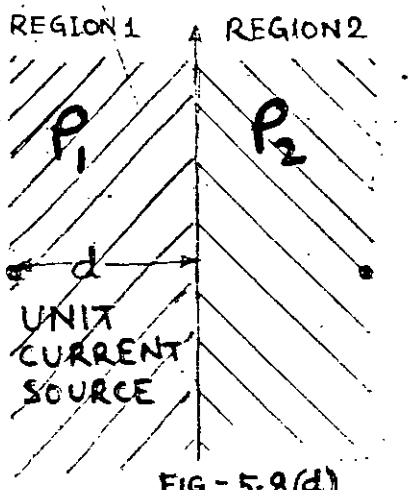


FIG - 5·8(d)

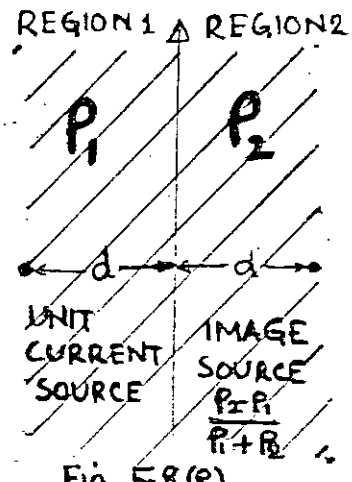


Fig 5·8(e)

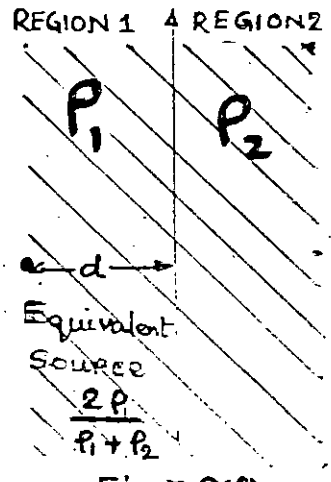


Fig 5·8(f)

IMAGE TECHNIQUE FOR POINT CURRENT SOURCE. (d) ACTUAL UNIT SOURCE NEAR INTERFACE BETWEEN TWO CONDUCTING MEDIA. (e) EQUIVALENT SOURCES IN MEDIUM OF RESISTIVITY ρ_1 VALID FOR REGION 1 ONLY. (f.) EQUIVALENT SOURCES IN MEDIUM OF RESISTIVITY ρ_2 VALID FOR REGION 2 ONLY.

Since the normal component of current density are continuous across a conductive interface, the current density within the muscle at the front and back (i.e. points A, and B in figure) is equal to that within the blood mass. Along the sides (C & D) the continuity of the tangential electric field requires that the ratio of current density in the heart muscle to that in the blood equals the respective conductivity ratio. Since the latter is, roughly, 0.4, an enhancement of radial to tangential dipole source of 2.5 results. The effect is reduced when anisotropy is considered.

If we assume the combined right and left ventricle to be represented by a spherical conductor divided in half by a septum (dotted lines in 5.8.c) then to first order the direction of the flow lines within the blood is not effected by the septum. Within a thin septum, as shown, the magnitude of the current density is the same as in the surrounding blood (continuity of the normal current); consequently, the lead field in the septum exceeds that in the external tissue by a factor $3/(1+2a)$ where a is the ratio of tissue to blood conductivity. When $a = 0.4$, then a lead vector enhancement of 1.7 occurs. On the other hand, if the direction of the applied lead vector field is parallel to the thin septum, then the electric field in the blood and in the septum are approximately same (continuity of tangential component); both are reduced from that in the external tissue by a factor of $3/(2+\frac{1}{a})$ or 0.67 for $a = 0.4$. The ratio of the two is $\frac{1}{a}$. Thus for the septum also the radial dipole component is

enhanced and tangential dipoles are attenuated.

Another inhomogeneity of importance is the muscle layer just beneath the skin. The conductivity in a direction parallel to the surface can be considered as isotropic with resistivity 280 ohm-cm, while in the direction normal to the fibers (and torso surface) the resistivity is 2,300 ohm-cm. This consideration was made by McFee and Rush. In their torso model, the heart is surrounded by a 'lung' medium, with a resistivity of 2,000 ohm-cm, bounded by the aforementioned anisotropic muscle layer; the heart is imbeded in the lung medium and of interest is the effect of the muscle layer on the lead field strength at the heart. A planar model of the muscle layer can be converted into an equivalent isotropic medium by a method of scale transformation e.g. 1cm anisotropic muscle layer with resistivity becomes equivalent to a 3 cm-thick isotropic layer with resistivity of 800 ohm-cm.

In the above model the lead field for a stratified planar conducting media is neded. This is found by the application of image principle. For example let us consider the following figures 5.8.d. 5.8.e, 5.8.f . Figure 5.8.d shows a unit point current source in a region of resistivity ρ_1 which has a planar interface with a region of resistivity ρ_2 . Then, for fields within region 1, the effect of discontinuity can be accounted for by assuming all of space at ρ_1 and an image source located at the mirror image point of strength $(\rho_2 - \rho_1)/(\rho_2 + \rho_1)$. This

condition is illustrated in figure 5.8.e. For the fields within region 2, all of the space may be considered to have a resistivity ρ_2 provided the actual source is replaced by an equivalent one at an amplitude $2\rho_1/(\rho_2+\rho_1)$. This condition is illustrated in the figure 5.8.f.

Chapter 6

Experimental Investigation of the Effect of Blood Flow Constraint on Electrocardiographic record with comments and suggestion.

In order to invetigate the effect of blood flow constraint in the limbs on ECG record, an experiment was set up with human as subject. ECG record was taken from the body surface by placing leads at different standard positions. The electrocardiographic records so obtained is analyzed and compared with normal electrocardiogram.

In the analysis of the electrocardiogram in either normal condition or abnormal condition the following features concerning the ECG record are examined [16]. These features are :

(1) Rhythm.

(2) Rate.

(3) Pwave.

(4) PR interval.

(5) QRS complex and intervel.

(6) ST segment.

(7) Twave.

(8) QT duration.

Rate of heart beat as determined from electrocardiogram

The rate of heart beat can be determined easily from the electrocardiograms, because the time interval between two successive beats is the reciprocal of the heart rate. The time interval is calculated between two successive peaks of the QRS complex amplitude.

PQ or PR interval:

The duration of time between the begining of the P wave to the begining of QRS wave is the time interval between the begining of contraction of the atrium and the begining of the contration of ventricle.

QRS Complex:

QRS complex wave occurs immediately before the begining contraction of ventricles.

T-Wave: This wave corrresponds to the repolarization of the ventricles.

QT-duration:- Contraction of ventricles lasts essentially between the begining of the Q wave and the end of the T-wave. This interval of time is called the QT interval.

Tne linear graph paper, on which ECG is recorded, has horizontal axis as time axis and the time elapsed between two consecutive vertical line is 0.04 Sec. The vertical axis represents the amplitude of the voltage and is calibrated in m.m. The amplitude of voltage is compared with standard 1mv standard signal.

The recorded electrocardiogram is shown in Appendix-A. A comparison of electrocardiogram, which was recorded with restricted blood flow, with that of normal ECG is made. In this comparison QRS complex amplitude, heart beats and QT interval are chosen as features to be investigated because these features of electrocardiogram are related with ventricular polarization and repolarization and these are the dominant characteristics of ECG.

The differences between the normal value and the values obtained for restricted blood flow condition for QRS complex amplitude, heart beats and QT interval are presented in Table-(6.1 - 6.3).

For this experiment seven sets of electrocardiogram were recorded from seven individuals. They are of different age group as recorded in the set.

DESCRIPTION OF THE LEADS USED TO RECORD ELECTROCARDIOGRAM

In the experiment to record ECG either in normal condition or in restricted blood flow condition, we used 12 standard leads of present ECG recording system. The position of attachment of four limb electrodes and six chest electrodes which constitute the 12 standard ECG system are as follows.

RA	LA	LF	RF	V1	V2	V3	V4	V5	V6
Right wrist	Left wrist	Left leg	Right leg	Chest 1	Chest 2	Chest 3	Chest 4	Chest 5	Ches 6

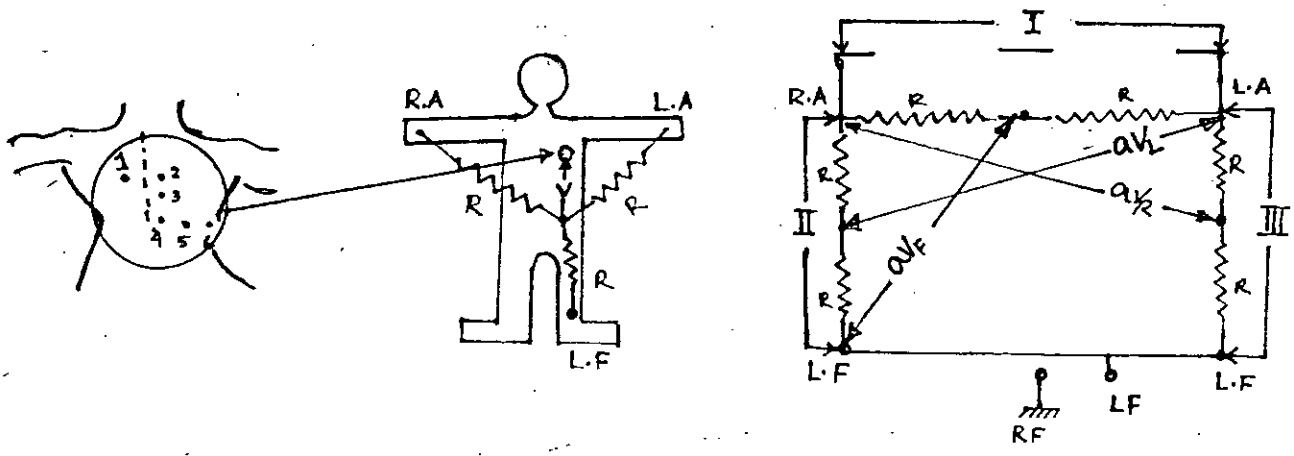


Fig-6. electrode placement and the formation of 12 standard leads, I, II, III, aV_R, aV_L, aV_F, V_1, V_2, V_3, V_4, V_5, V_6 respectively.

From the above figure the electrode placement and their formation of 12 standards lead show that only 10 electrodes are used to obtain 12 leads.

QRS COMPLEX AMPLITUDE MEASUREMENTS

In electrocardiogram QRS(complex) wave occurs immediately before the contraction of ventricle.

Amplitude of QRS complex is measured from the peak of R wave to the peak of s wave. The vertical axis of the recorded ECG represents voltage amplitude and is calibrated in m. m.

The values of the QRS complex amplitude for different persons are shown in figures 6.1 - 6.7.

Table 6-1 shows the difference of QRS complex amplitude from the normal value for differrnt persons.

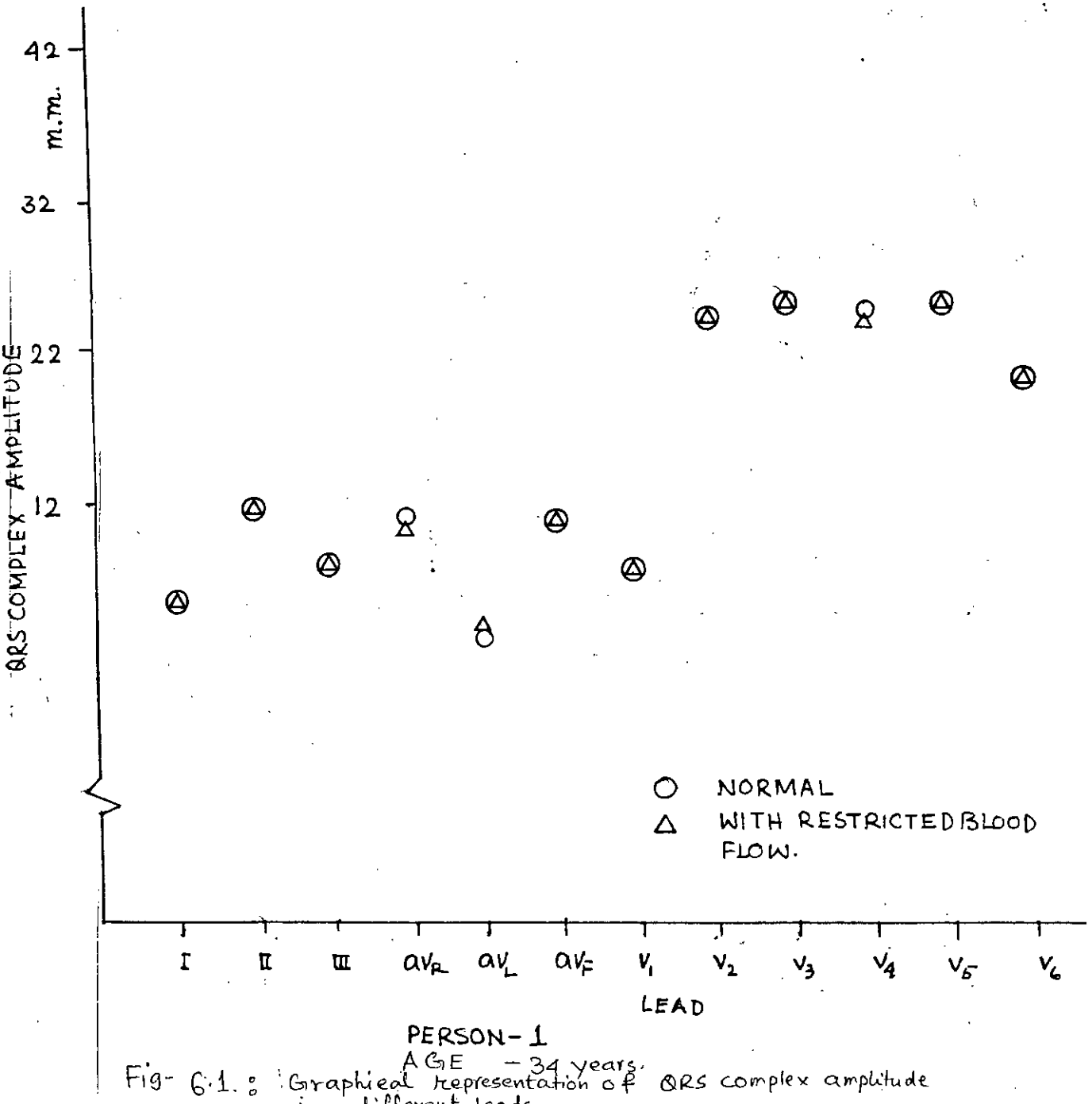
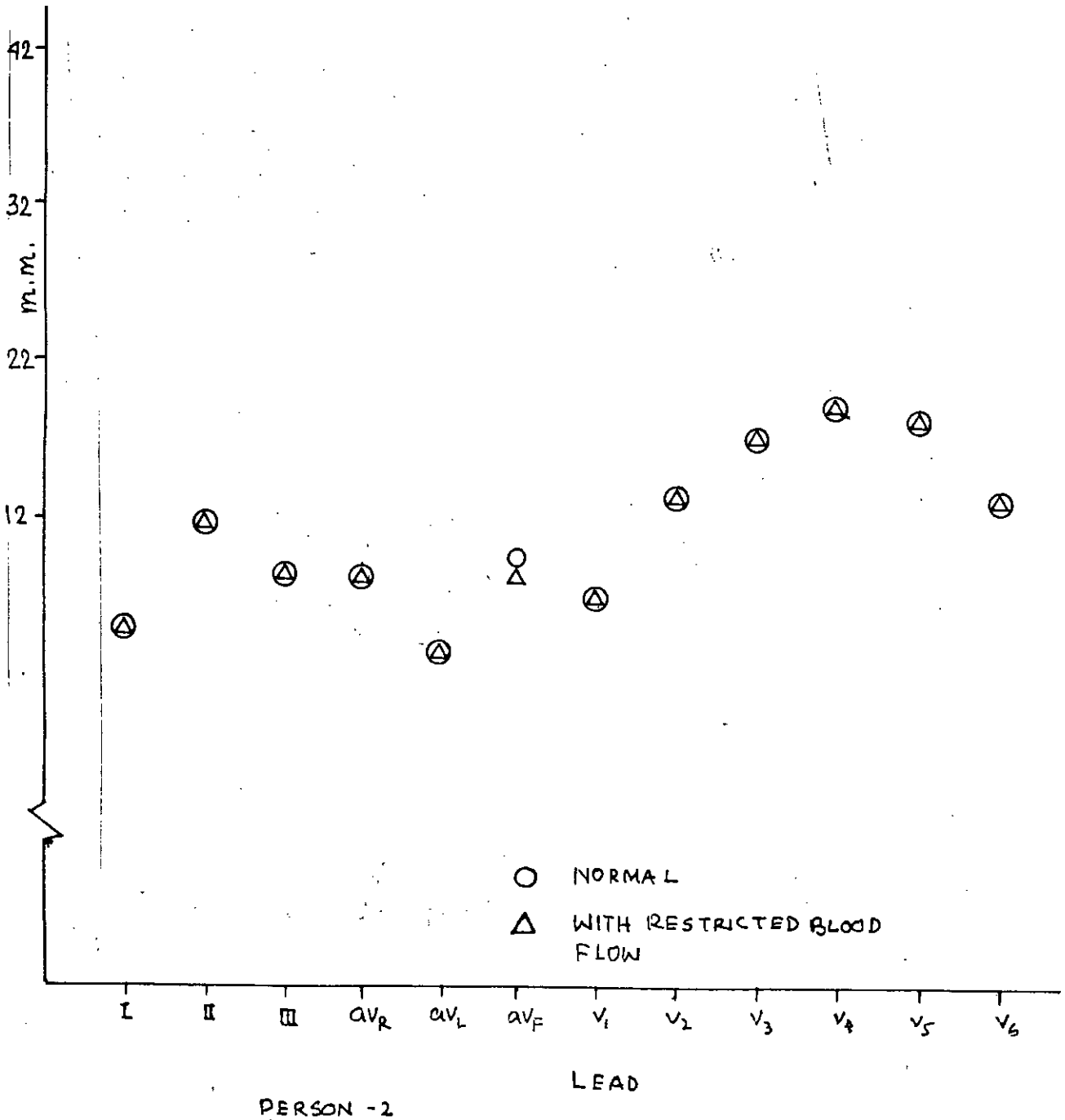
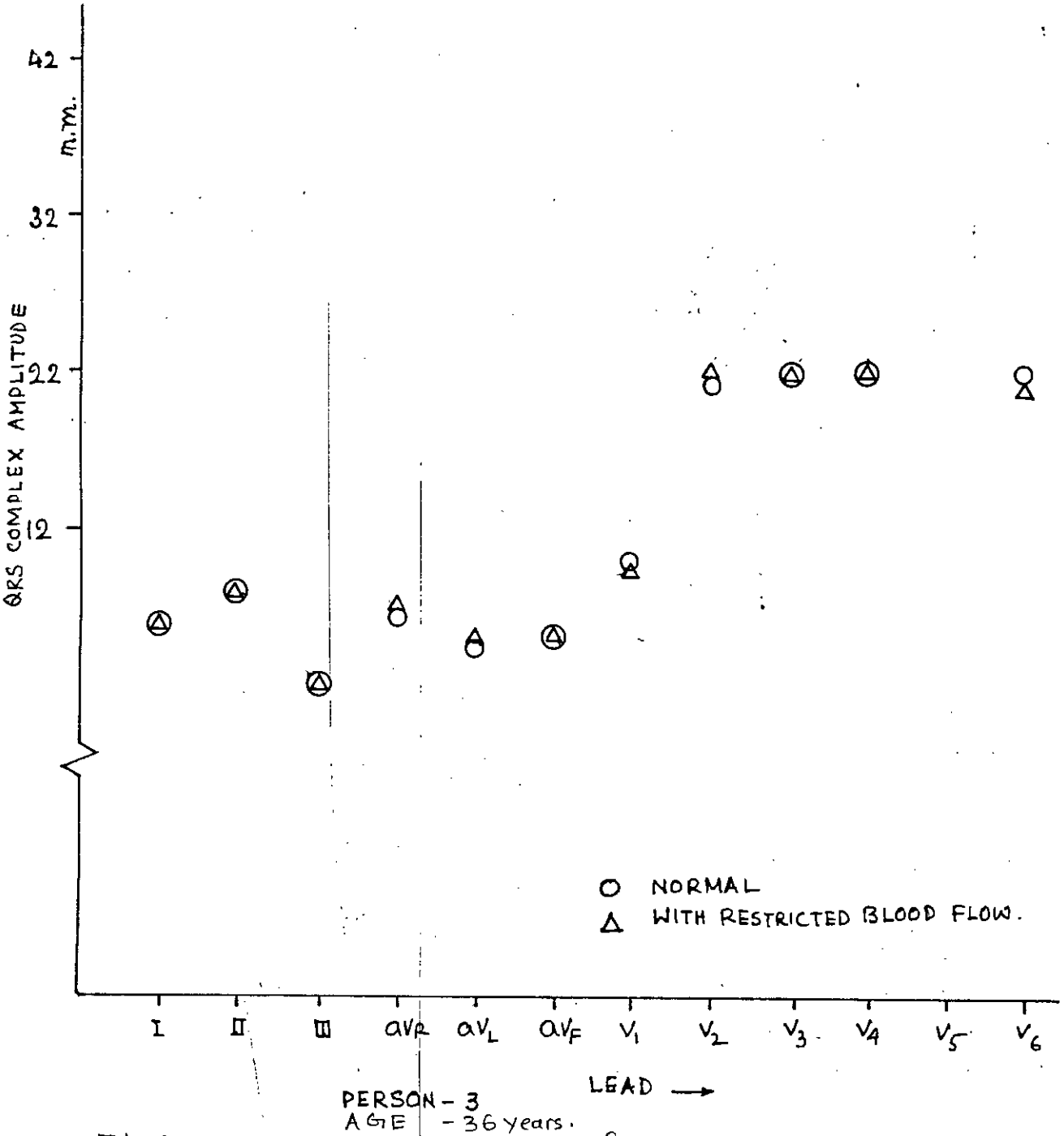


Fig- 6.1. : Graphical representation of QRS complex amplitude in different leads.



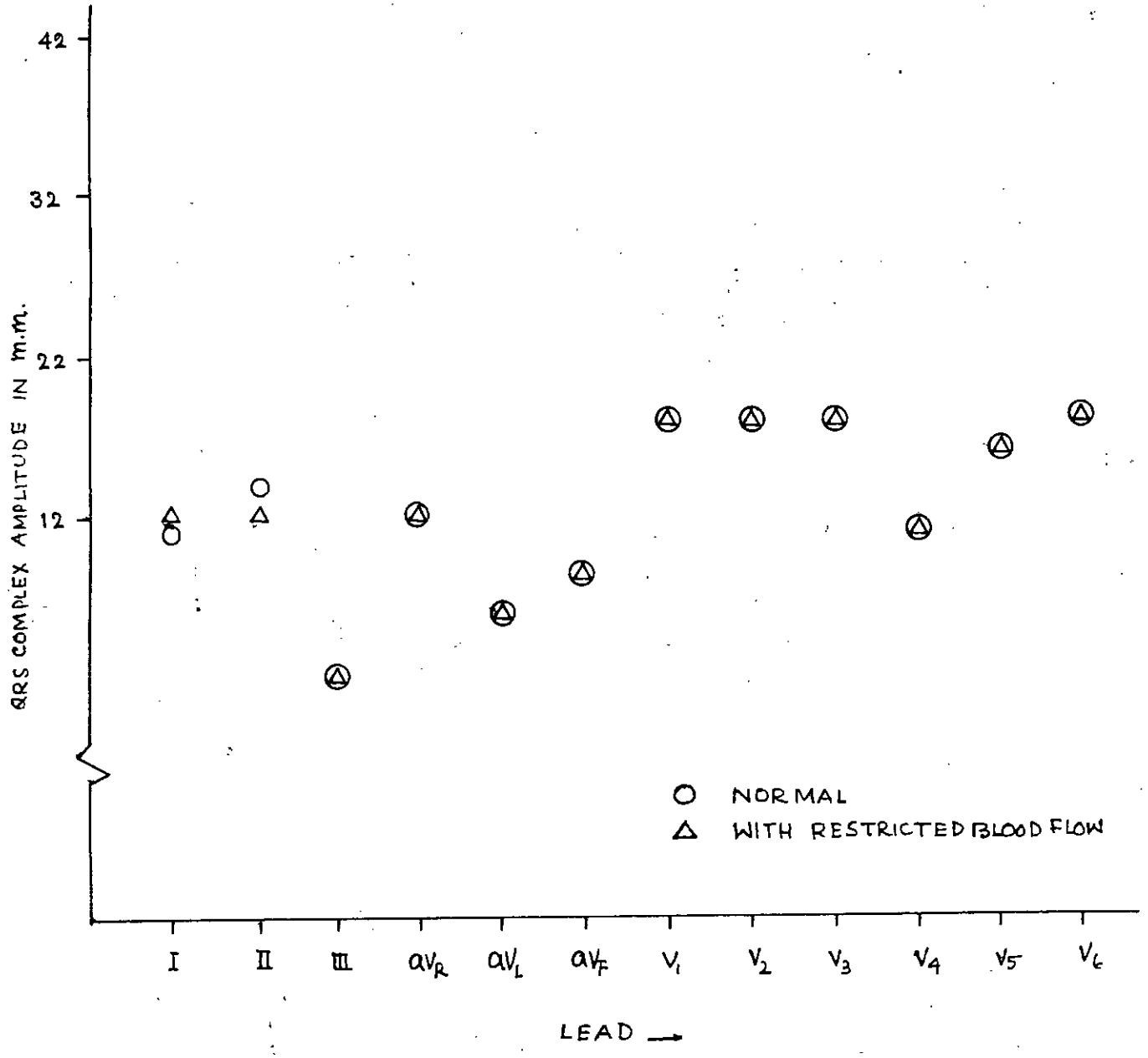
PERSON - 2
AGE - 36 years.

Fig 6.2: Graphical representation of QRS complex amplitude in different leads.



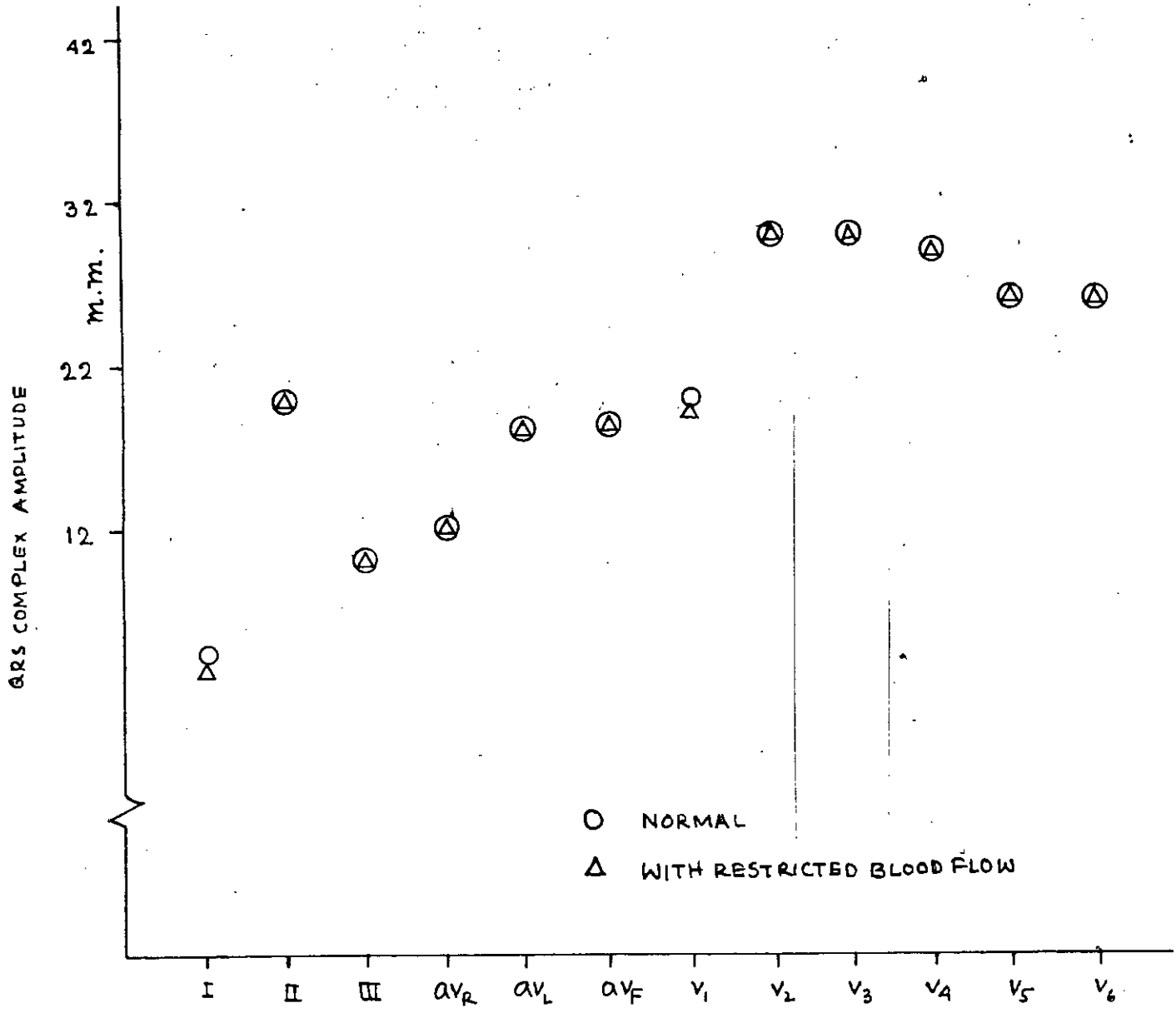
PERSON - 3
AGE - 36 years.

Fig-6.3. Graphical representation of QRS complex amplitude in different leads.



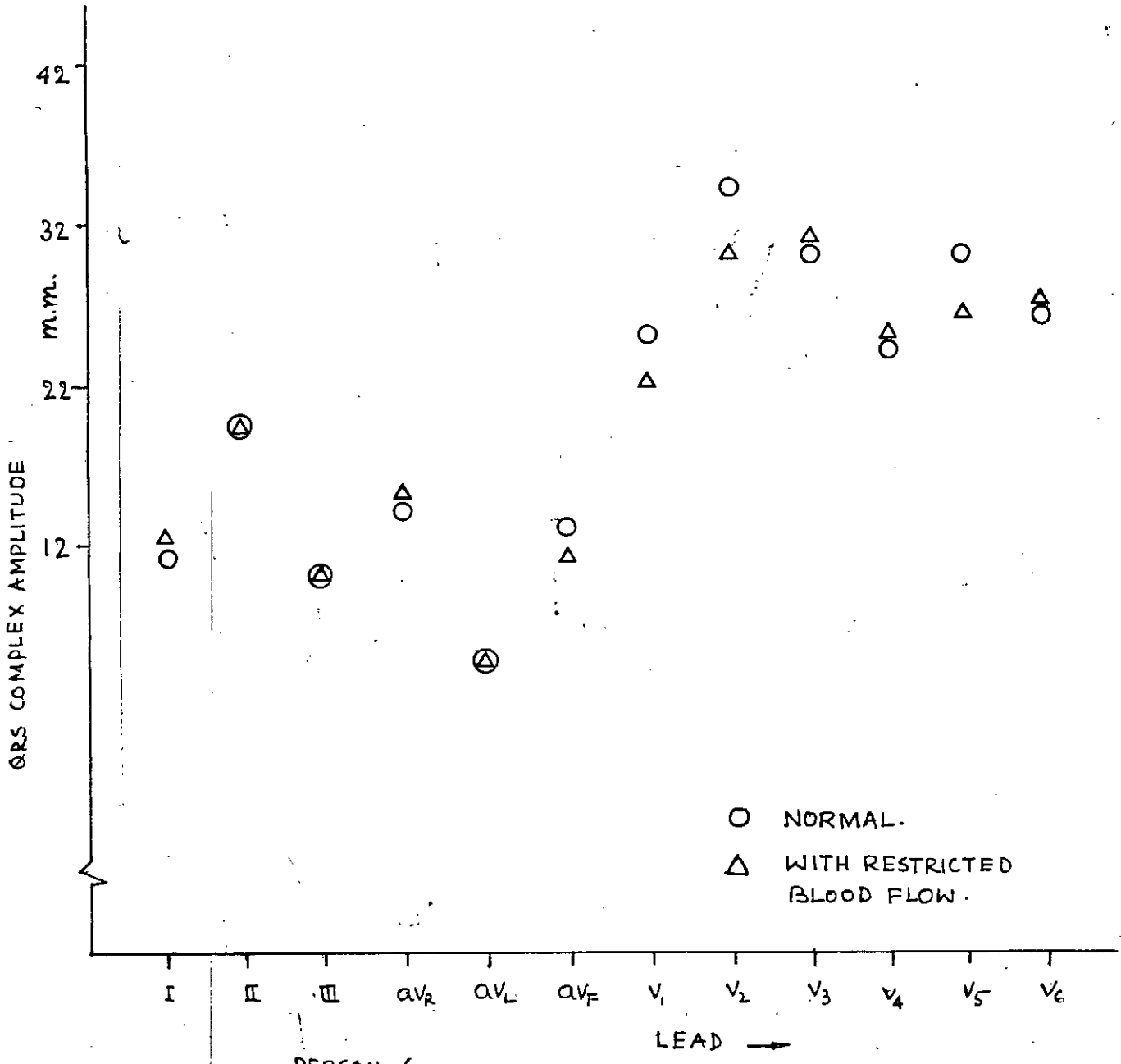
PERSON - 4.
AGE - 56 years.

Fig - 6.4 : Graphical representation of QRS complex amplitude in different leads.



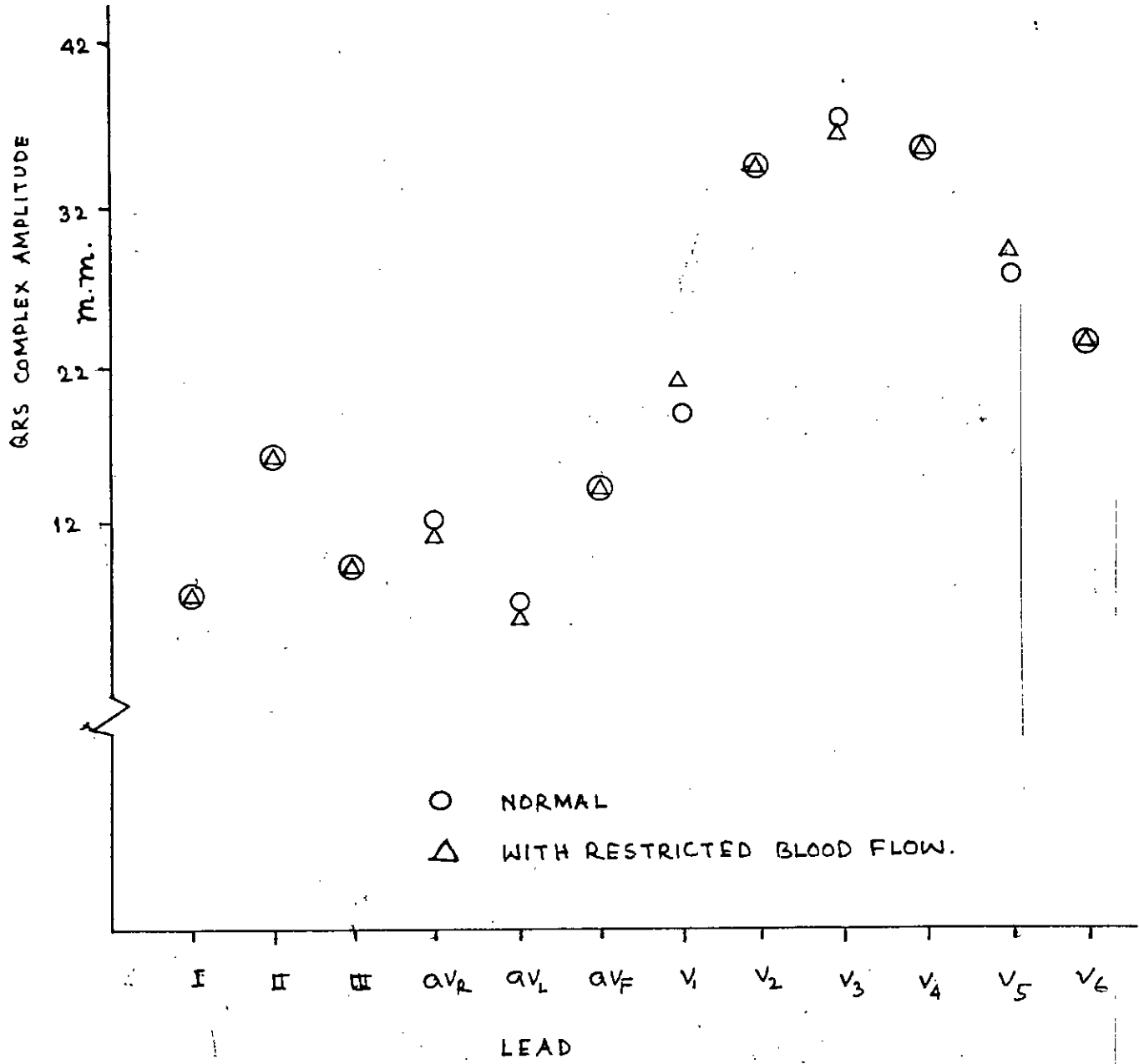
PERSON - 5
AGE - 24 years.

Fig 6.5% Graphical representation of QRS complex amplitude in different leads.



PERSON-6
AGE - 22 years.

Fig 6.6 : Graphical representation of QRS complex amplitude in different leads.



PERSON - 7
AGE - 14 years.

Fig 6·7: Graphical representation of QRS complex amplitude in different leads.

TABLE 6·1

DIFFERENCE OF QRS COMPLEX AMPLITUDE FROM NORMAL VALUE

LEADS / PERSON	I	II	III	aV_R	aV_L	aV_F	V_1	V_2	V_3	V_4	V_5	V_6
1	0.0	0.0	+0.5	+0.5	−0.2	0.0	0.0	0.0	0.0	−0.1	0.0	0.0
2	0.0	0.0	0.0	0.0	0.0	+1.0	0.0	0.0	0.0	0.0	0.0	0.0
3	0.0	0.0	0.0	−0.2	0.2	0.0	0.5	−1.0	0.0	0.0	+1.0	+1.0
4	−1.0	+2.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
5	+1.0	0.0	0.0	0.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.0
6	−1.0	0.0	0.0	−1.0	0.0	+2.0	3.0	4.0	−1.0	−1.0	−4.0	−1.0
7	0.0	0.0	0.0	+1.0	+1.0	0.0	−2.0	0.0	+1.0	.0	−1.5	0.0
STANDARD DEVIATION σ_n	0.638	0.699	0.174	0.570	0.396	0.728	1.380	1.498	0.534	0.345	1.581	0.534

$$\sigma_n = \sqrt{\frac{\Sigma (x_i - \bar{x})^2}{n}}$$

DISCUSSION ON THE EFFECT OF BLOOD FLOW CONSTRAINT (ON RIGHT ARM) ON QRS COMPLEX AMPLITUDE:

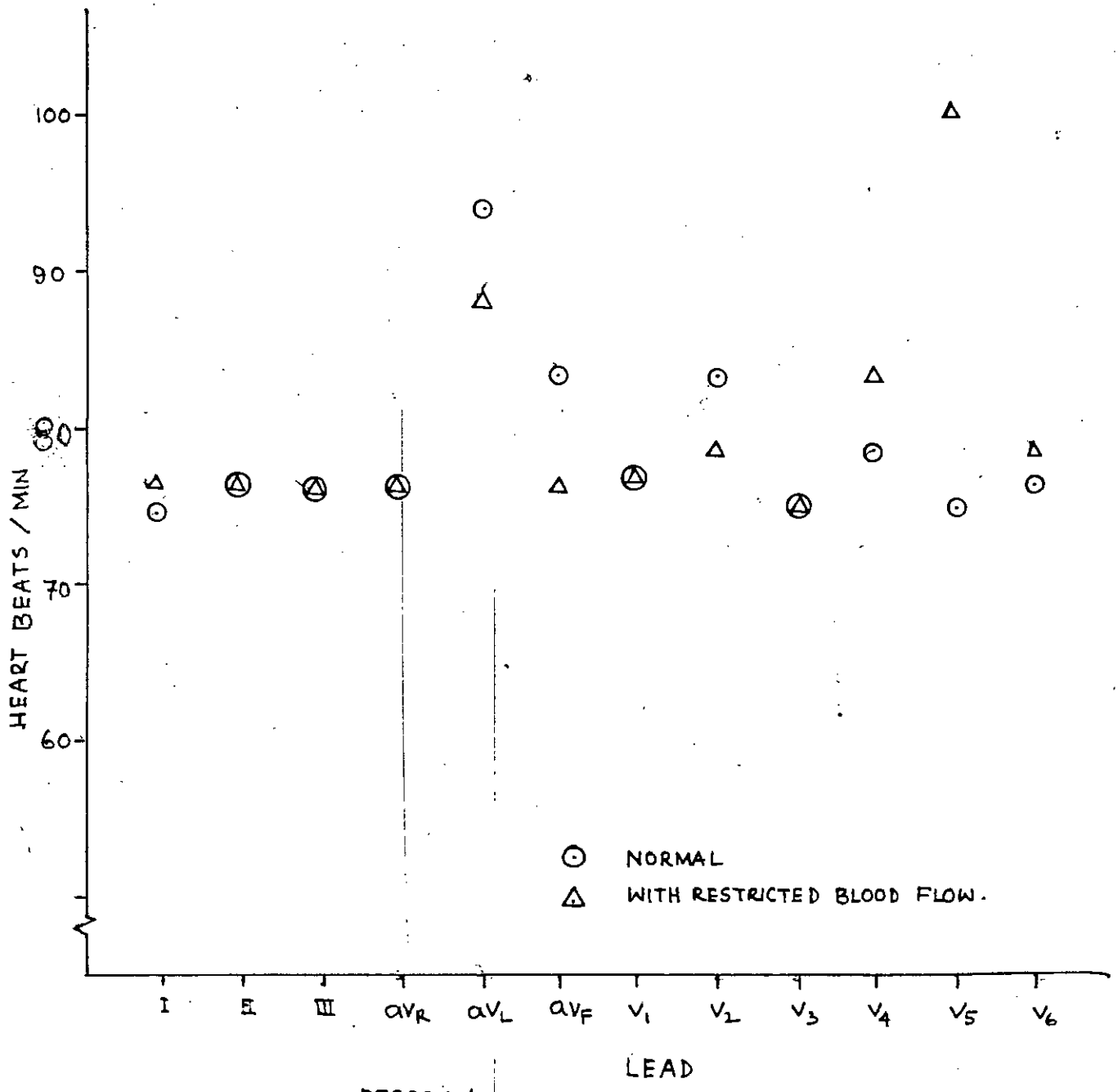
Standard deviation is calculated for the deviation of QRS complex amplitude from its normal value in 12 standards leads. The standa rds deviation is highest in lead V5 and is 1.581 and the lowest value is for lead III. Examination of standard deviation so calculated shows that the deviation is greater for leads near the chest e.g. V5, V2, and V1 respectively.

HEART BEATS MEASUREMENT

The rate of heart beat is calculated from the recorded electrocardiograms. The time interval between two successive beats is the reciprocal of the heart rate. The time interval is calculated between two successive peaks of the QRS complex.

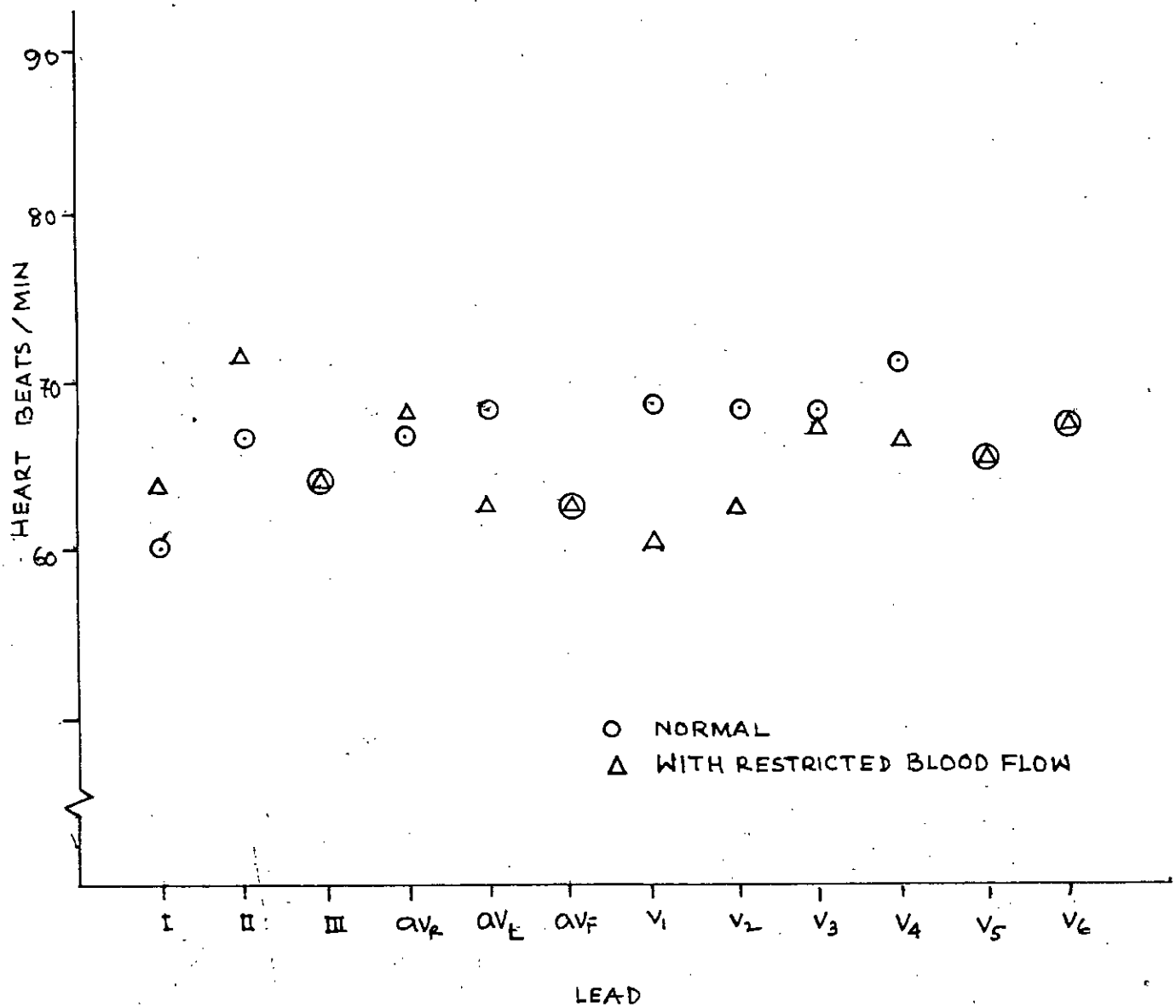
The heart beats calculated for different persons is shown in Figs.(6.8 - 6.14).

Table 6.2 shows the difference of heart beats from its normal value for different persons. This deviation of heart beats are calculated for 12 different leads.



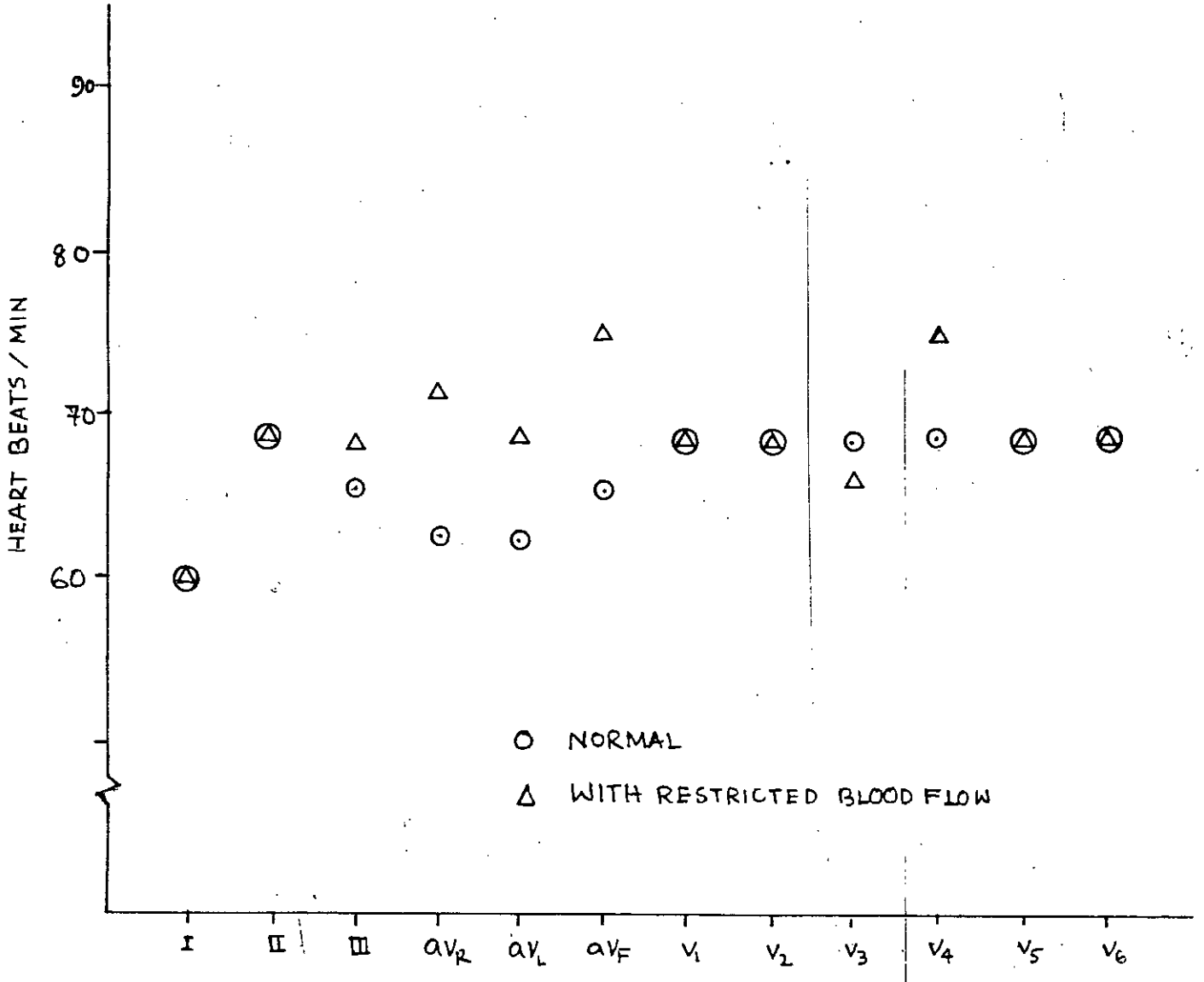
PERSON-1
AGE - 34 years.

Fig-6.8: Graphical representation of heart beats in different leads.



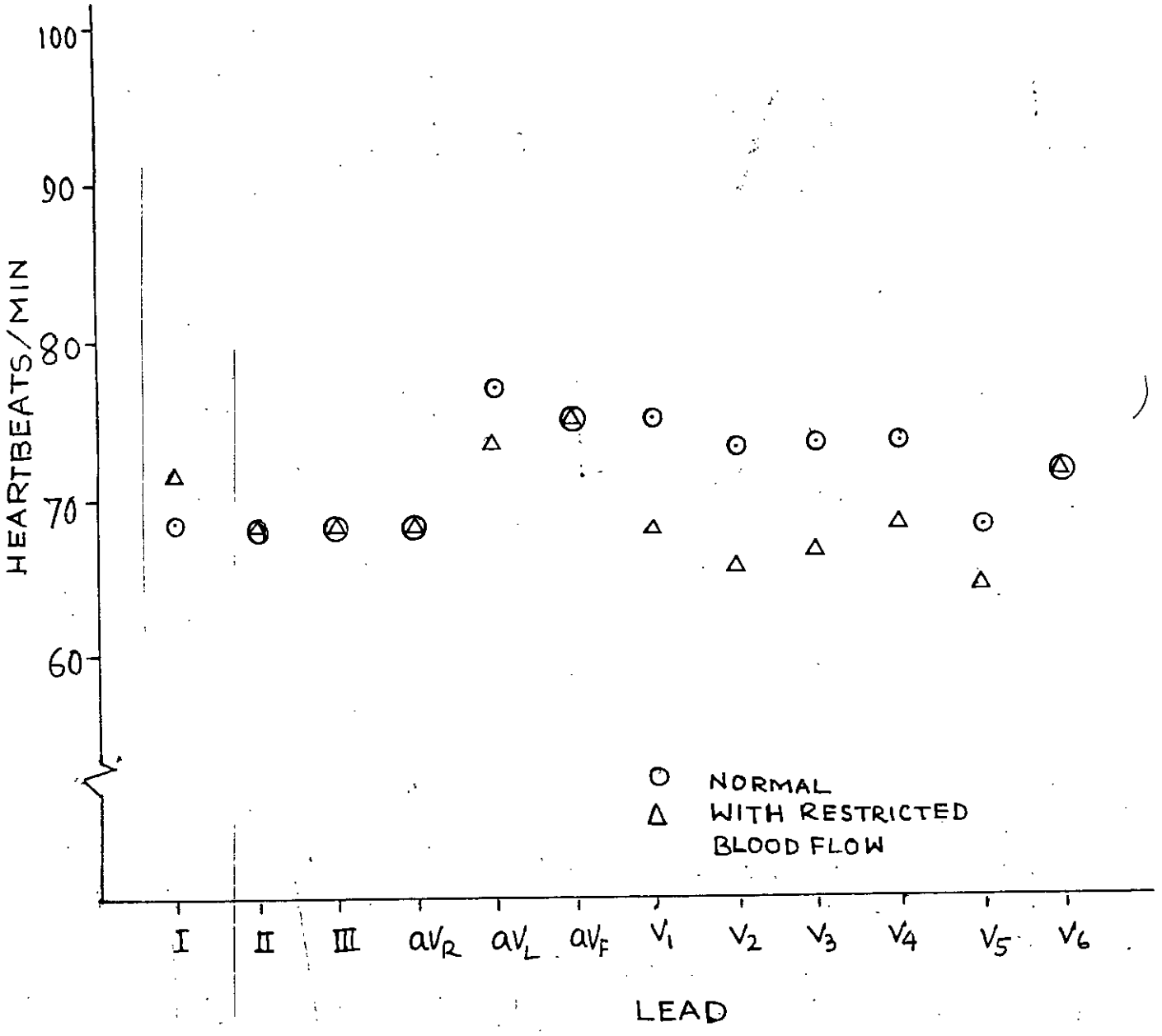
PERSON-2
AGE - 36 YEARS.

Fig 6.9 : GRAPHICAL REPRESENTATION OF HEART BEATS IN DIFFERENT LEADS.



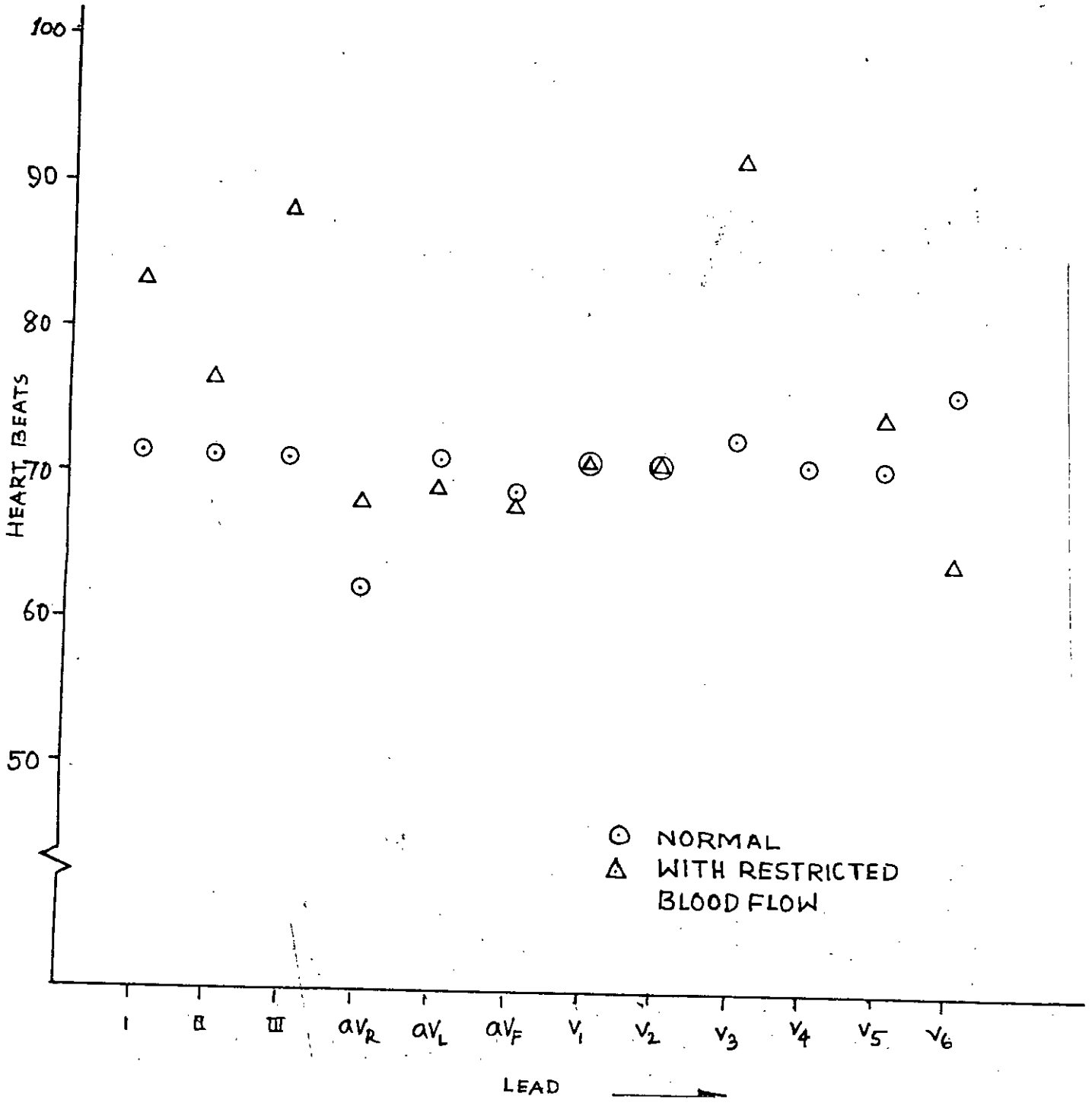
PERSON-3. LEAD
AGE -36 YEARS

FIG. 6.10: GRAPHICAL REPRESENTATION OF HEART BEATS IN DIFFERENT LEADS.



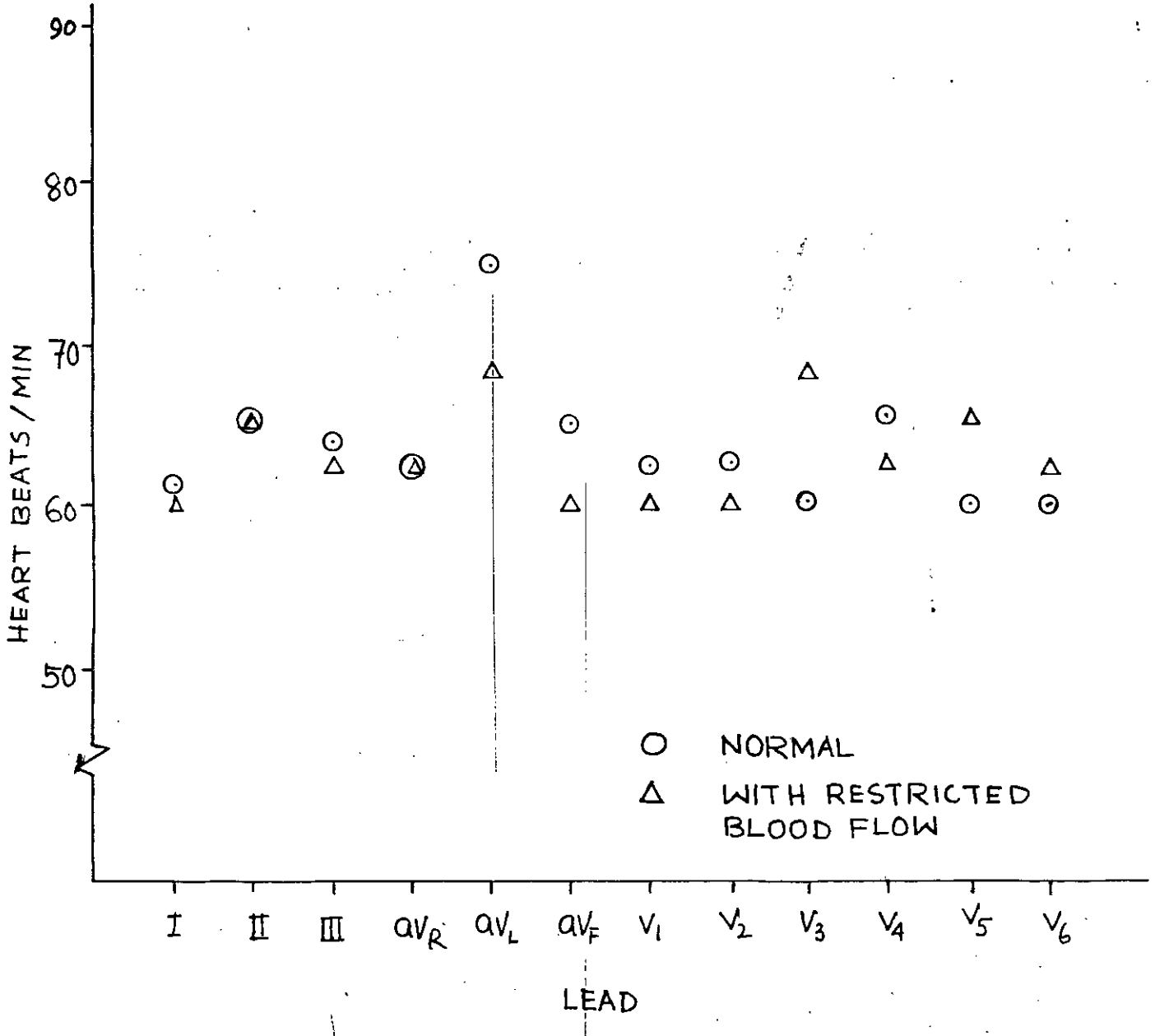
PERSON-4, AGE 56 YEARS;

FIG. 6.11: GRAPHICAL REPRESENTATION OF HEART BEATS IN DIFFERENT LEADS.



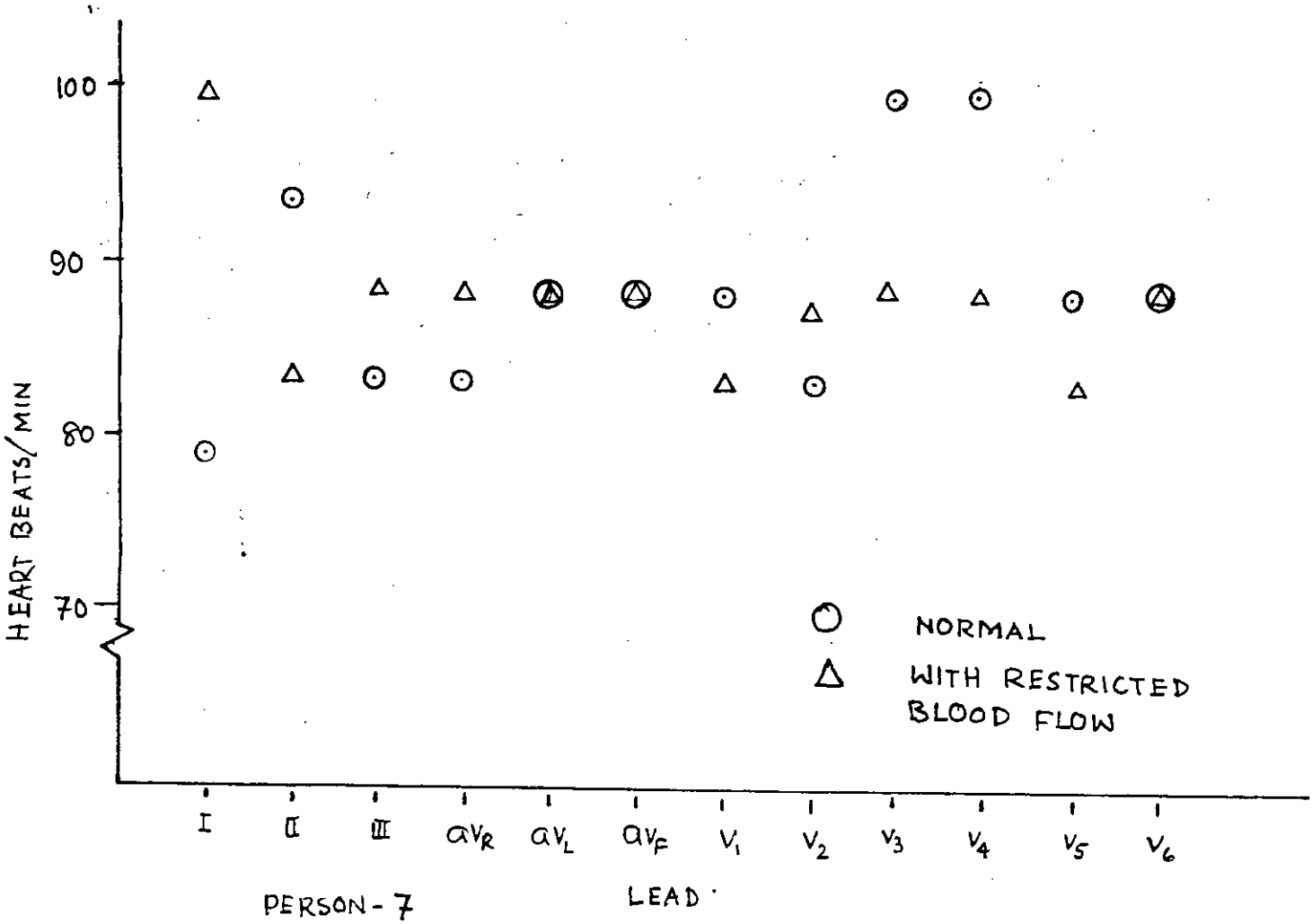
PERSON 5, AGE 24 YEARS.

FIG. 6·12: GRAPHICAL REPRESENTATION OF HEART BEATS IN DIFFERENT LEADS.



PERSON 6, AGE • 22 YEARS.

FIG: 6·13: GRAPHICAL REPRESENTATION OF HEART BEATS IN DIFFERENT LEADS.



PERSON - 7
AGE - 14 YEARS.

FIG: 6.14: GRAPHICAL REPRESENTATION OF HEART BEATS IN DIFFERENT LEADS.

TABLE 6.2

DIFFERENCE OF HEART BEATS FROM NORMAL VALUE

LEADS / PERSON	I	II	III	a_{VR}	a_{VL}	a_{VF}	V_1	V_2	V_3	V_4	V_5	V_6
1	-1.9	0.0	0.0	0.0	5.55	6.4	0.0	4.3	0.0	-4.4	-25	-1.7
2	-3.8	-4.8	0.0	-1.58	5.68	0.0	8.18	+5.68	1.58	4.8	0.0	0.0
3	0.0	0.0	-2.5	-8.93	-5.68	-9.8	0.0	0.0	2.28	-7.18	0.0	0.0
4	-3.12	0.0	0.0	0.0	3.75	0.0	6.82	7.95	6.50	5.0	2.96	0.0
5	-11.9	-5.6	-16.17	-5.68	1.73	1.52	0.0	0.0	-20.52	0.0	-3.57	11.7
6	+1.22	0.0	+1.33	0.0	6.82	5.0	2.5	2.5	-8.18	2.72	-5.22	-2.5
7	-21.05	10.45	-4.9	-4.9	0.0	0.0	4.93	-3.9	17.8	11.8	4.93	0.0
Standard Deviation σ_n	7.35	4.83	5.83	3.27	4.02	4.82	3.21	3.71	11.05	5.88	9.27	3.96

$$\sigma_n = \sqrt{\frac{\Sigma(x_i - \bar{x})^2}{n}}$$

144.9

Discussion on the effect of blood flow constraint (in right arm) on heart beats.

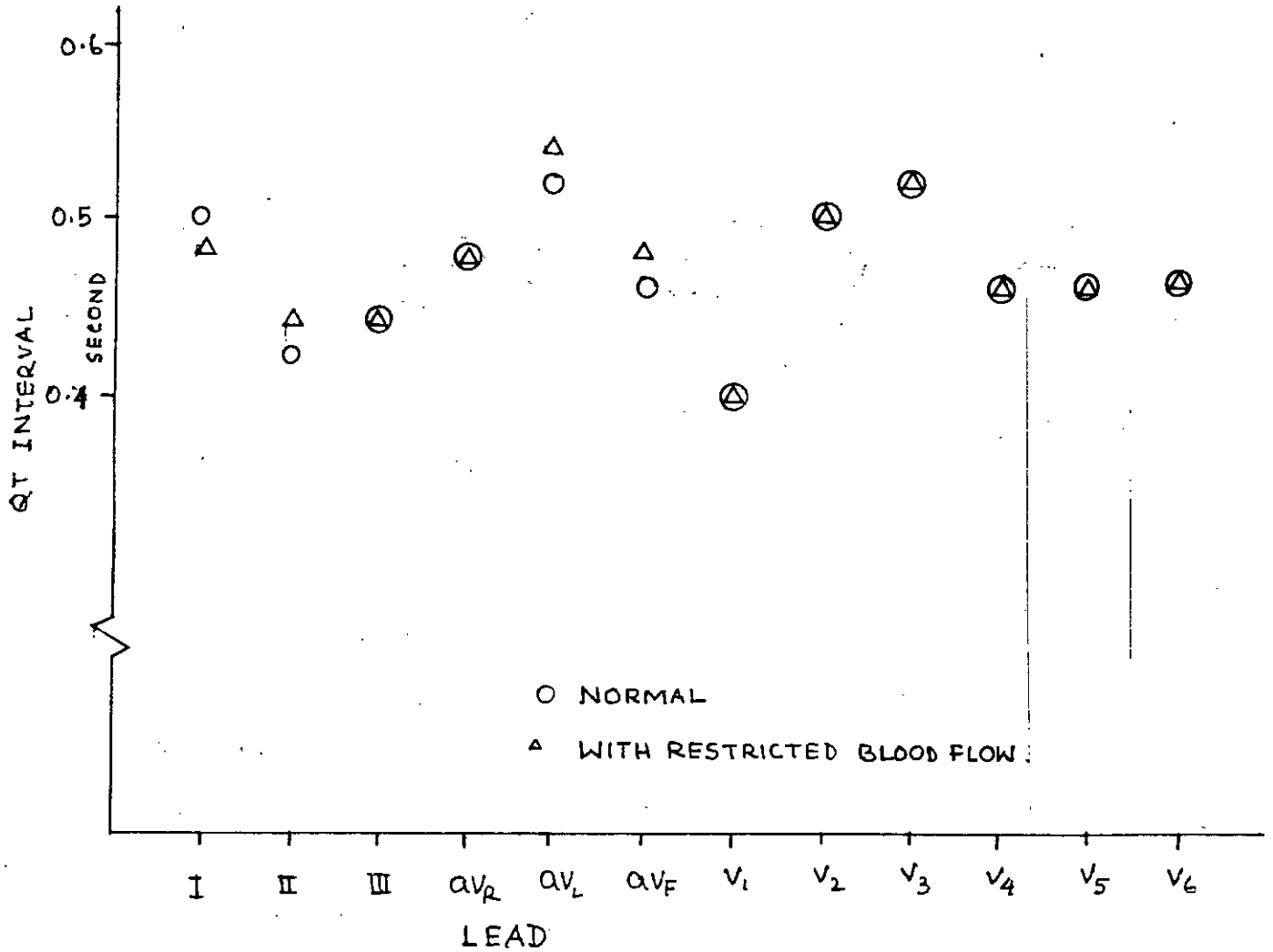
Standard deviation is calculated fro the deviation of heart heart beats from its normal value. Standard deviation calculated is seen to be highest in lead V_3 qhere the value is (S_D = 11.05). In lead V_5 the value of the standard deviation is seen to be 9.27. Here it is seen that the variation of heart beats is of high value in the Chest leads. The variation of heart beat is high in comparison with QRS complex amplitude variation.

QT interval Measurement

QT interval corresponds to the time of begining of the Q-wave to the end of the T wave. This is the total time required for ventrieular depolarization and repolarization. It is measured is seconds.

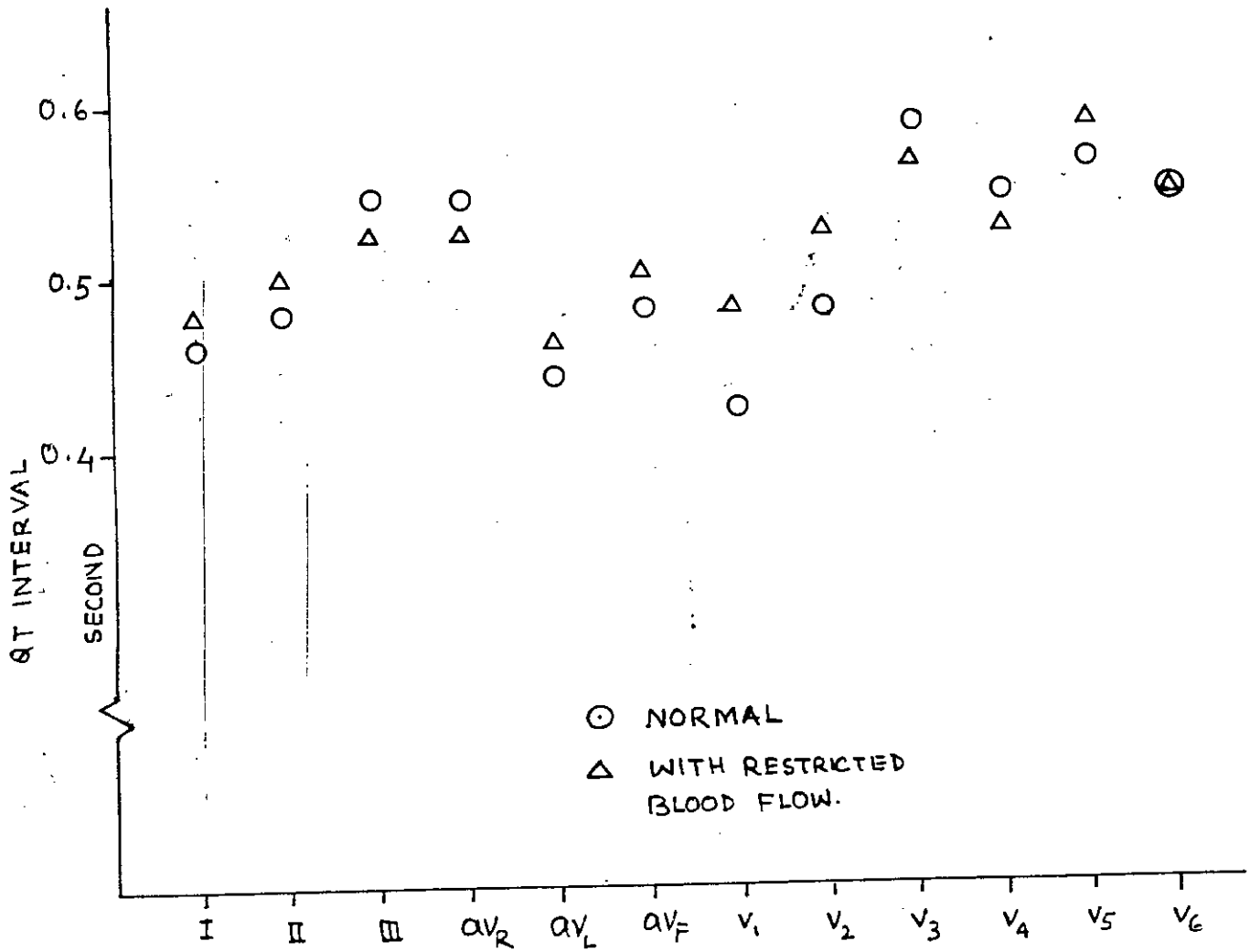
QT interval calculated for different persons is shown in Fig(6.15 - 6.21.)

Table 6.3 shows the difference of QT interval from the normal value for different persons.



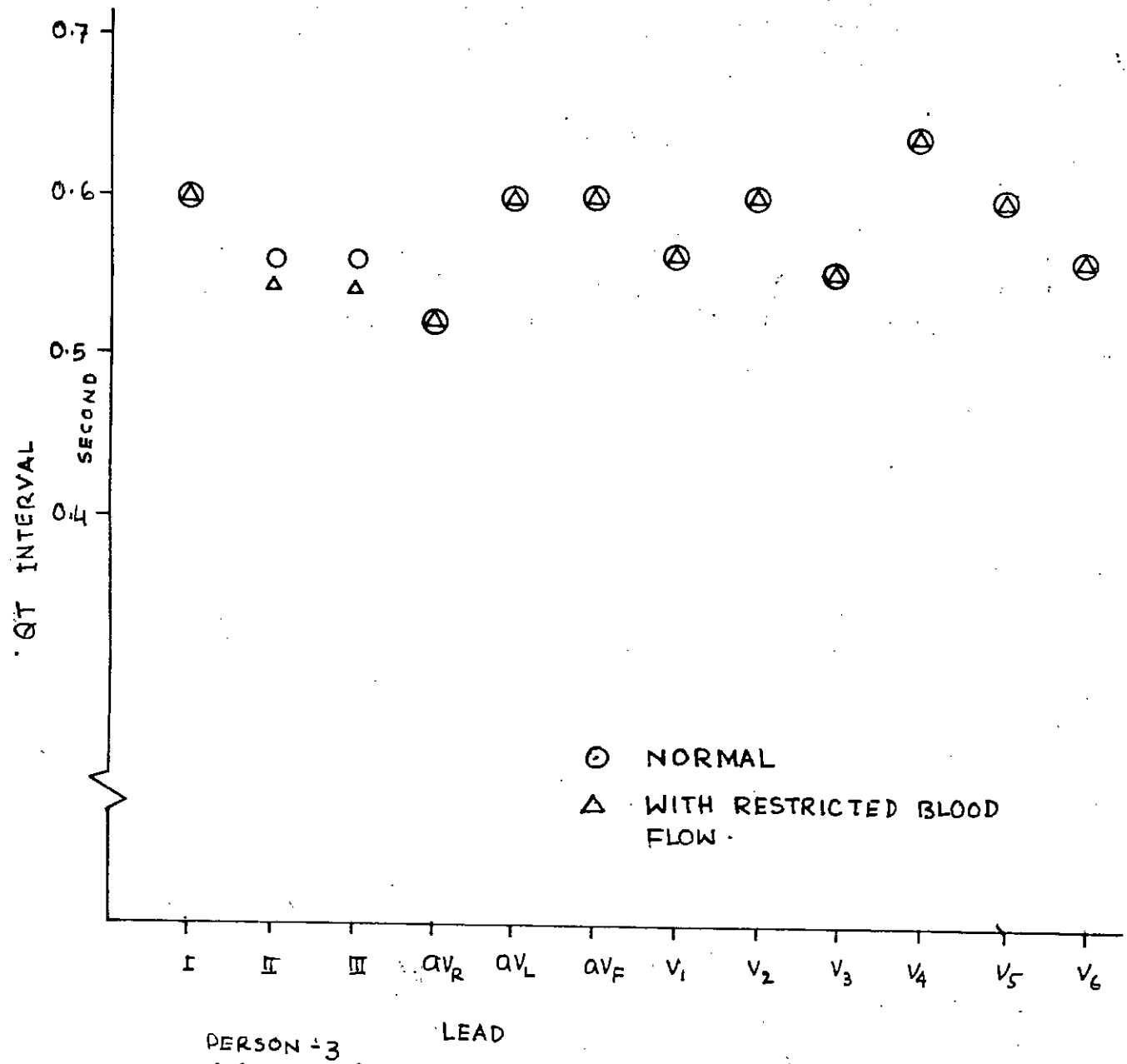
PERSON-1, AGE - 34 YEARS.

FIG. 6.15: GRAPHICAL REPRESENTATION OF QT INTERVAL IN DIFFERENT LEADS.



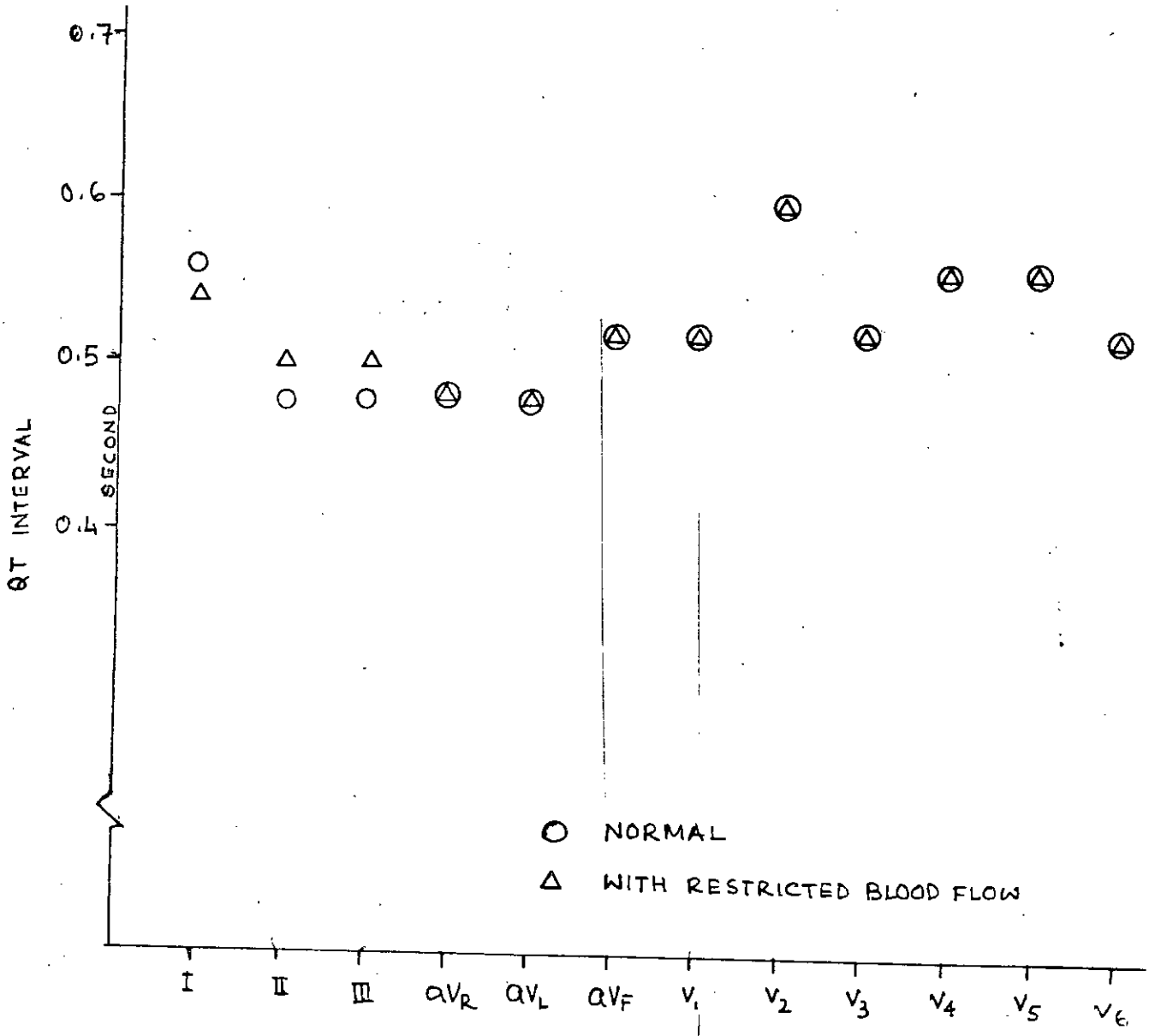
PERSON-2 LEAD
AGE -36 YEARS

FIG. 6.16 : GRAPHICAL REPRESENTATION OF QT INTERVAL IN DIFFERENT LEADS.



PERSON - 3
AGE - 36 YEARS.

FIG. 6.17 : GRAPHICAL REPRESENTATION OF QT INTERVAL IN DIFFERENT LEADS.



PERSON - 4
AGE - 56 YEARS.

FIG 6·18: GRAPHICAL REPRESENTATION OF QT INTERVAL IN DIFFERENT LEADS.

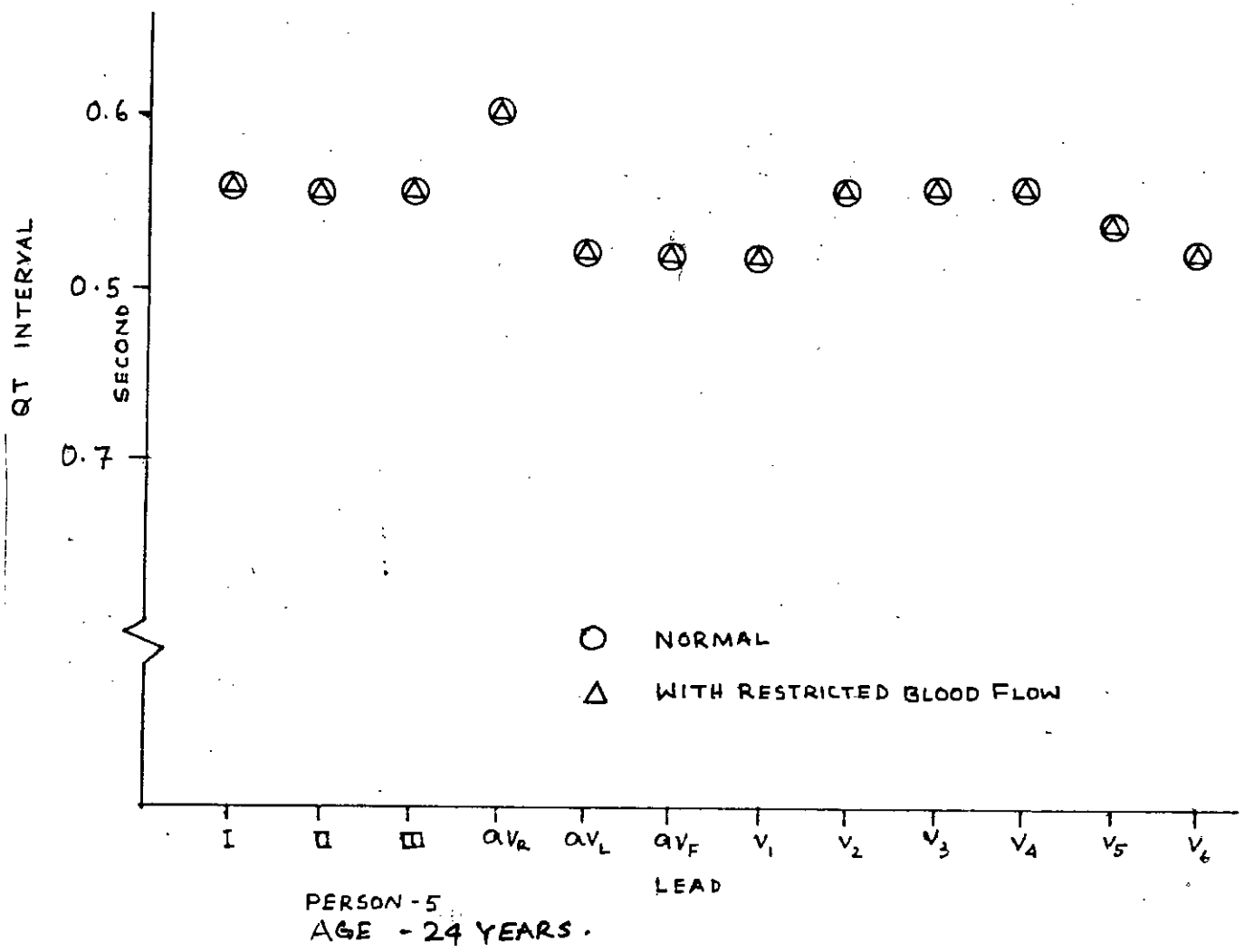
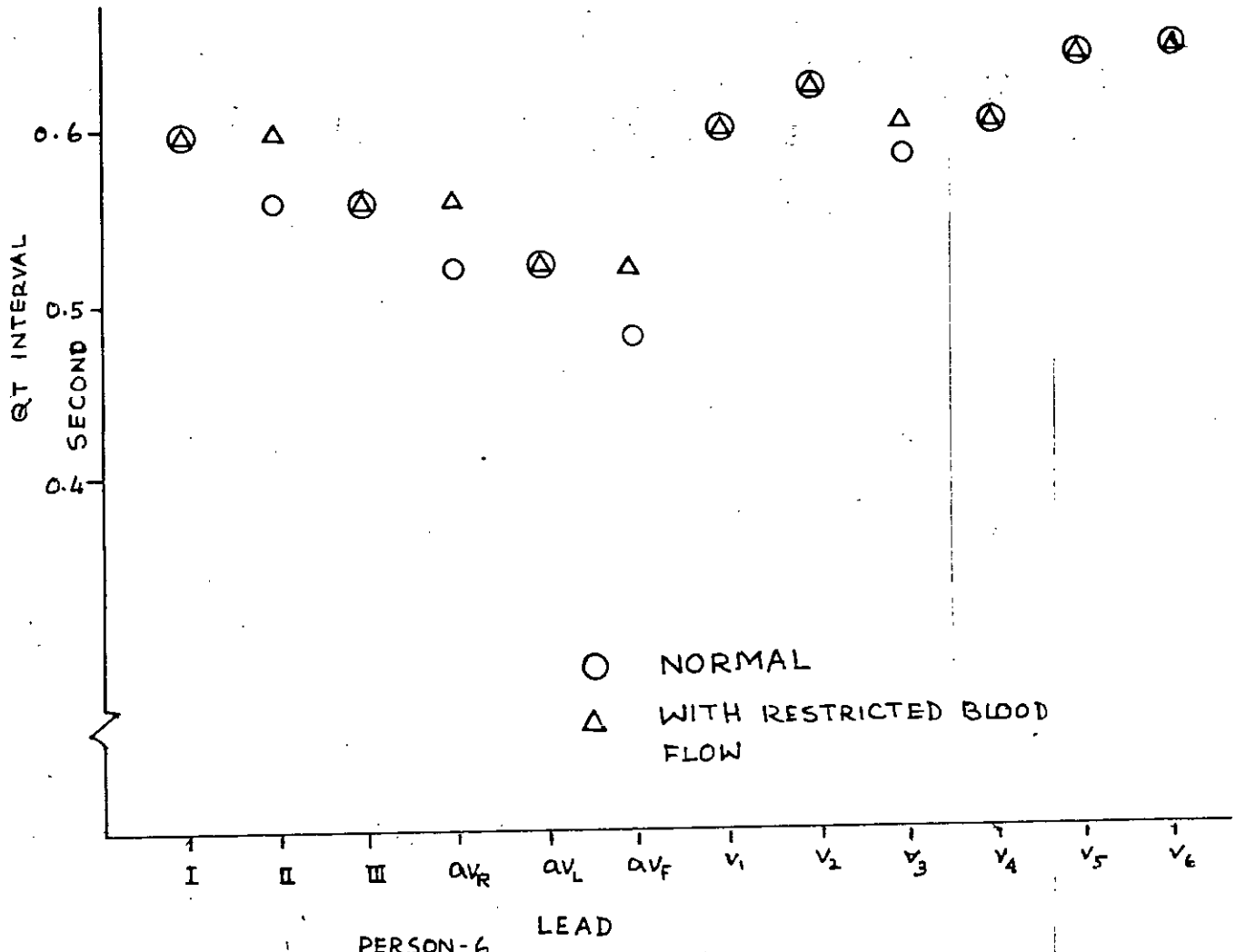
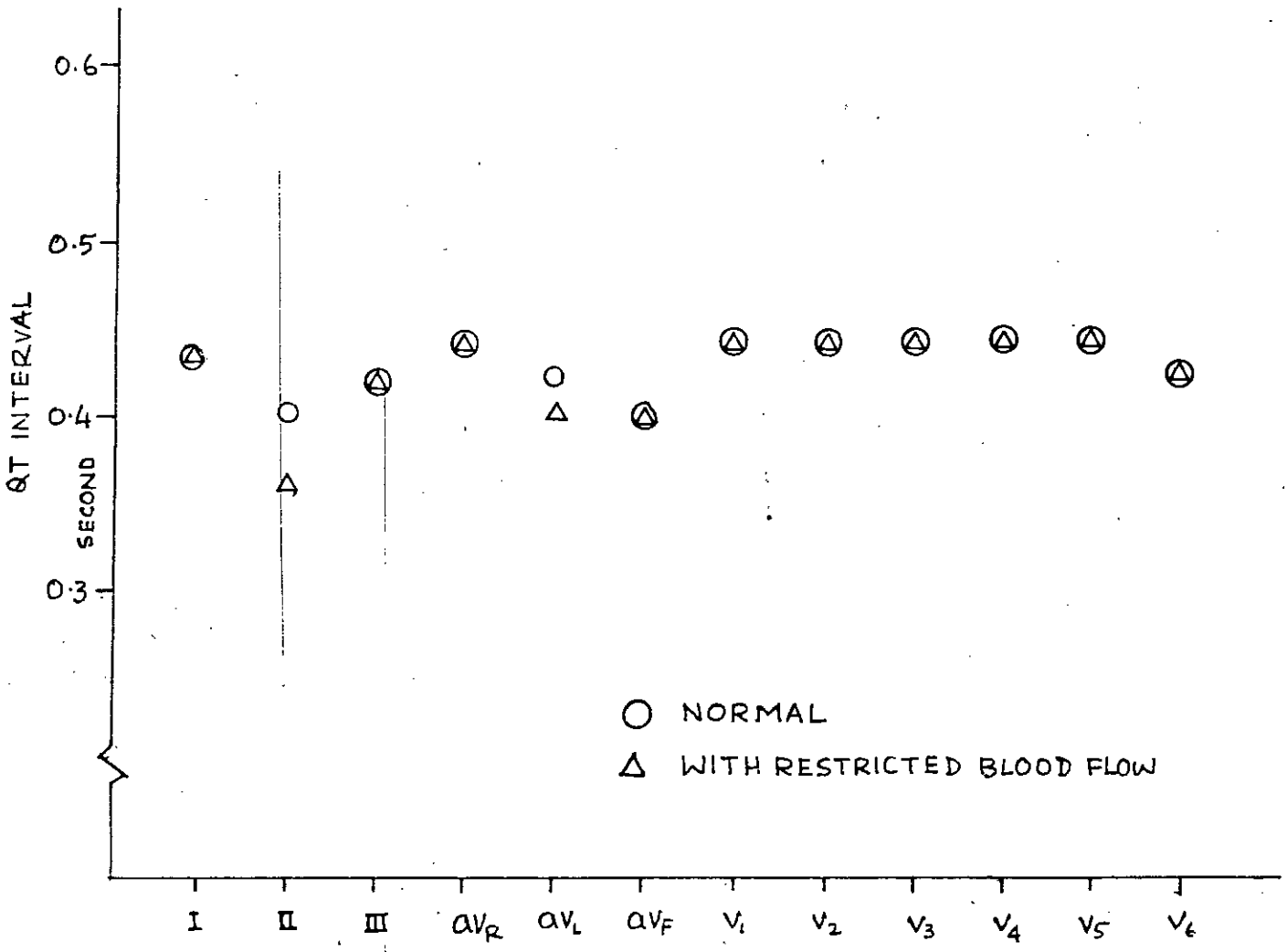


FIG. 6.19 : GRAPHICAL REPRESENTATION OF QT INTERVAL IN DIFFERENT LEADS.



PERSON-6
AGE - 22 YEARS.

FIG. 6·20: GRAPHICAL INTERPRETATION OF QT INTERVAL IN DIFFERENT LEADS.



PERSON - 7
AGE - 14 YEARS.

FIG. 6.21 : GRAPHICAL REPRESENTATION OF QT INTERVAL IN DIFFERENT LEADS.

TABLE 6·3

DIFFERENCE OF QT INTERVAL FROM NORMAL VALUE.

LEADS / PERSON	I	II	III	a_{VR}	aV_L	aV_F	V_1	V_2	V_3	V_4	V_5	V_6
1	0·02	-0.02	0.00	0.00	-0·02	- 0.02	0.00	0.00	0.00	0.00	0.00	0.00
2	-0·02	- 0.02	0·02	0.02	- 0·02	- 0· 02	- 0.06	- 0·04	+0·02	+0·02	-0·02	0.00
3	0.00	0·02	0.02	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
4	0.02	-0.02	-0.02	0·00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0·00
6	0.00	-0.04	0.00	-0.04	0.00	-0.04	0·00	0.00	-0·02	0·00	0.00	0.00
7	0.00	0.04	0·00	0.00	0·02	0·00	0·00	0.00	0.00	0·00	0.00	0.00
Standard Deviation σ_n	0·0127	0·0255	0·0127	0·016	0·0120	0·0145	0·209	0·0139	0·0106	$6·9\times10^{-3}$	$6·9\times10^{-3}$	0·000

$$\sigma_n = \sqrt{\frac{\Sigma(x_i - \bar{x})^2}{n}}$$

DISCUSSION ON THE EFFECT OF BLOOD FLOW CONSTRAINT ON QT INTERVAL

Standard deviation is calculated for the deviation of QT interval from the normal value. From the table it is seen that the deviation of QT interval from its normal value is zero in lead V_6 for all persons. In other leads the variation is small in comparison with the variation of QRS complex amplitude and heart beats. Here lead V_1 shows the highest value of the standard deviation (SD = 0.209).

From the discussion of the experimental results it is seen that the change in values of QRS complex amplitude, heart beat and QT duration at constraint flow of blood in the right arm from their normal is well reflected in precordial leads than any other leads used for recording of ECG.

Comments and Suggestion:

If the wave shapes of the electrocardiogram for the above said conditions are examined, it is seen that in some cases the line traced on the electrocardigram recorded with blood flow constraint is not as smooth as on the electrocardiogram in normal case for a person recorded in the same lead. The reason for this somewhat wavy line in electrocardiogram for restricted blood flow in arm may be due to some difficulty (excess load ?) in the pumping function of the heart. A reliable conclusion about the pumping load on the heart can be made with precision if the number of persons examined is increased. Similar is the case with QRS complex amplitude heart beats and QT interval because reliability of such comment lies on the volume of data obtained for different persons is different pathologic condition. Further detailed work on this aspect may produce useful information regarding desirable pumping effectiveness of the heart.

Chapter 7

(7.1) Discussion and Recommendation

Discussion:

A goal of electrocardiography is the solution of what has become known as the inverse problem i.e the determination of the electrical activity of the myocardium from potential measurement on the body surface. Unfortunately, theoretical considerations indicates that this inverse problem can not be solved uniquely . However, solution of the forward problem can provide information about the inverse problem. The forward problem is defined as the situation in which the electrical activity of the myocardium as well as shape of the other characteristics of the body are known and it is desired to calculate the potentials at the body surface. Because of the complex natures of both electrical activity of the myocardium and the actual human body shape, various simplified models representing these quantities are employed in the study of forward and inverse problem.

Even if it were possible to ascertain the electrical condition of each myocardial cell from the surface potential measurements, the amount of data would be over whelming and, for clinical purpose, certainly, be unwieldy. So, a finite number of parameters, that represents the electrical condition of heart is required. A first step in this regard is that the net dipole activity at finite number of regions which subdivide the heart may be specified. But this dipole representation is crude and has great number of short comings.

An electrocardiographic model can be formulated which assumes the primary sources to be located in an inhomogeneous medium, or the combined primary and secondary sources in a homogeneous medium. It is feasible in forward problem. However, in the inverse problem, as it is based on external measurements, it will be difficult to distinguish the two. In particular, this seems true when considering the effect of intracardiac blood, which depends critically on geometry that can only be guessed at. Because of this difficulty, the equivalent cardiac generator is normally defined with respect to a homogeneous volume conductor.

Measurements of the surface-potential do not contain sufficient information to permit a determination of the distributed electrical sources in the heart. When the myocardial source is describable as a uniform double layer, it is difficult to determine its shape from potential measurements over the surface. Because the potential field produced by the double layer depends only on the solid angle subtended by the field point, thus measurements can determine only the solid angle and not the shape. That means surface measurements record integrated effects produced by the source and that there are basic difficulties in resolving the summation into its components.

It is seen that the intensity of field falls off with the distance from the source. It provides a limitation in the

determination of nature of two sources. Because of in-evitable noise, signals that might be of importance in resolving two different distribution could lie below the noise level. Thus two sources may look alike from practical stand point, given the ambiguity in meausrement. Multipole theory gives some insight into this question because it shows that higher order source components involve higher inverse powers of r and hence will, in general, have decreasing contributions to the total surface potential. As the order of multipole increases, the difficulty in detecting its contribution, in general, also increases. So there is also limitations on the 'Equivalent cardiac Generators' represented by multipole expansions. But the experiments, conducted by Plonsey , Heppner and others, show that the inclusion of quadrupole and octopole component as an addition to dipole model provides a close resembled ECG record with actual ECG of beating heart.

The"multiple dipole distribution" as a heart model is a useful choice amongst the category. Such model permits direct physiological interpretations; e.g. ; the absence of excitation of a dipole could be interpreted as an infraction in that region. It is hoped that the lack of uniqueness can be reduced or eliminated by adding known physiological constraints. Untill these questions of uniqueness are resolved, a conservative approach is to choose an equivalent heart model that is uniquely defined by surface potentials as a fundamental property. Such a model is the spherical harmonic multipole model.

In addition to the fixed-dipole, multipole and multiple dipole cardiac representations, other formulations have been proposed[8]. One is the moving dipole; in this model, the dipole location is permitted to shift so that some reflection of the activation region may occur. The criterion for origin location would be to achieve the best fit to the measured data. This model has the disadvantage related to the uniqueness problem, namely that no unique physical interpretation of the significance of the shifting origin can be given theoretically. On the other hand, one could view this model as increasing the heart vector from three to six dimensions, there by providing additional possibilities in the specification of heart condition.

From the above discussion it is seen that the theoretical analysis of electrical activity of the heart does not play a dominant role in advancing clinical ECG because the value of clinical ECG not only depends on ECG data but also on their corelation with pathologic conditions[10]. The recordings of the magnetic field created by cardiac current known as Magnetocardiogram are now a days investigated in a number of laboratories to supplement the additional information about the condition of heart but this depends upon the accuracy and sensitivity of the instrument used to record MCG.

So far as measurement is concerned, MCG is subjected to less limitations than ECG as; MCG measurement is more localized (above the region of chest) ; MCG is independent of the boundary between human body and sensor electrode; and there is no

problem of contact impedance in MCG. There is a good potentiality of its use for clinical diagonosis.

(7.2)

Recommendation:

The aim of the research work related with the mathematical development of electrocardiography is to know the condition of heart during activation. The determination of the condition of conductive system of heart as well as heart muscle cell depends not only on the potential obtained from the body surface but also on pathologic condition. There is a scope for investigation on the effect of external electric field on heart's electrical activity and heart muscle itself. The effect of external electric field on the propagation of action potential in the cardiac muscle and their response to such external field may be investigated. Mathematical investigation so far available considered cardiac source as dipole and multipole. In actual, cardiac potential source is distributed all throughout. So a continuous distribution of potential source within heart with certain time lag will give much more accurate mathematical modelling.

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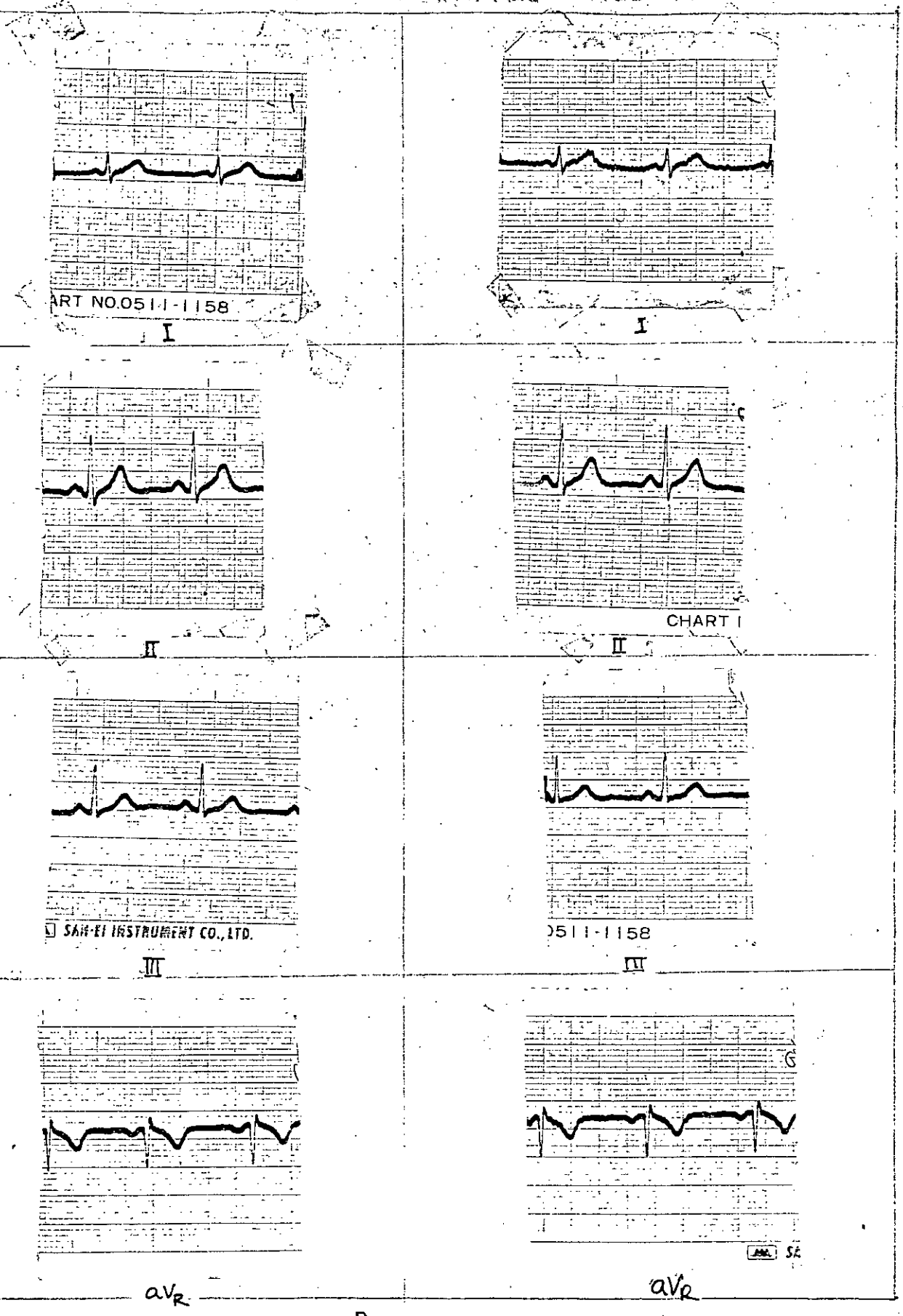
APPENDIX A

RECORDED ELECTROCARDIOGRAMS.

NORMAL

WITH RESTRICTED BLOOD FLOW

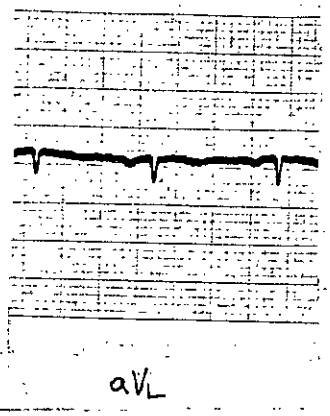


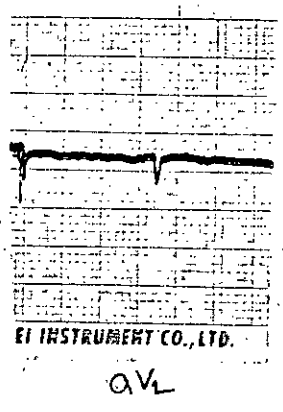
Person 1 - Age - 34 yrs.

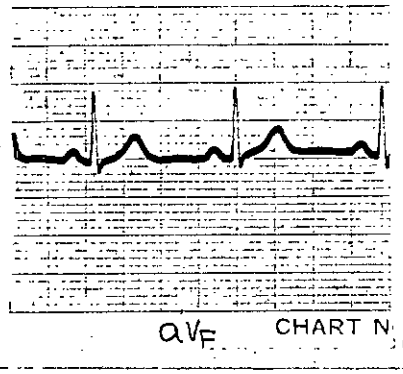
FIG A-1. RECORDED ECG OF PERSON 1, LEAD USED I, II, III, aV_R.

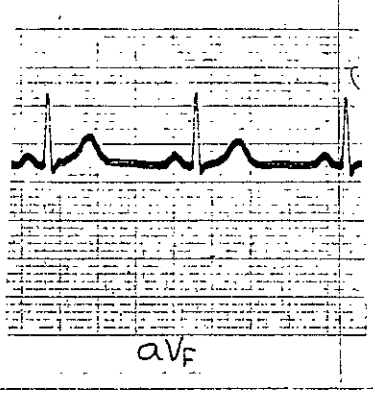
NORMAL

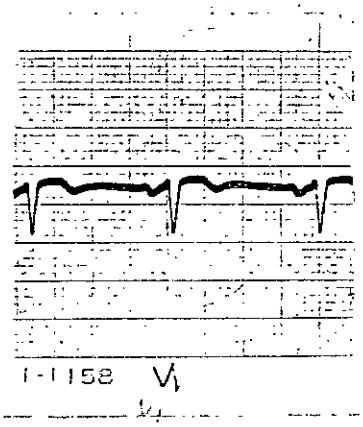
WITH RESTRICTED BLOOD FLOW.

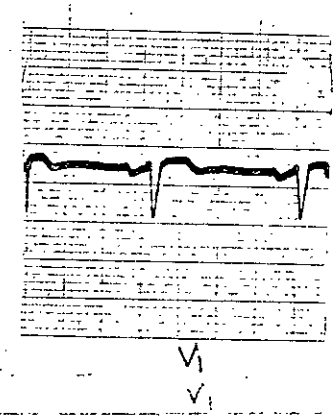


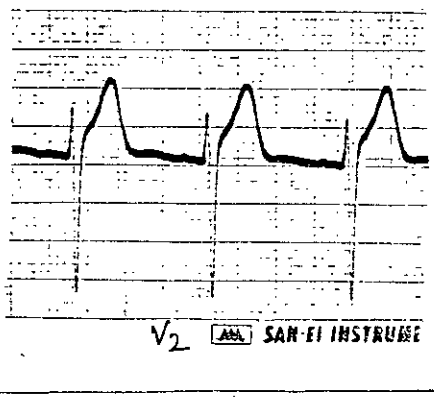


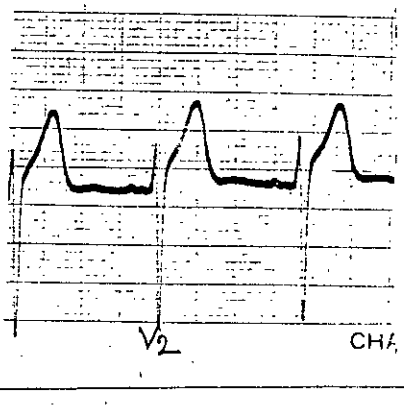










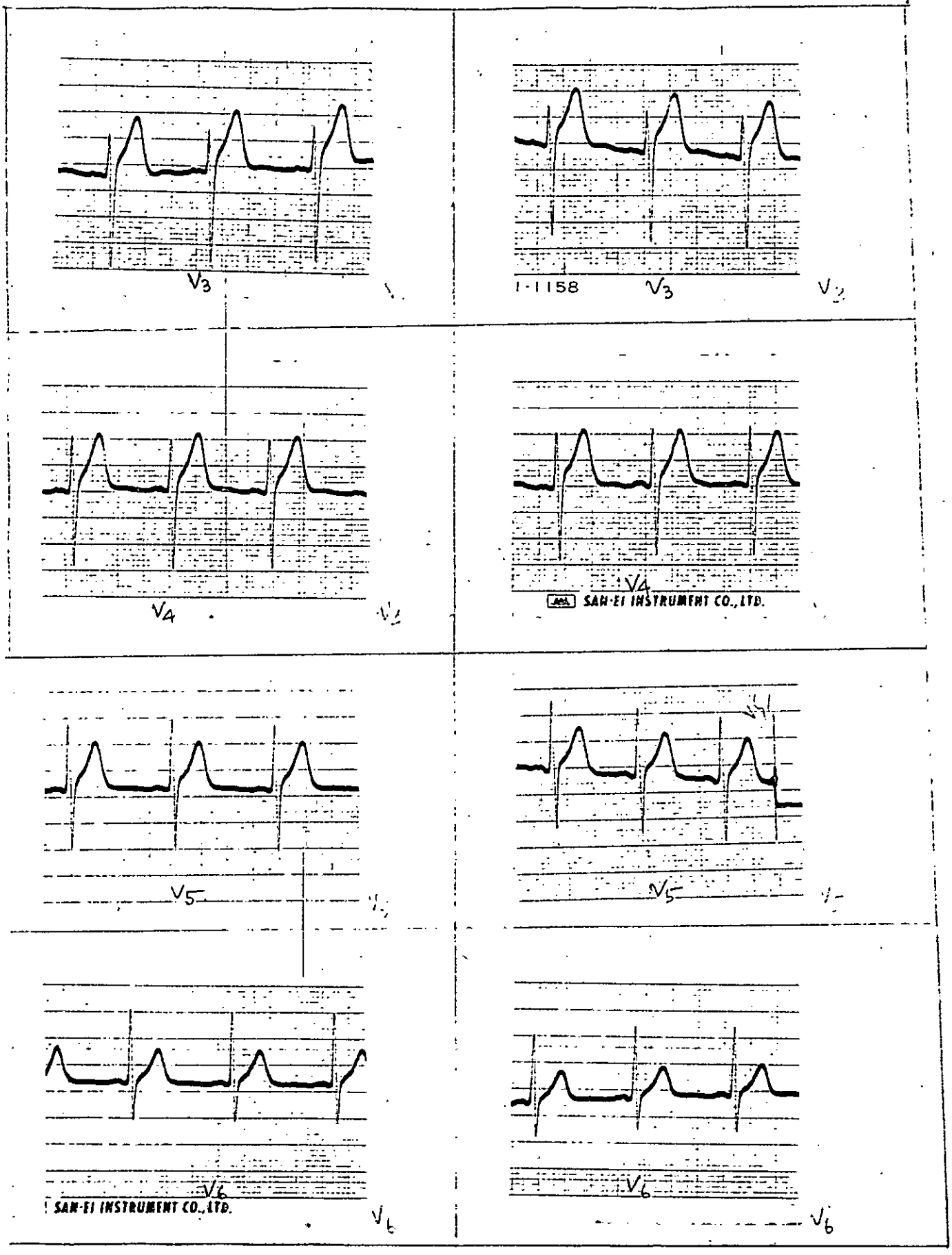


Person-1, AGE. 34 YEARS

FIG A-2. RECORDED ECG OF PERSON 1. LEAD USED, aV_L, aV_F, V_1, V_2.

NORMAL

WITH RESTRICTED BLOOD FLOW

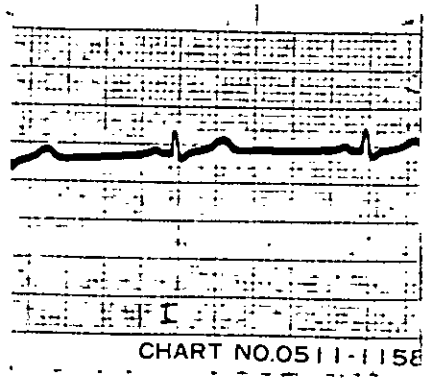


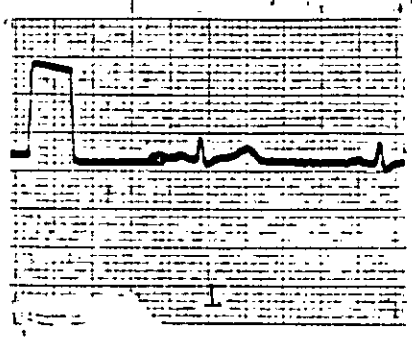
Person 1, AGE - 34 YEARS.

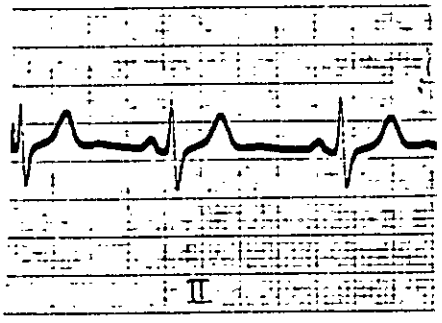
FIG. A-3. RECORDED ECG OF PERSON 1. LEAD USED V_3, V_4, V_5, V_6.

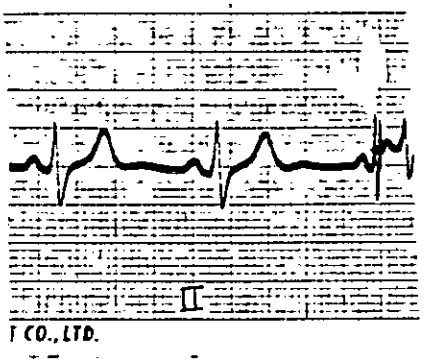
NORMAL

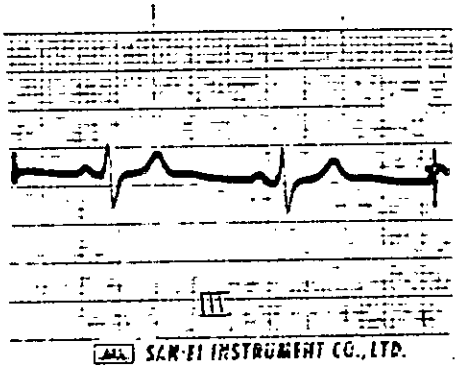
WITH RESTRICTED BLOOD FLOW

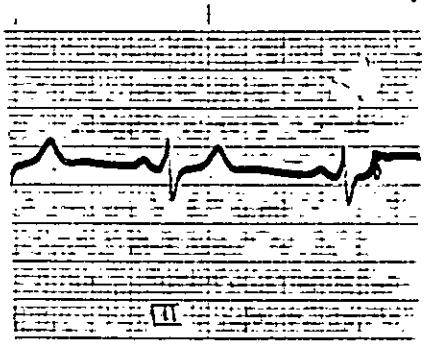


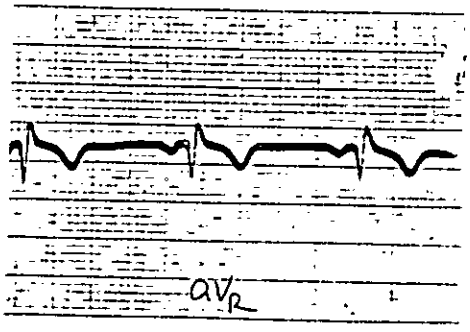


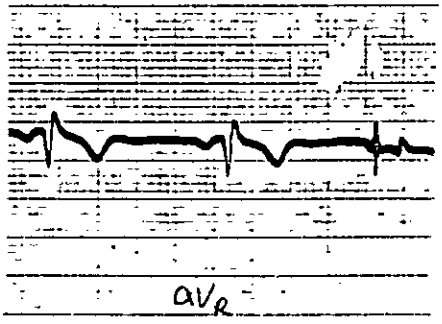










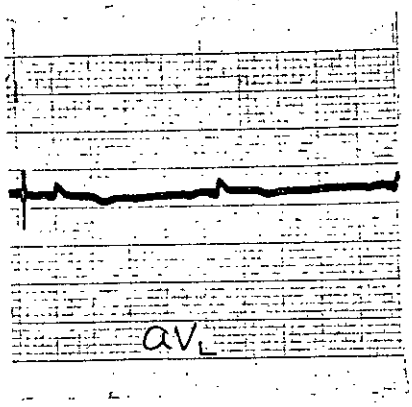


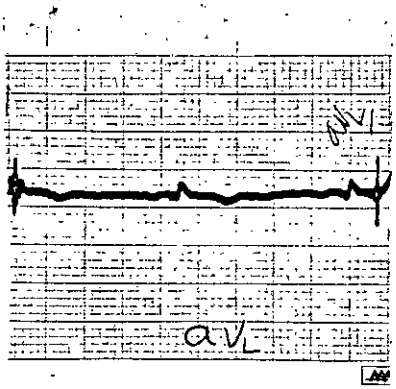
Person 2 Age: 36 yrs.

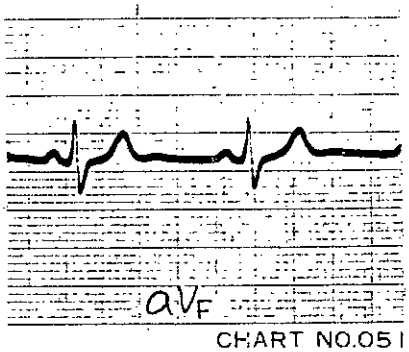
FIG: A-4. RECORDED ECG OF PERSON 2. LEAD USED; I, II, III, aV_R.

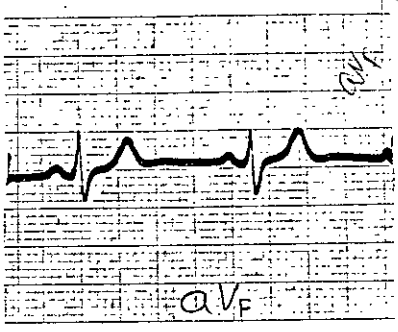
NORMAL

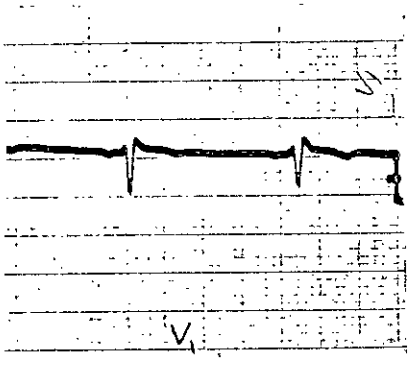
WITH RESTRICTED BLOOD FLOW.

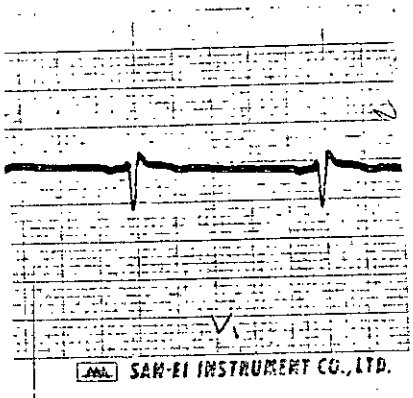


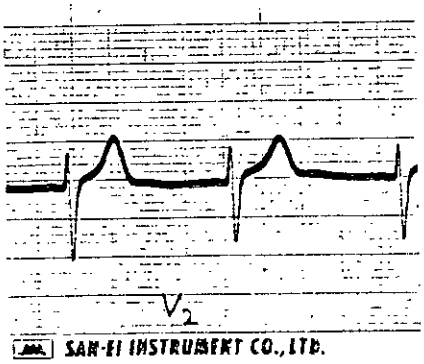


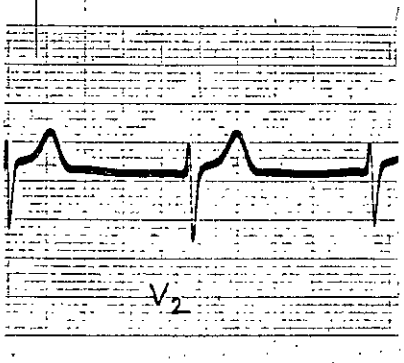












Person 2, AGE 36 YEARS.

FIG A-5. RECORDED ECG OF PERSON 2. LEADUSED aV_L, aV_F, V_1, V_2.

NORMAL

WITH RESTRICTED BLOOD FLOW

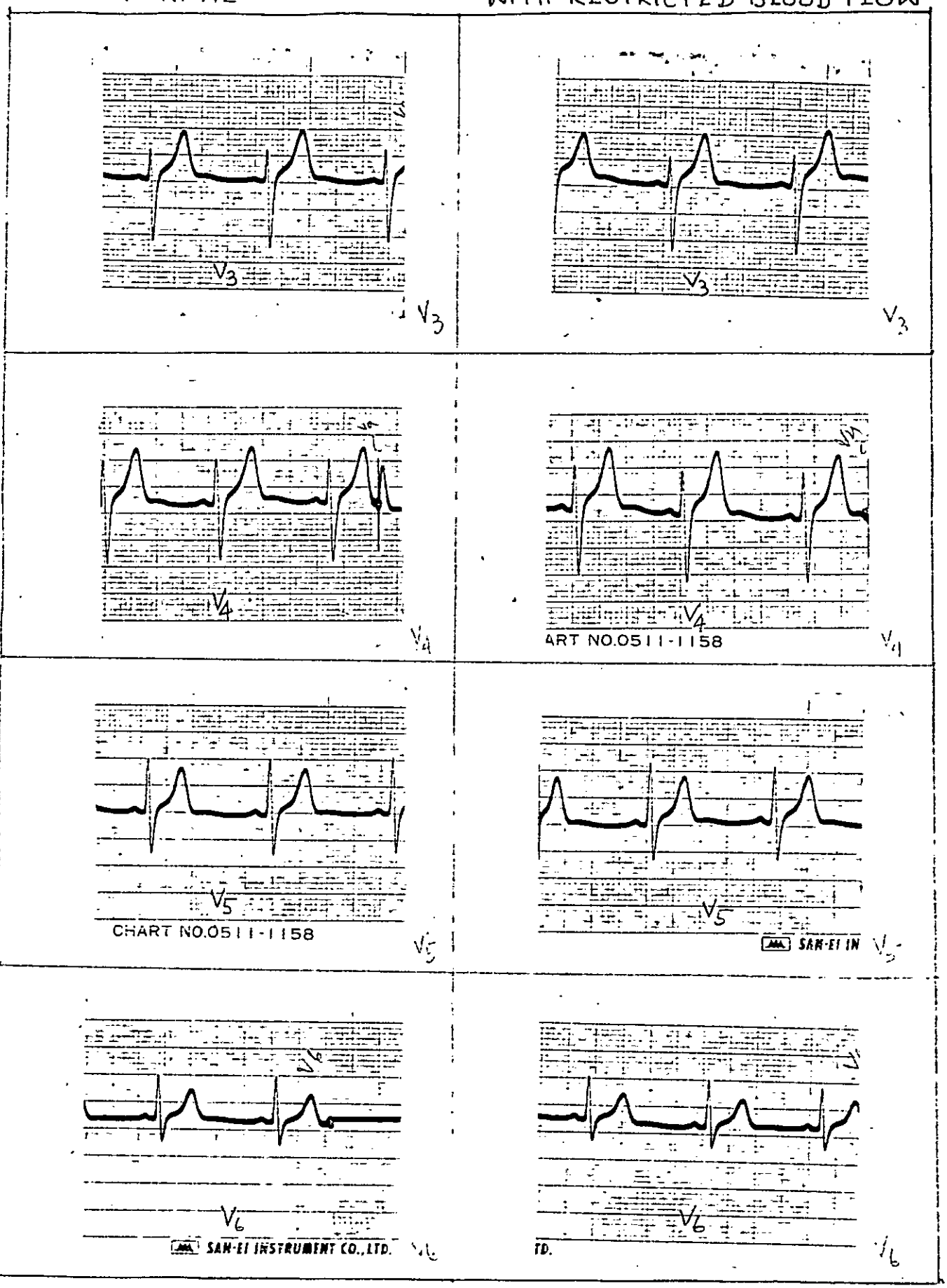
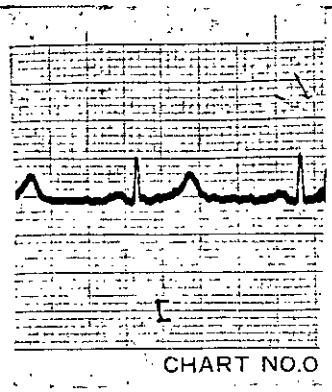
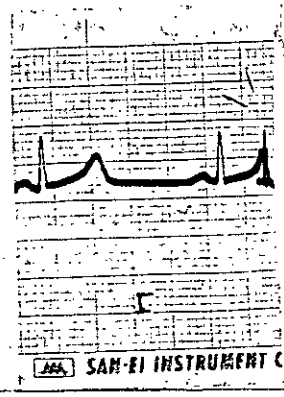


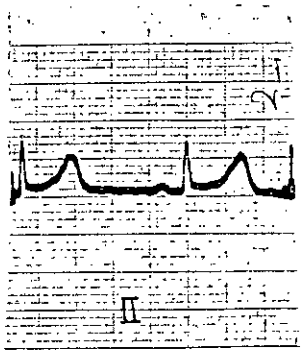
FIG. A.6. RECORDED ECG OF PERSON 2, AGE 36 YEARS. LEAD USED V_3, V_4, V_5, V_6.

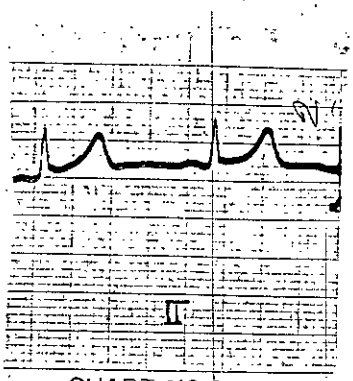
NORMAL

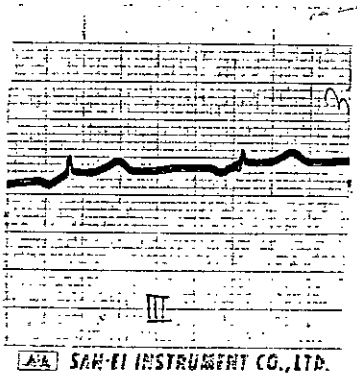
WITH RESTRICTED BLOOD FLOW

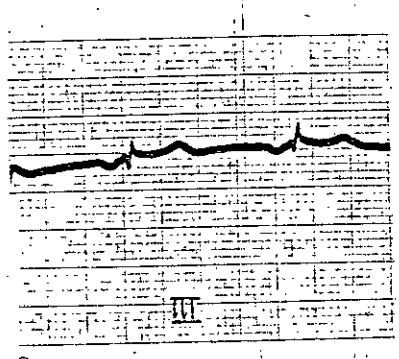


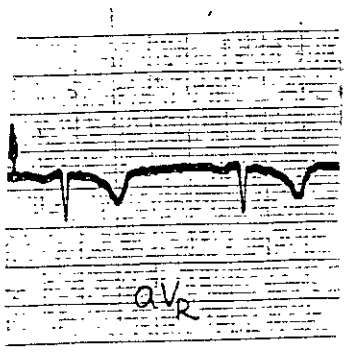


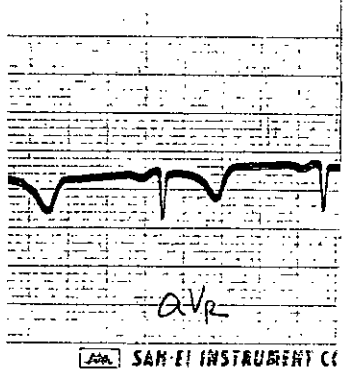










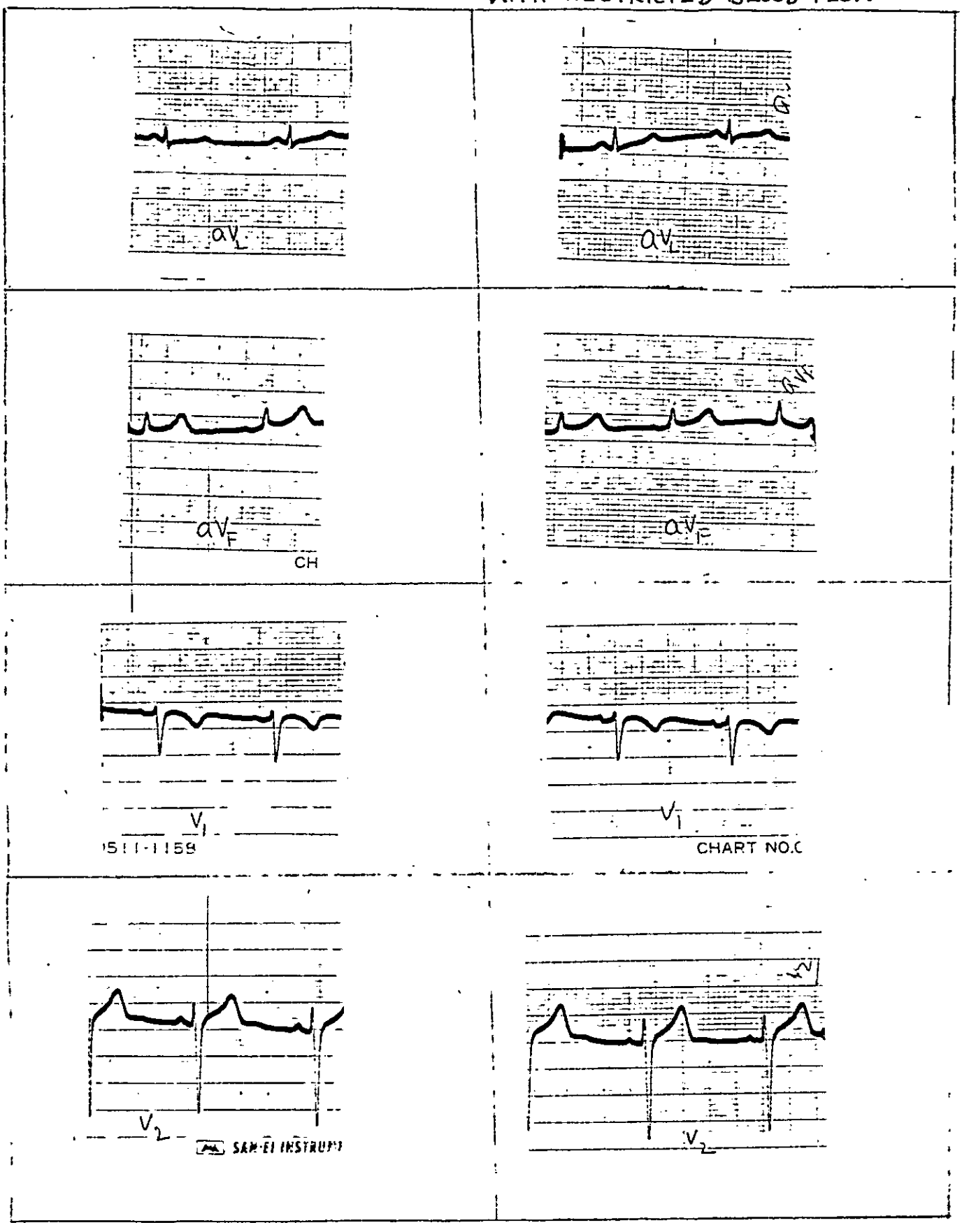


Person - 3 Age: 36 yr.

Fig A-7 · RECORDED ECG OF PERSON-3 · LEAD USED I, II, III, aV_R.

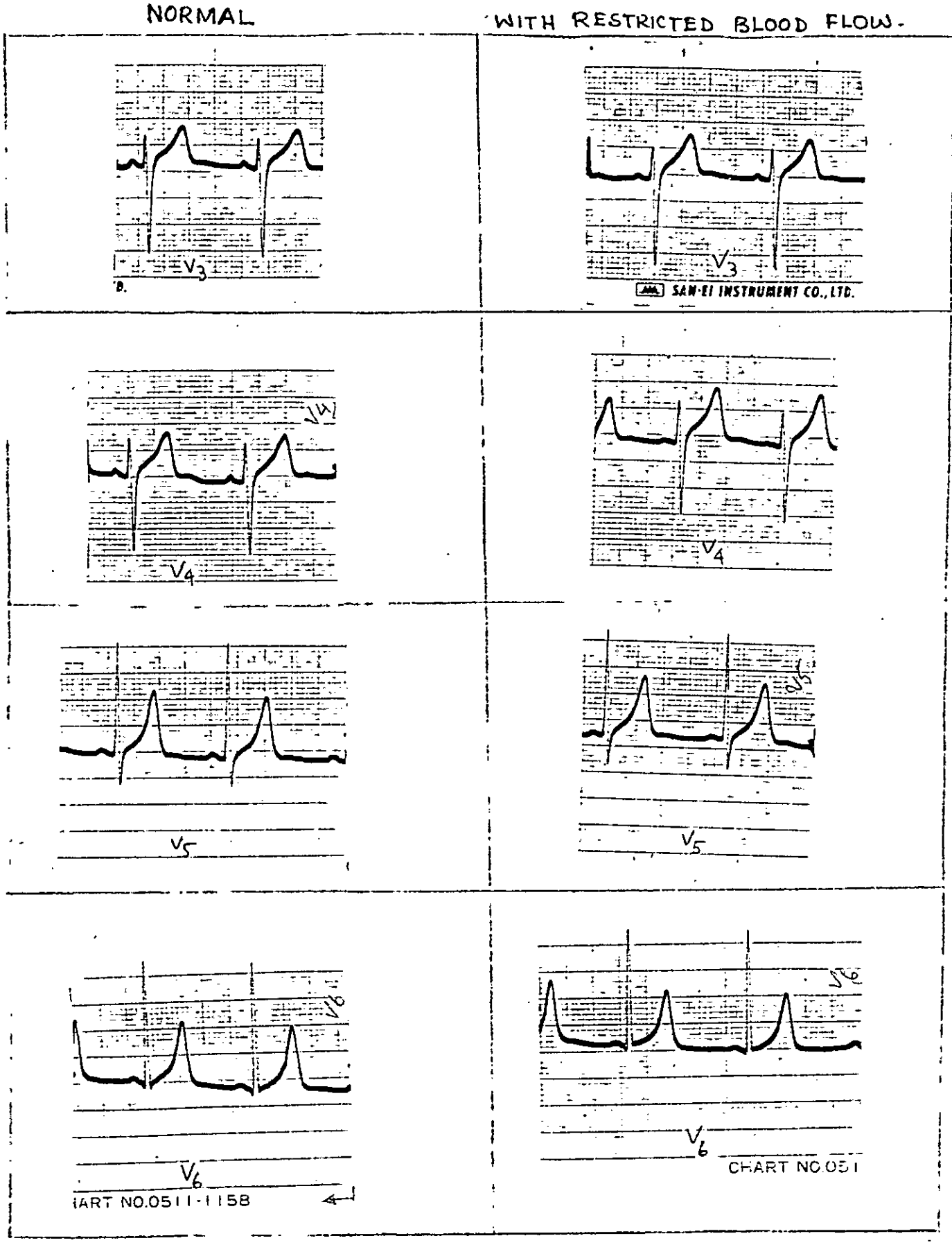
NORMAL

WITH RESTRICTED BLOOD FLOW.



Person 3, AGE - 36 YEARS.

FIG A-8. RECORDED ECG OF PERSON 3, LEAD USED aV_L, aV_F, V_1, V_2.

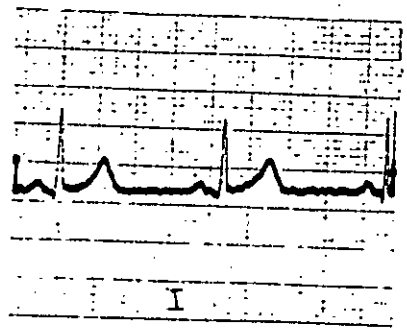


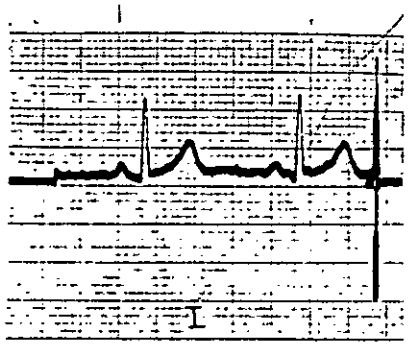
Person-3, 36 years of age.

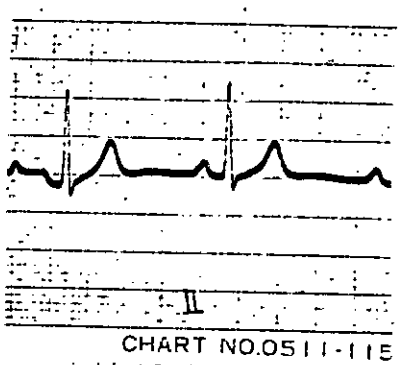
FIG A-9 : RECORDED ECG OF PERSON 3. LEAD USED V_3, V_4, V_5, V_6

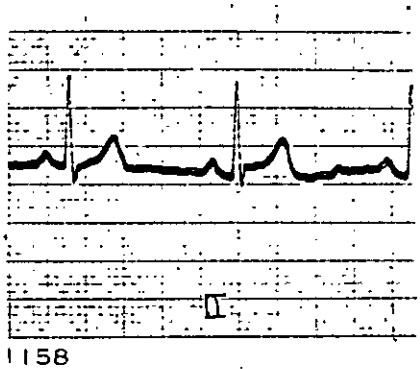
NORMAL

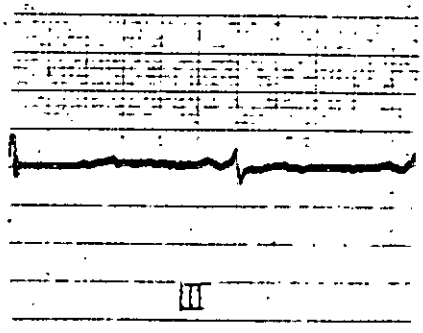
WITH RESTRICTED BLOOD FLOW.

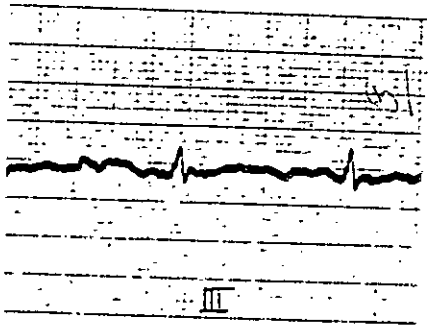


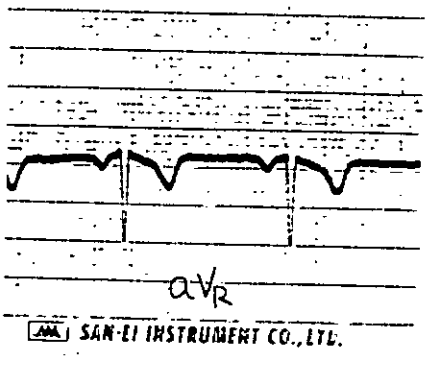


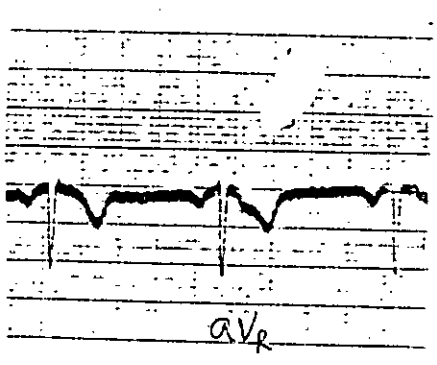






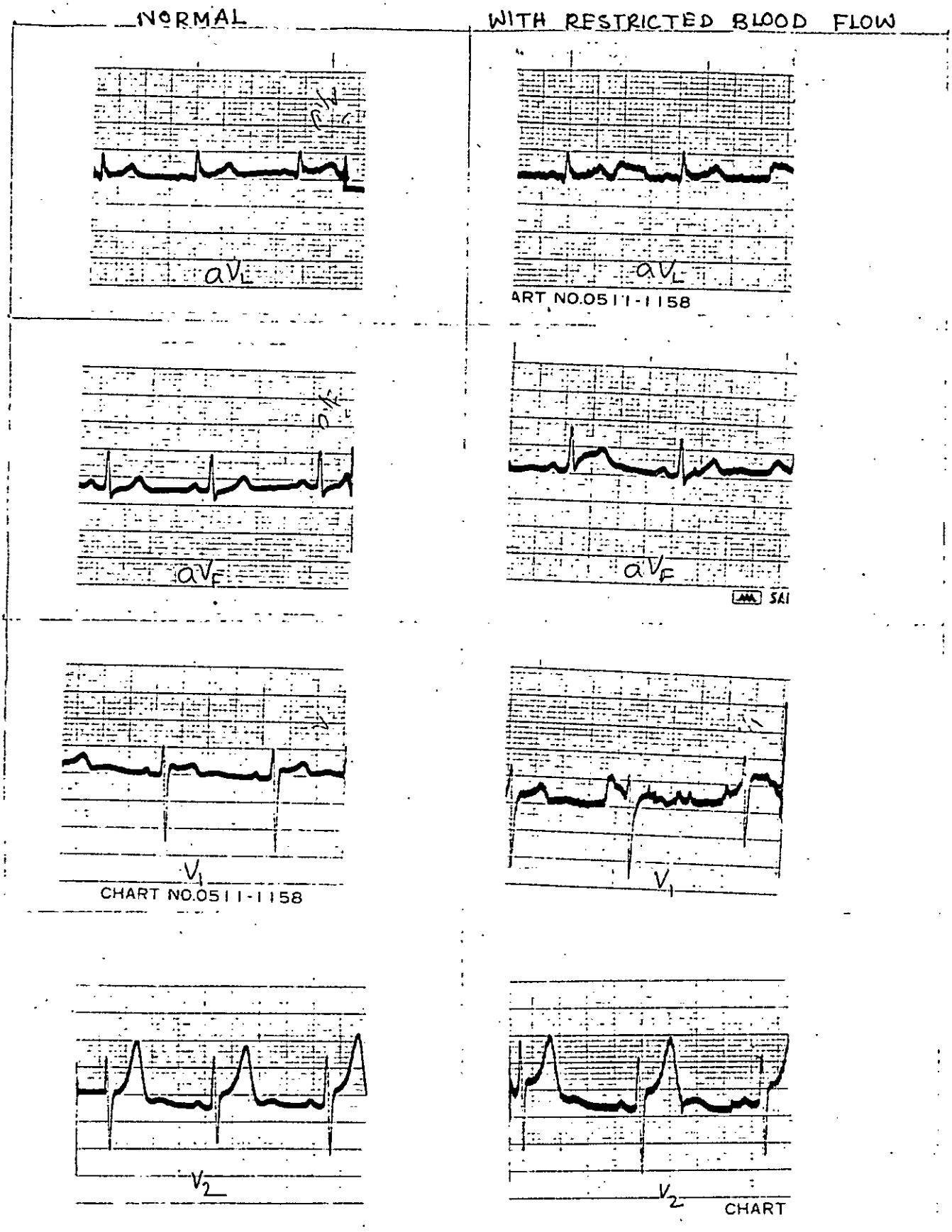






Person-4 Age-56

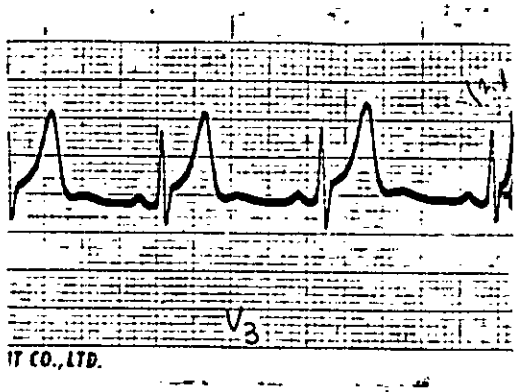
Fig. A-10. RECORDED ECG OF PERSON 4. LEAD USED I, II, III, aVR.

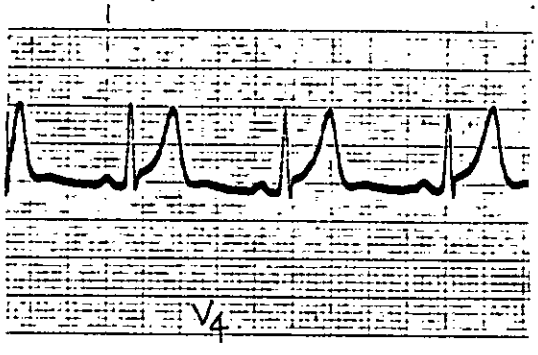


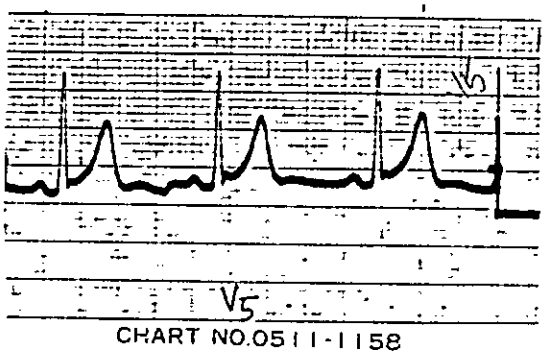
Person - 4, age 56 years.

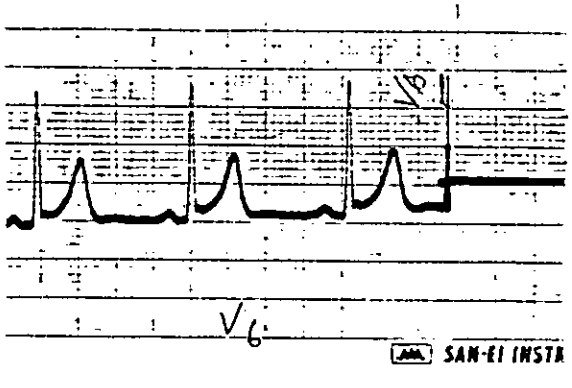
FIG A-11: RECORDED ECG OF PERSON-4, LEAD USED aV_L, aV_F, V_1, V_2.

NORMAL

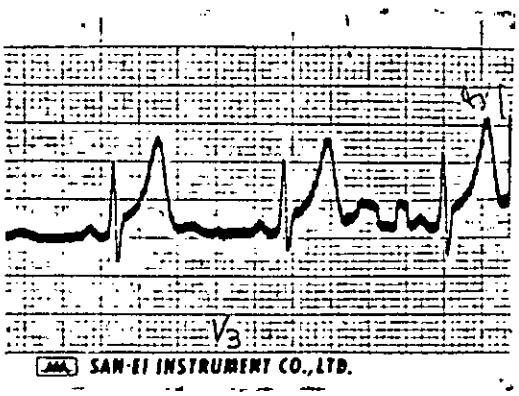


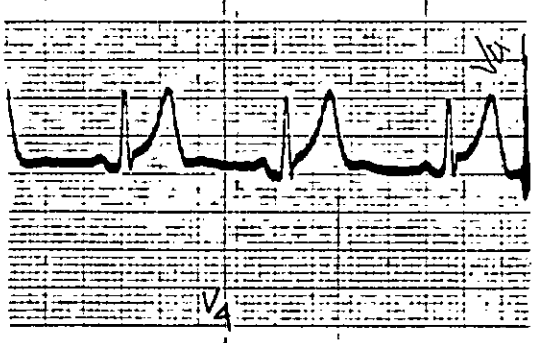


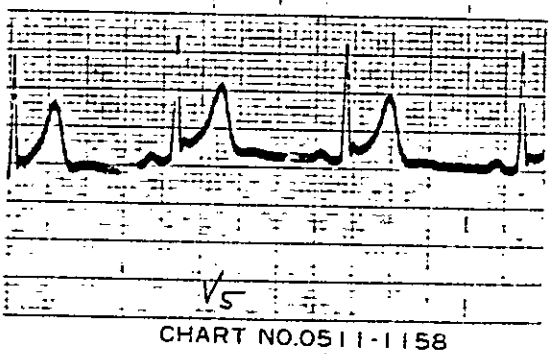


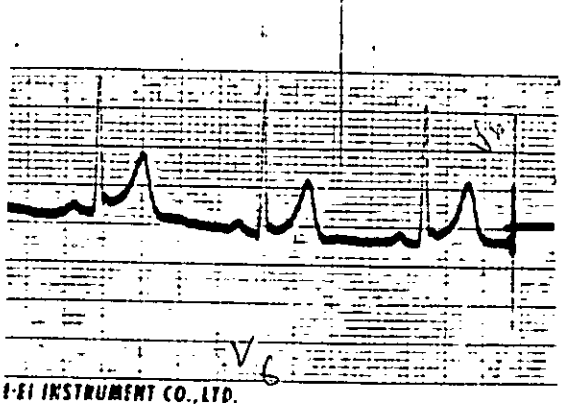


WITH RESTRICTED BLOOD FLOW







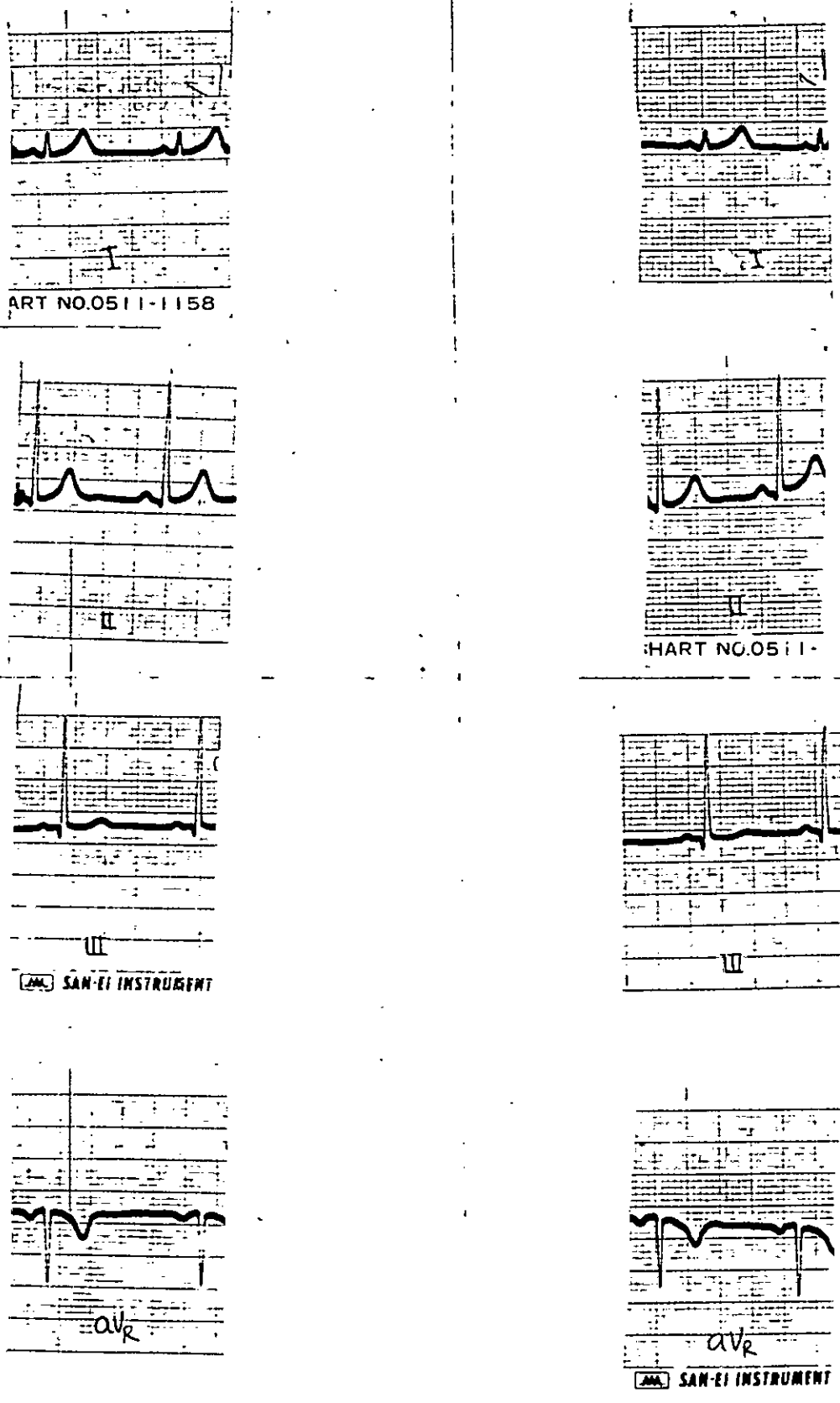


Person-4, age 56 years.

FIG.-A-12. RECORDED ECG OF PERSON-4, LEAD USED V_3, V_4, V_5, V_6

NORMAL

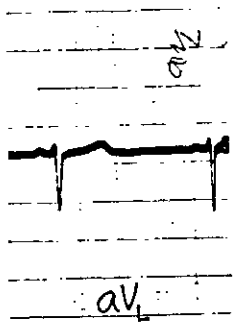
WITH RESTRICTED BLOOD FLOW.

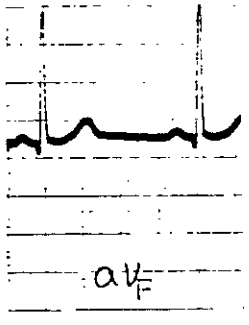


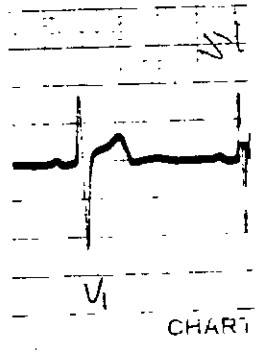
Person-5, Age-24 yr.

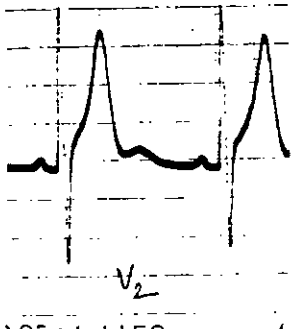
Fig A-13. RECORDED ECG OF PERSON-5, LEAD USED I, II, III, aV_R.

NORMAL

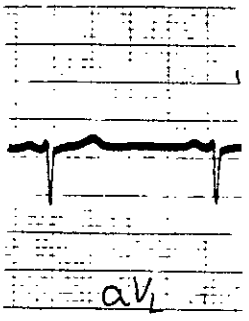


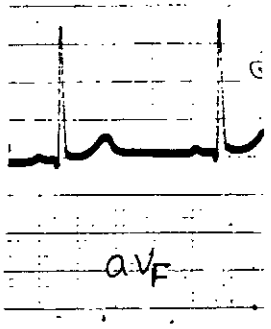


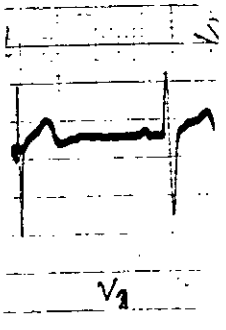


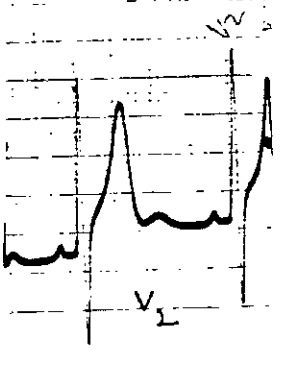


WITH RESTRICTED BLOOD FLOW





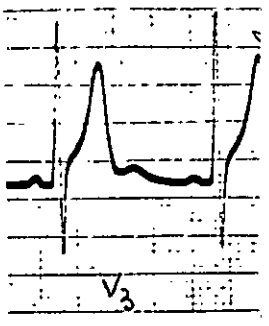


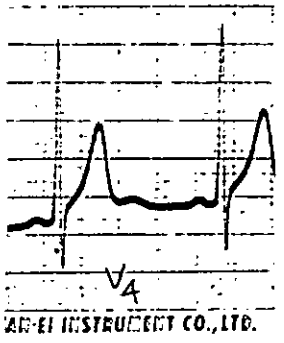


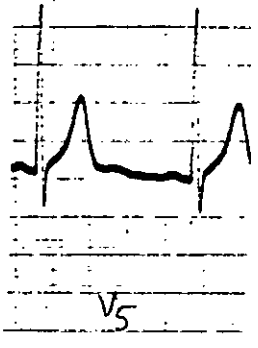
Person-5, age 24 years.

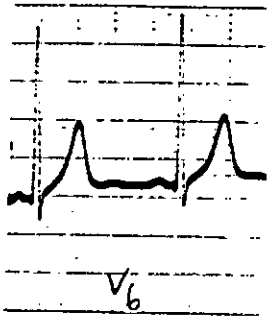
FIG: A-14: RECORDED ECG OF PERSON-5. LEAD USED aV_L, aV_F, V_1, V_2

NORMAL

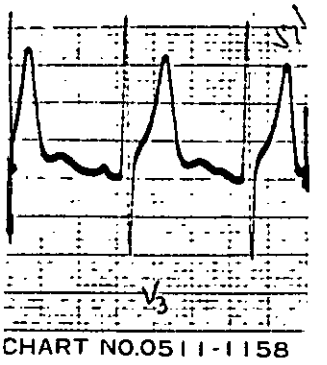


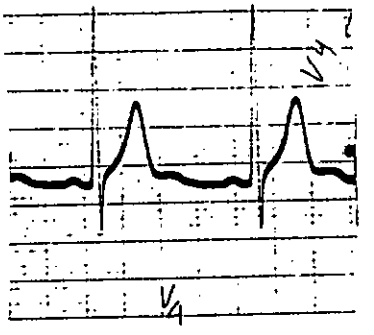


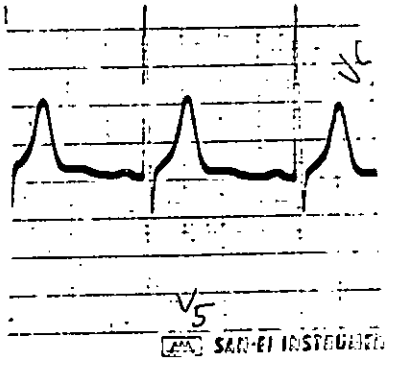




WITH RESTRICTED BLOOD FLOW







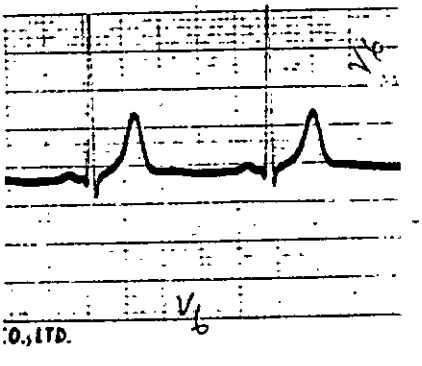
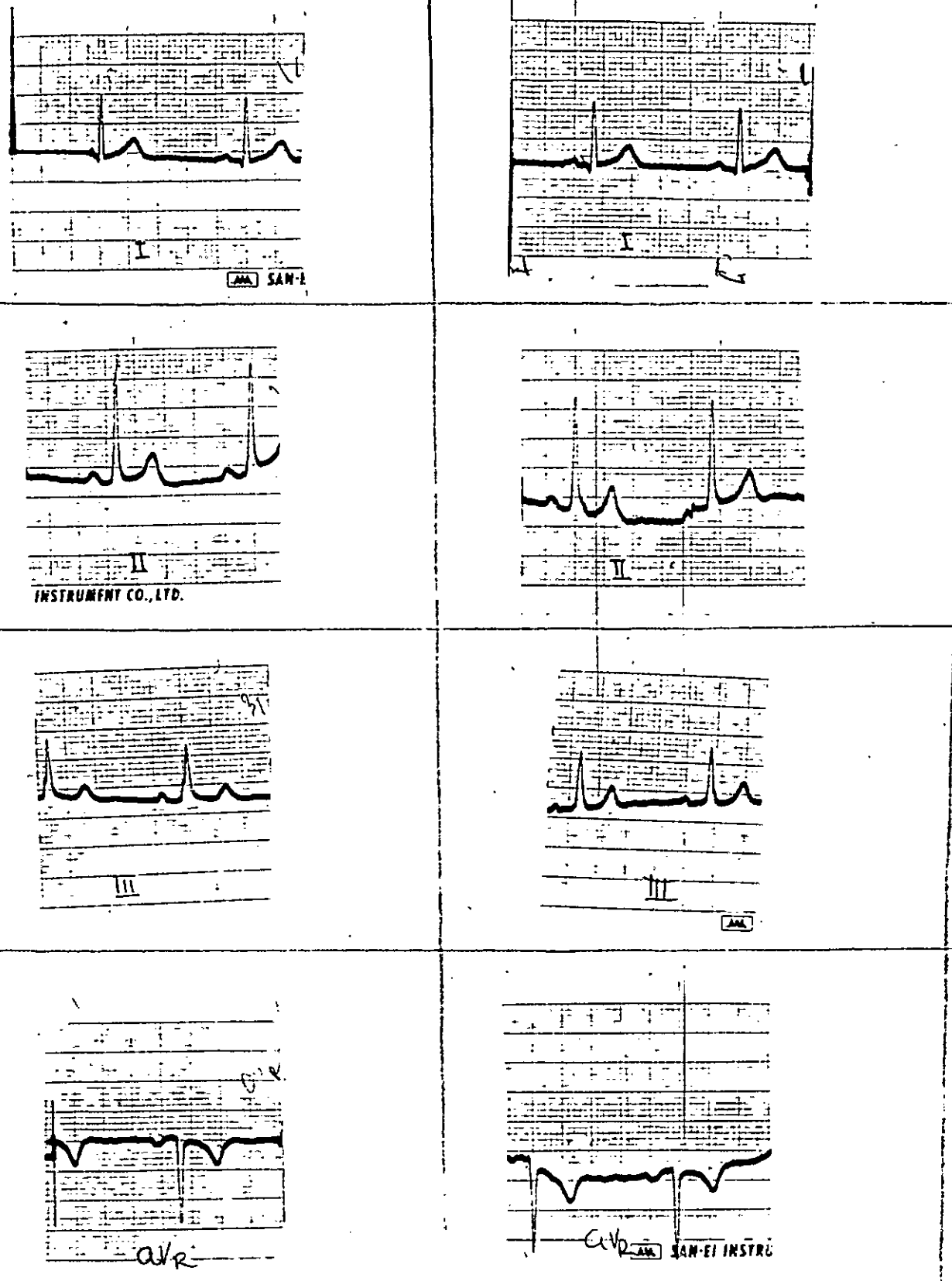


Fig. A-15. RECORDED ECG OF Person-5, age 24 years. PERSON-5. LEAD USED V_3, V_4, V_5, V_6.

NORMAL

WITH RESTRICTED BLOOD FLOW.

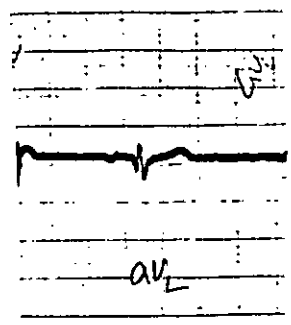


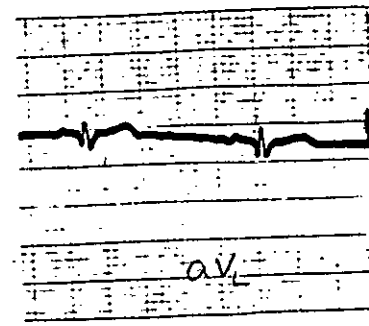
Person-6, Age - 22 yr.

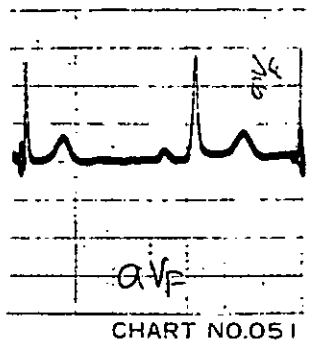
FIG. A-16. RECORDED ECG OF PERSON-6, LEAD USED I, II, III, aV_R.

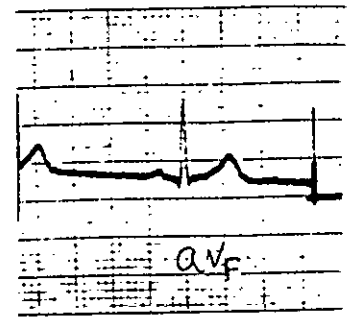
NORMAL

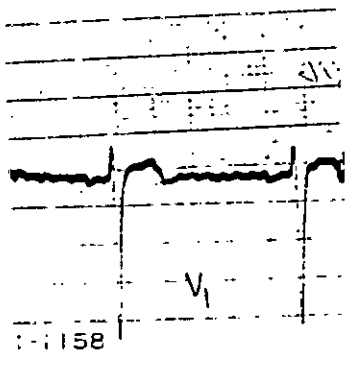
WITH RESTRICTED BLOOD FLOW

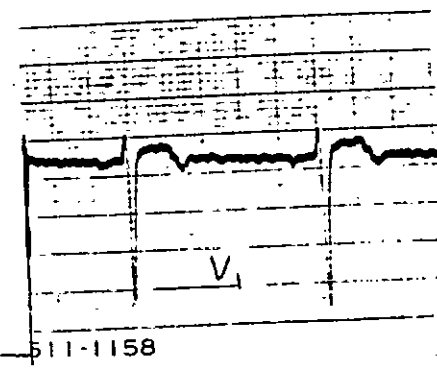


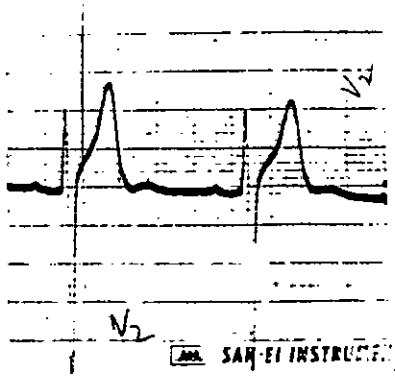


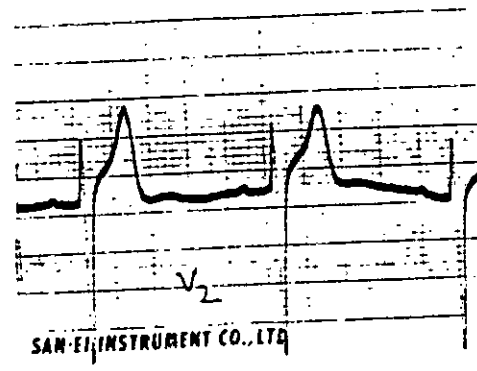










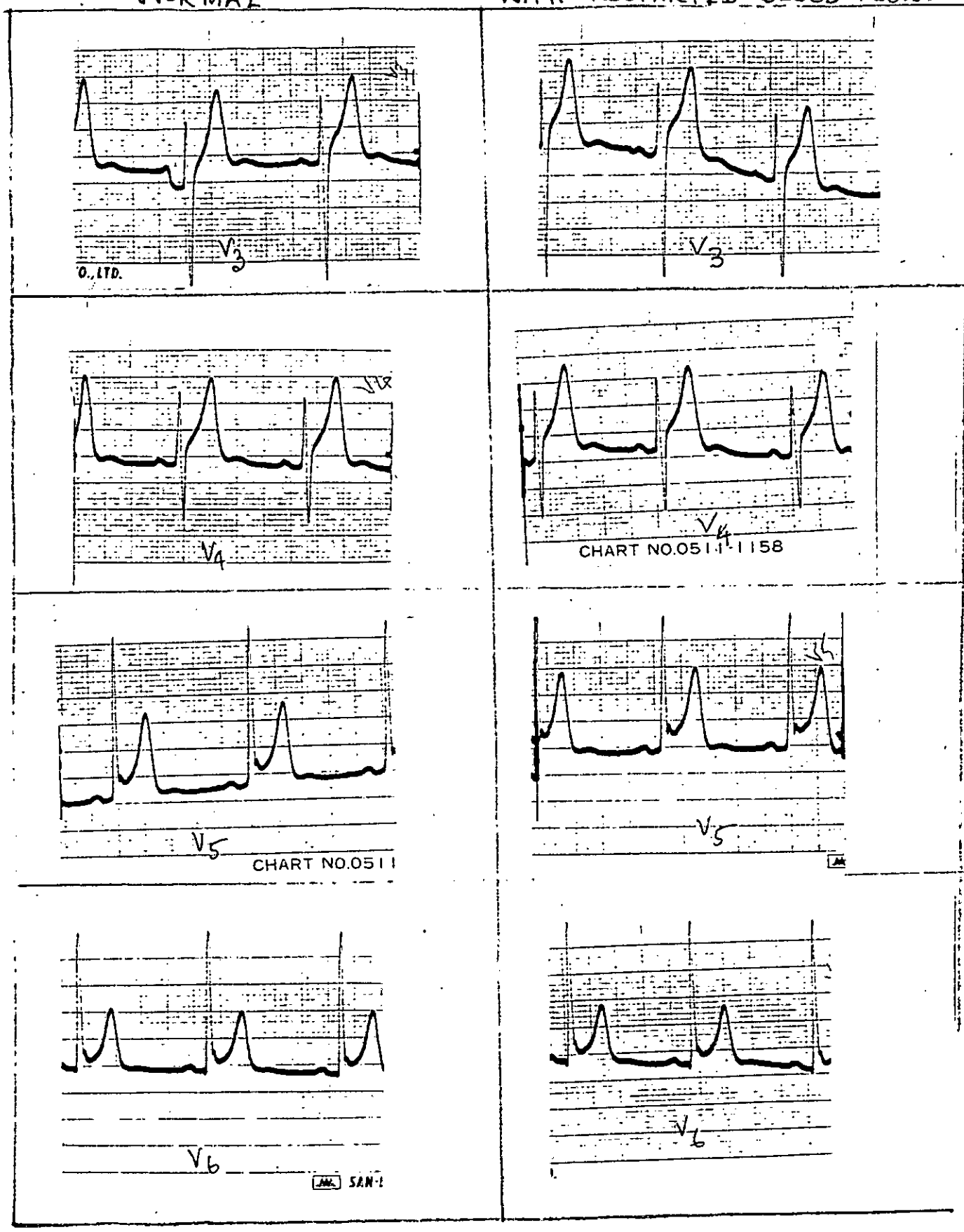


Person-6, age - 22 years.

FIG. A-17. RECORDED ECG OF PERSON 6; LEAD USED aV_L, aV_F, V, & V_2.

NORMAL

WITH RESTRICTED BLOOD FLOW.

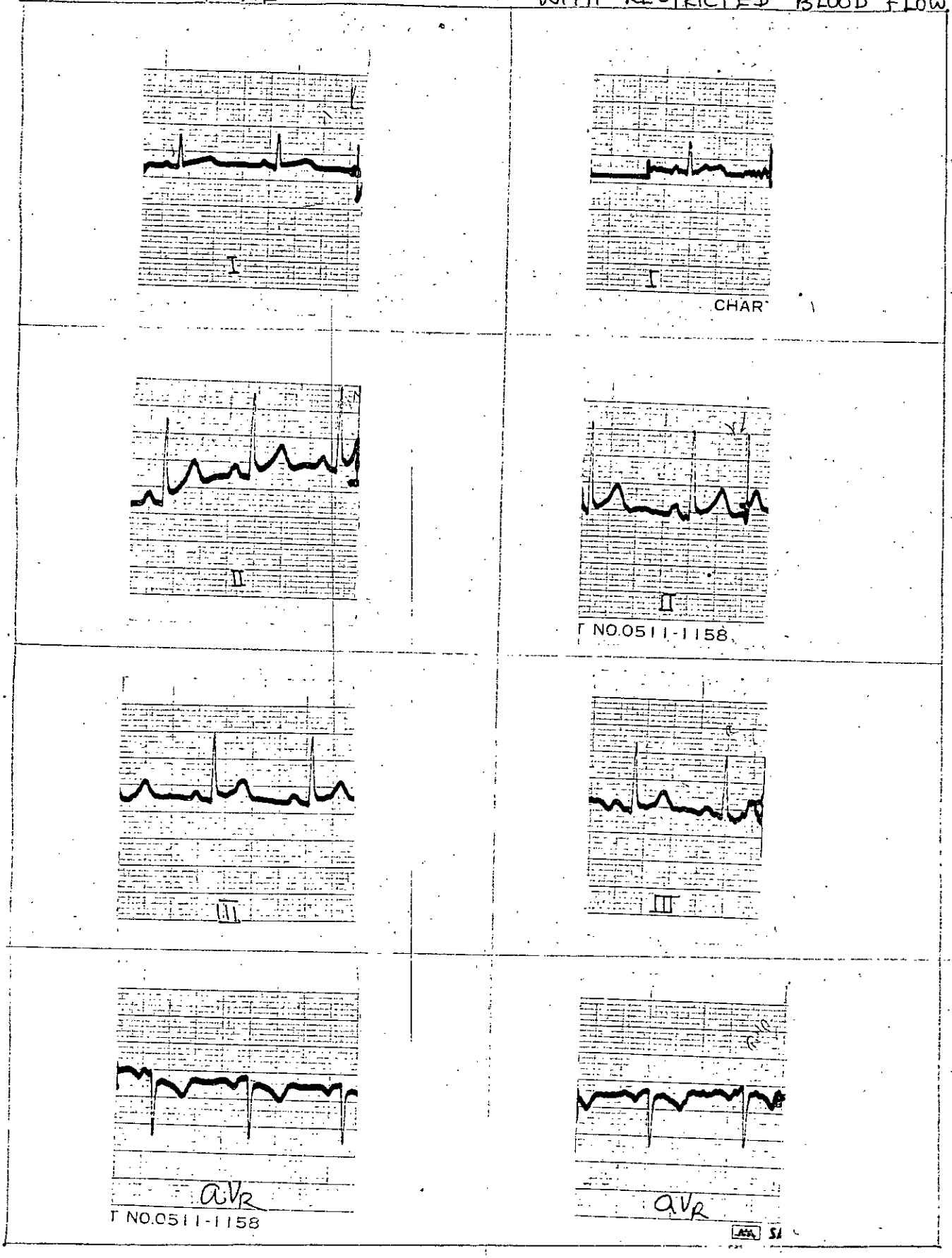


Person 6, Age 22 years.

FIG: A-18. RECORDED ECG OF PERSON 6. LEAD USED V_3, V_4 V_5. V_6.

NORMAL

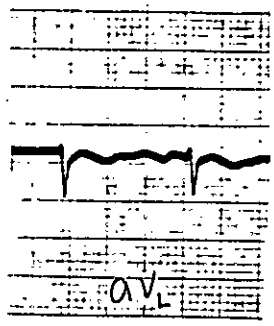
WITH RESTRICTED BLOOD FLOW



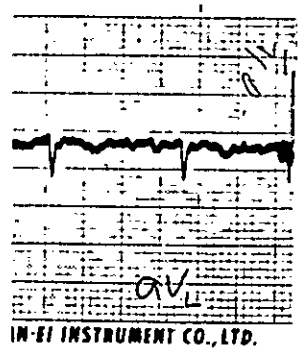
Person 7, Age 14 yrs.

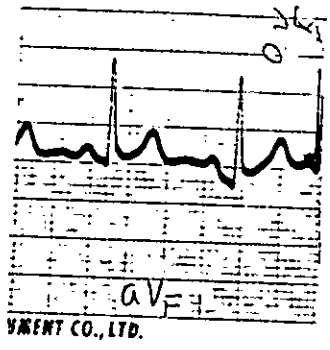
FIG. A-19. RECORDED ECG OF PERSON 7. LEAD USED I, II, III, aV_R.

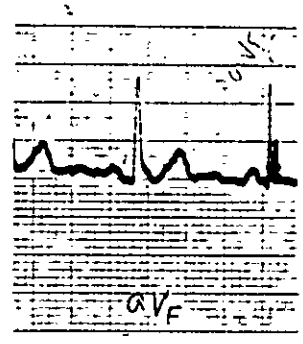
NORMAL

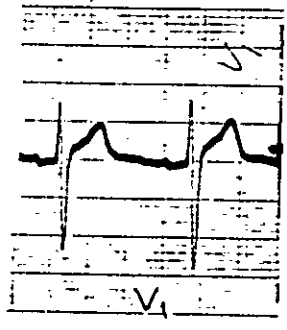


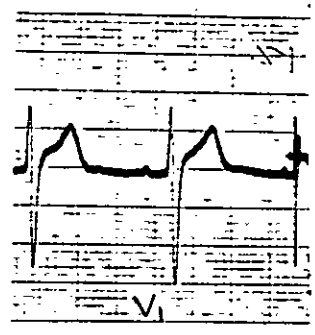
WITH RESTRICTED BLOOD FLOW.

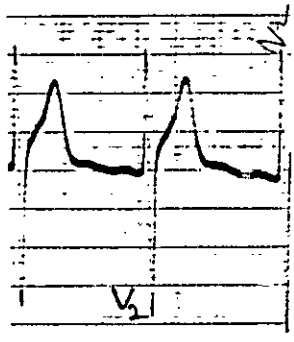


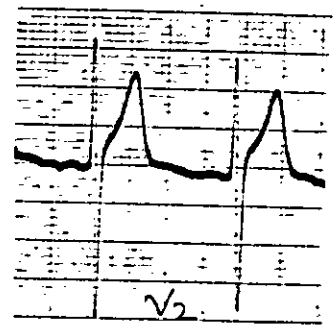












Person 7 age - 14yrs.

FIG. A-20. RECORDED ECG OF PERSON 7. LEAD USED aV_L, aV_F, V_1, V_2.

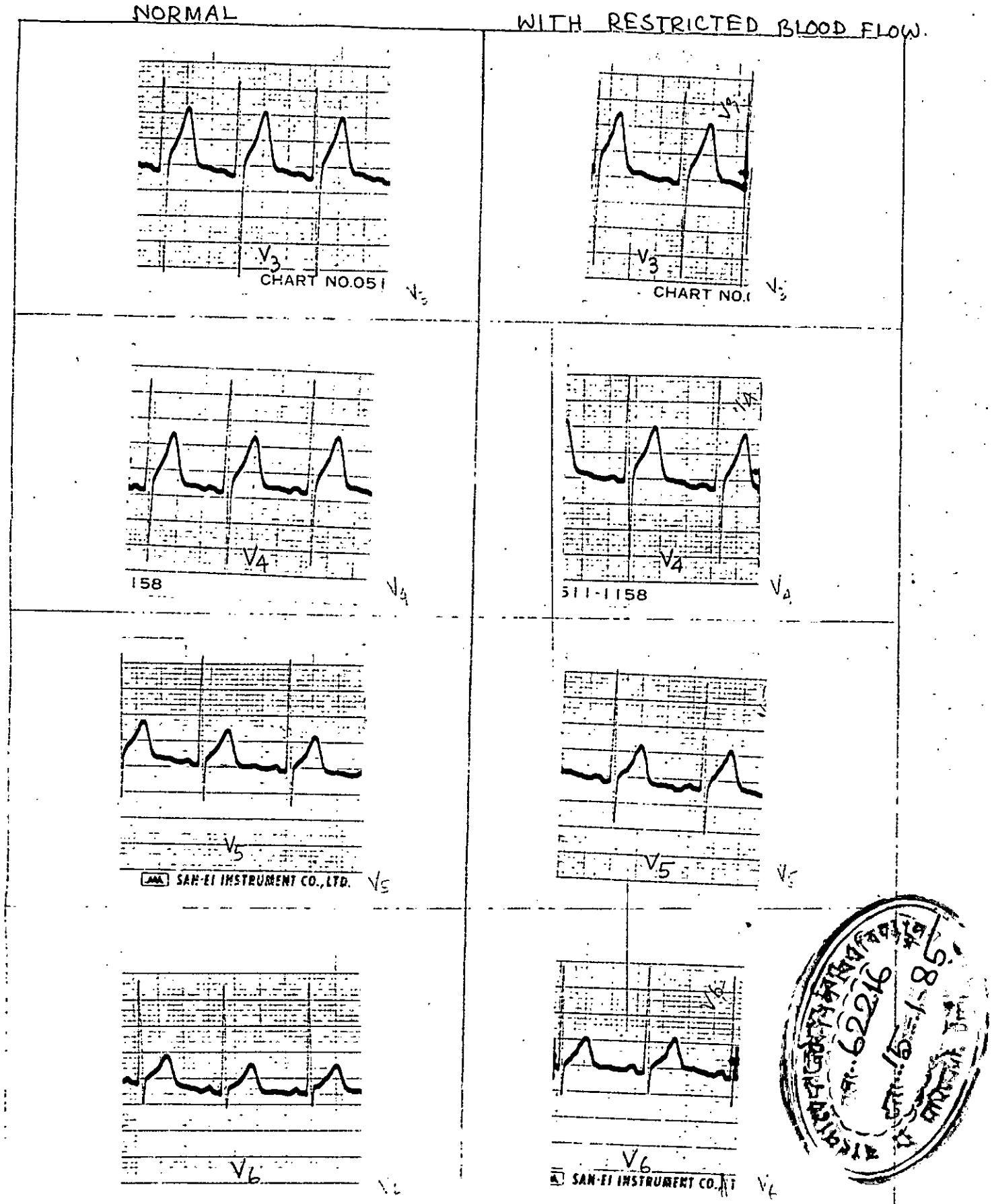


FIG. A-21. RECORDED ECG OF PERSON 7, age 14 years. PERSON 7. LEAD USED V_3, V_4, V_5, V_6.