within a few days, urgent transfer to a surgical unit should be arranged. The neural plaque should be covered with a sterile dressing kept moist with sterile saline or aqueous chlorhexidine solution. The infant should be kept warm during transfer in a portable incubator, and the father should, if at all possible, accompany him to discuss his management further.

The back should be closed and any gross kyphosis corrected within 48 hours (preferably 24 hours) of birth as the risk of infection of the back and the meninges becomes too great if there is further delay. With satisfactory wound healing, full assessment can then begin of the urinary tract, of leg activity and deformity, and of the hydrocephalus, and appropriate early treatment started to minimize these disabilities.

Prognosis for the Child

The wide variety of form and extent of the lesions of the spinal cord, and the varying use of descriptive terms for them, make classification and comparison of different series of results difficult. But most experts agree that a policy of early surgical treatment of open myelomeningocele ensures that a higher proportion of children survive who are either normal or who have only minor handicaps. Varying degrees of paraplegia and urinary incontinence occur in half the cases, but are coped with remarkably well by those children who are of reasonable intelligence.

The results of treatment of hydrocephalus both in isolation and associated with myelomeningocele depend largely on the amount of associated brain damage or the presence of any other deficit. Nowadays the surgeon can almost always control the actual size of the head, so that the vast or unmanageable heads of untreated hydrocephalus should be a thing of the past. Measurements of the intelligence quotient, for what they are worth, have shown surprisingly good results in many of the treated children who are now reaching school age. No long-term detailed studies are yet available.

Prognosis for the Family

The arrival of a new child in a family always has profound effects on that family. If the child is malformed the effects are far reaching and may be disastrous. The fact of the infant's malformation is apt to produce feelings of inadequacy and guilt especially in the mother, and she needs reassurance and help. Early death may seem to solve a good many problems in the doctor's view, but not so obviously to the parents. If the child survives and is handicapped then the parents will continue to need a great deal of support and practical help and advice on how to handle him in relation to any siblings. Finally, the parents will always be anxious about the outcome of any future pregnancies. In Britain any pregnancy carries risk of 0.4% of spina bifida cystica. If a family already has one child with a major anomaly of the central nervous system, then the risks in future pregnancy are perhaps as high as 4%. If there are two or more close relatives who have had such lesions, the risk rises to 20%, which is very high indeed.

Conclusion

The proper course of treatment for the infant with a myelomeningocele is the subject of considerable debate at present. The survival rate of children with myelomeningocele with prompt surgery is about 60%, as opposed to 15% with no treatment. In my opinion early and vigorous treatment is justified in the great majority of cases by the considerable advantages accruing for the operated child compared with the increased disability in the unoperated child who survives.

Early treatment of hydrocephalus is again essential if brain damage is not to progress, and investigation and surgery in the neonatal period should be undertaken.

Small "closed" lesions of the spine are not associated with the same urgency, but early referral to a specialized unit for full assessment is desirable.

Scientific Basis of Clinical Practice

Electrical Activity of the Heart

D. R. WESTBURY

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The heart is essentially a specialized muscle. Its function is to contract rhythmically and by its contraction move blood from an area of low hydrostatic pressure in the great veins to an area of high hydrostatic pressure in the aorta. So the heart is a muscular pump whose activity provides the work necessary to drive the blood round the circulation. As in any other sort of muscle, mechanical contraction depends on the electrical activity of the cells. To function efficiently as a pump, a certain sequence of events must occur—the heart cycle. This sequence of events is controlled by the electrical pro-

perties of the cells, and the heart beat owes its inherent rhythmicity to these characteristics.

Membrane Potential

Heart cells are like other muscle cells. They have similar constituents including actin and myosin filaments forming the contractile machinery, and a cell membrane. Like other cells there is an electrical potential difference between the inside and the outside of the heart cell, the inside being negative with respect to the outside in the resting state. The electrical properties of the cells depend largely on the properties of the membrane. This is selectively permeable to small inorganic ions, and its permeabilities to these ions varies during the heart cycle because of changes in the potential difference across the membrane. Conversely, the potential difference

Department of Physiology, The Medical School, Birmingham
D. R. WESTBURY, B.SC., B.M., B.CH.,

across the membrane depends on the relative permeabilities of the membrane to ions. This complex interplay of ionic permeabilities and membrane potential determines the activity of the heart cells.

The fluid inside the heart cells contains mainly potassium ions and large organic anions of many species, together with some sodium, chloride, and calcium. The extracellular fluid contains principally sodium and chloride ions with some potassium. These relatively high concentrations of potassium ions and low concentrations of sodium ions inside the cells are maintained by the metabolism of the cells. In particular, sodium ions from inside the cell may be exchanged for potassium ions from outside by the sodium pump mechanism of the membrane. As both ions are positively charged, such an exchange does not contribute significantly to the transmembrane potential, but as the concentration gradient of each increases energy must be supplied by the cell. The ionic events occurring during one heart cycle do not significantly alter the concentrations of the ions inside and outside the cells as the actual quantities of ions involved in the movements are so small. Nevertheless, such movements of ions, which are charged particles, produce relatively large changes in the potential difference between the inside and the outside of the cells.

IONIC BALANCE

The ionic balance occurring on each side of the cell membrane during the resting part of the heart cycle (diastole) is shown in Fig. 1. In its resting state the cell membrane is permeable to potassium ions and to a certain extent to chloride ions also. Sodium ions, which have a relatively large hydration shell of bound water molecules, can pass through the cell

FIG. 1—A representation of the distribution of ions on each side of the cell membrane in the resting state (diastole). The large concentration of potassium ions inside the cell, and of sodium and chloride outside are represented by large letters. The arrows indicate the tendency for ions to move down their concentration gradients. Such movements are opposed by the negative intracellular potential, establishing an equilibrium.

membrane only with difficulty in the resting state, and large organic anions cannot pass through the membrane at all. In the resting state the behaviour of potassium and chloride ions will dominate the situation. Potassium ions inside the cell will tend to move down their concentration gradient through the cell membrane to the outside. In doing so they will carry positive charge out, making the inside of the cell negative. At the same time chloride ions will tend to diffuse into the cell down their concentration gradient carrying negative charge to the inside. Thus the movement of these ions will build up an electrical gradient which opposes their movement down their concentration gradients. In the resting state, an equilibrium will be established in which the forces of the concentration

gradients are balanced by those of the electrical gradient. As sodium ions and organic anions are fixed on their respective sides of the membrane, the situation is one of a Gibbs-Donnan equilibrium. The inside of the cell will be negative with respect to the outside. The value of this polarization will depend upon the precise concentrations of potassium and chloride ions in each cell, and will be different in the different tissues of the heart. Sinus node cells have rather low membrane potential, perhaps -60 mV (volts $\times 10^{-3}$); atrial and ventricular cells have a rather high potential, perhaps -80 mV.

THE ACTION POTENTIAL

When heart cells receive a stimulus which reduces the resting polarization towards zero—that is, a depolarizing stimulus—the situation at the cell membrane is changed and an action potential is produced. Such a depolarizing stimulus would normally come from another heart cell, or it could come from an electrical stimulator or an external electrical pacemaker. During an action potential the inside of the cell becomes positively charged and remains so for a brief time, about 0.1 sec. This electrical change causes a mechanical contraction and corresponds roughly to the systole of that heart cell. The action potential of heart cells is different in its duration and shape from those of nerve cells and of other muscle cells, but is probably generated by a similar mechanism.

Fig. 2 shows schematically an action potential, recorded from the inside of an atrial or a ventricular cell. The action potential rises very rapidly and at its peak the cell becomes positively charged inside with respect to the outside to the extent of about $+20$ mV. The membrane potential then falls back to a plateau at or rather above zero, and remains at this value for about 0.1 sec. Then the membrane repolarizes fairly rapidly to the resting state, where the cell is negatively charged inside. The actual size and shape of the action potential varies from one heart tissue to another because the precise properties of the cells vary.

The changes in membrane potential reflect changes in the permeability of the membrane to ions. These changes are not fully understood, nor are the mechanisms underlying them in the membrane known. The membrane has sodium and potassium permeabilities which depend upon the potential difference across the membrane. When a depolarizing stimulus reduces the membrane potential the properties of the membrane change. The permeability to potassium ions decreases to a low level and the membrane becomes very permeable to sodium ions. This change in the membrane properties has a definite threshold for its initiation at a value of membrane potential which varies from cell to cell, but is about -50 mV. The increase in sodium permeability is very rapidly established and allows sodium ions to move into the cell down their concentration gradient. The movement of sodium ions carries positive charge into the cell causing the rapid rising phase of the action potential and the overshoot into the positive region. A sodium ion electrochemical equilibrium is not reached because the sodium mechanism is rapidly inactivated, and the sodium permeability falls back towards its normal low value. Nevertheless, it remains considerably above its resting value for about 0.1 sec and potassium permeability remains low. This causes the membrane potential to be held at about zero for the duration of the plateau. As the membrane potential gradually falls a value is reached at which potassium permeability rises again to its resting value, causing fairly rapid repolarization because potassium ions are able to move out of the cell carrying positive charge and re-establish the resting state with its potassium-dominated equilibrium.

The changes in ionic permeabilities which are believed to occur during the action potential are shown in Fig. 2. The depolarization—that is, the action potential—is responsible for activating the contractile mechanism of the cell and a mechanical contraction occurs which is coupled to the electrical behaviour of the cell. For the duration of the action potential

and for a short time afterwards, the heart cells are inexcitable by another depolarizing stimulus. As this refractoriness lasts about as long as the mechanical contraction, the cells can produce only a series of twitches. Each single contraction corresponds to mechanical systole.

CONDUCTION OF ACTION POTENTIAL

An action potential initiated at one point on a heart cell will be conducted along the muscle fibre at a speed which is characteristic of the fibre. The ionic solutions inside and outside the cells are conductors of electrical current. So a region of depolarization (the action potential) is connected to a region which is in the resting state and negatively polarized. A current will flow, tending to depolarize the resting membrane. When the threshold is reached an action potential will be set up in this next section of the fibre and so on. The action potential is conducted along the muscle fibres without any decrease in its size. The speed at which the conduction occurs depends on many factors. Of particular importance are the diameter of the fibres, the extent to which they branch, and the electric current available to depolarize the next section of the fibre—which depends on the resting membrane potential and the rate of rise of the upstroke of the action potential.

Individual muscle fibres meet one another end to end. These junctions consist of complex foldings of the two cell membranes in close contact and are called "intercalated discs." The cells are in relatively good electrical contact at these junctions allowing action potentials to be conducted from cell to cell over the whole myocardium. The heart is thus an electrical and functional syncytium. An action potential initiated at a pacemaker cell in the sinu-atrial node will spread over all the heart cells as a wave of electrical activity closely followed by one of mechanical activity. This wave of activity is called the heart cycle.

The Heart Cycle

The electrical events of the heart cycle begin at a pacemaker cell in the sinu-atrial node. The action potential generated here is conducted slowly, at about 5 cm/sec, out to the atrial cells. It then passes as a wave of activity over the thin muscular walls of the atria as a wavefront in two dimensions, like a pool of water spreading over a surface. The speed of conduction is about 1 m/sec. The action potential then reaches the atrioventricular node, which it must traverse to reach the bundle of His. The cells of the atrioventricular node have a low membrane potential, and the action potential has a low rate of rise. The cells are rather small and there is much branching. As a result the velocity of conduction through the node is low, about 5 cm/sec. This causes a delay between atrial and ventricular systole, which probably plays an important part in the proper filling of the ventricles with blood.

The fibres of the bundle of His arise from the atrioventricular node and spread out to reach the ventricular cells, with which they form junctions. These specialized cells, the Purkinje cells, have a high conduction velocity of 2-4 m/sec because they have high membrane potentials and their action potentials rise very rapidly. Ventricular cells must contract almost together to produce an effective pumping action. The ventricular cells thus depend on the conducting system of fibres for their activation, and all are activated within about 0.02 sec. There would appear to be one-way conduction in the Purkinje fibres, so ventricular events are prevented from reaching the atria; this probably has a protective function. Ventricular cells do most of the work of the heart in pumping. The action potential spreads through the thick muscular wall as a complex wavefront of depolarization in three dimensions, like water soaking into a slab of cotton wool. This has consequences for the form of the electrocardiograph.

Fig. 2

Fig. 3

Fig. 4

FIG. 2—A schematic drawing of the changes in membrane potential occurring inside an atrial or ventricular cell during an action potential, and the changes in sodium and potassium permeabilities which underlie it. The ionic permeabilities are drawn on a logarithmic scale to accommodate the very large increase in sodium permeability at the beginning of the action potential. FIG. 3—A schematic drawing showing the changes in membrane potential occurring inside a pacemaker cell of the sinu-atrial node, and the permeability changes underlying these. The ionic permeabilities are again shown on a logarithmic scale. The slow depolarization of the pacemaker potential to threshold with the consequent production of an action potential is shown. FIG. 4—The factors which influence the time taken for the pacemaker potential to reach threshold, and thus control the heart rate. A, the rate of rise of the pacemaker potential; B, the threshold level; C, the membrane potential of the cell after repolarization.

The Pacemaker

Heart muscles are rather unusual in that their resting membrane potentials are unstable. These cells are subject to spontaneous depolarization. This is because the cell membrane at rest (diastole) is not impermeable to sodium ions as in most other cells. At rest, sodium ions leak into the cells carrying positive charge. Normally potassium ions would move out of the cell to maintain the potassium-dominated equilibrium. Nevertheless, if the sodium leak is greater than the outward movement of potassium can account for, the net effect is one of slow depolarization. All heart cells have this property to some degree but it is particularly well developed in the cells of the sinu-atrial node.

The situation occurring in the cells of the sinu-atrial node is depicted in Fig. 3. These cells have a rather high resting sodium permeability allowing a sodium leak at rather a high rate. After repolarization from an action potential the potassium permeability is high and dominates the situation. However, this declines during diastole and at some point the sodium leak cannot be compensated by the movement of potassium ions; a slow depolarization occurs, which is called the pacemaker potential. When the membrane reaches threshold, the sodium mechanism is activated and an action potential results. This rises rather slowly to its peak and has a poorly defined plateau. The cells of the sinu-atrial node have the highest rate of pacemaker activity so that under normal conditions action potentials from them suppress pacemaker activity elsewhere in the heart.

The heart rate is determined by the time taken for the slow depolarization of the pacemaker potential to reach the threshold for another action potential. Several factors influence this (Fig. 4.) The rate of rise of the pacemaker potential determines the time taken to reach threshold (Fig. 4A.) A potential which rises rather rapidly will result in a high heart rate. The rate of rise of the pacemaker potential depends on the balance between the movement of sodium ions into the cell and that of potassium ions to the outside. If the sodium leak is large, or the potassium permeability is low, the heart rate will be high.

The exact level of membrane potential which is threshold—at which an action potential is initiated—also influences the time interval between beats (Fig. 4B.) A cell in which the threshold was changed from Th2 to Th1 would have a lower rate of beating, as a given pacemaker potential would take a longer time to discharge an action potential. The time taken for a pacemaker potential to reach threshold also depends on the value of membrane potential from which it starts. A cell which repolarizes to a more negative resting potential would take longer to be depolarized to threshold again by a sodium ion leak (Fig. 4C.) Probably all three of these factors act together to determine the rate at which any heart muscle cell will beat spontaneously. It is unlikely that any one cell will remain the pacemaker for long. The pacemaker region will move as circumstances dictate. The arrival of an action potential conducted from elsewhere effectively resets the cell after repolarization, so cells with the highest rate of spontaneous beating suppress pacemaker activity in all the others. Normally the pacemaker resides in the sinu-atrial node, but in pathological conditions it may be elsewhere, or there may be several pacemakers. Such a situation tends to disrupt the pumping action of the heart.

Autonomic Innervation

The heart receives innervation from both divisions of the autonomic nervous system. Parasympathetic nerve fibres reach the heart through branches of the vagus nerves, sympathetic fibres run in the cardiac nerves. The nerve supply, of course, does not drive the heart muscle to contract as in skeletal muscle; it merely modifies the inherent rate of beating. The

effects of the vagus nerve are mediated by the chemical transmitter acetylcholine, those of the sympathetic by noradrenaline.

Parasympathetic nerve fibres innervate only the atrial and nodal muscle; vagal effects on these parts are direct, the ventricles being affected only indirectly. The vagal actions on the heart probably occur because the acetylcholine released from these nerve endings increases the permeability of the cell membrane to potassium ions. At the sinu-atrial node weak activity of the vagus causes a more negative membrane potential to be established, and slows down of the rate of rise of the pacemaker potential—and hence of the rate of beating. This is because the increased potassium permeability allows the movement of potassium ions out of the cells to be more effective in countering the inward leak of sodium. Strong activity of the vagus stops the spontaneous depolarization altogether, and the membrane potential of the pacemaker cells becomes stabilized at a value of about -75 mv—the same as in other atrial cells—instead of the normal -60 mv. So the heart stops beating. In this situation the movement of potassium ions dominates the leak of sodium ions.

The effect of parasympathetic nerves on atrial cells is to reduce the duration of the action potential because repolarization occurs sooner and is more rapid. The shorter action potential leads to a less effective activation of the contractile part of the cell and so a weaker atrial contraction. The earlier repolarization also seems to lead to an increased susceptibility to atrial fibrillation, possibly because there is an earlier resumption of normal excitability after the initiation of an action potential. Activity of the vagus nerves leads to a reduction of the size of the action potentials being conducted along the branching fibres of the atrioventricular node. In addition the membrane potentials of these cells are stabilized. This leads to slower conduction through the node and an increased atrioventricular delay. Eventually a block to conduction will occur. Probably all of these manifestations of parasympathetic activity are due to a single change in the cell membrane—an increase in its permeability to potassium ions.

Nerve fibres from the sympathetic division of the autonomic system innervate all parts of the heart. The effects of sympathetic activity on the cells are less well understood than those of the vagi. The rate of beating of the pacemaker increases, (the chronotropic effect) and the force of contraction increases (the inotropic effect.) There is also an increase in the speed at which action potentials are conducted along muscle fibres. In addition, there is an increased tendency for ventricular arrhythmias and fibrillation to occur. Observations of the changes in the electrical behaviour of the cells have given equivocal results, except in the case of the sinu-atrial nodal cells. Here the rate of rise of the pacemaker potential is increased, so that the threshold for an action potential is reached sooner, and the rate of beating is increased (the chronotropic effect.) It would be convenient to explain these effects by suggesting that the permeability of the membrane to sodium ions was increased by sympathetic activity. Though this would fit with the experimental observations, the evidence in favour cannot be considered to be conclusive.

Artificial Pacemaking

Direct electrical stimulation of heart cells may be employed to produce a contraction of the ventricles if normal conduction has failed owing to some disease. Such stimulation can produce a normal heart rate, above the idioventricular rate, and can improve the clinical state. A "paced" heart is still at a disadvantage compared with a normal, innervated heart because its characteristics are not exploited fully. The abnormal stimulation of the ventricles is almost certainly different from the normal activation in the order of the contraction of the ventricular fibres. In particular, the conducting system of Purkinje fibres is not used to best advantage and the pumping

action of the ventricles may be less effective than normal. In simple pacing procedures ventricular contraction is dissociated from atrial contraction because only the ventricles are stimulated. Filling of the ventricles with blood may not be normal. Restoration of the normal heart sequence, with ventricular contraction occurring with a small delay after atrial contraction, would almost certainly increase the available cardiac output. This can be achieved by triggering off the ventricular stimulus, with a small delay, from the P wave of the E.C.G. This procedure has the additional advantage of allowing the paced ventricular rate of beating to be modified by the activity of the autonomic nervous system because the

beating of the normal sinu-atrial pacemaker is followed. Nevertheless, it may not be feasible to carry this out because the technical difficulties may not be compatible with day-to-day clinical practice.

This article is one of the Birmingham series of lectures (see B.M.J., 27 November, p. 510).

Further Reading

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General Practice Observed

Place of the General Practitioner in the Obstetric Service

J. OLIVER WOODS

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Summary

A survey of perinatal mortality in Northern Ireland has shown that despite a progressive fall in the proportion of deliveries at home the perinatal mortality rate in domiciliary practice has risen in recent years. Overall, however, the perinatal mortality rate for all deliveries compares well with English figures. It seems that as the proportion of deliveries in hospital and general-practitioner obstetric unit rises a hard core of high risk patients is left who insist on delivery at home. Three prerequisites for good general-practice obstetrics are careful selection of cases, intelligent antenatal care, and close co-operation between the general practitioner and the consultant.

Introduction

The role of the family doctor in the maternity services is at present being questioned. Can the family doctor maintain his position as an obstetrician, with the introduction of new techniques and the progressive development of midwifery?

The general-practitioner/obstetrician offers the advantages of continuity of care. This can be provided increasingly in general-practitioner maternity units—which, especially in country districts, are usually situated closer to the patient's home—than the larger district hospital. But does the standard of maternity services provided warrant the retention of the family doctor-obstetrician and has a general-practitioner maternity unit any real advantages over domiciliary confinement? This paper looks at these questions with particular reference to the maternity services in County Armagh, Northern Ireland.

Maternity Units in County Armagh

The main district hospital for County Armagh is the Lurgan and Portadown Hospital. This is a 30-bed specialist unit which during the period under review was staffed by one consultant obstetrician. It provides specialist cover for the Carleton General Practitioner Maternity Hospital, a 30-bed unit situated 6 miles (9.5 km) distant in Portadown. Daisy Hill Hospital is the other specialist hospital situated on the southern border of the country. A new general-practitioner unit has been opened in the City of Armagh but was not operational during the period under review. The number of births in the various units over the 10-year period 1960-9 and the dramatic fall in domiciliary confinements are shown in Table I.

TABLE I—Number of Births in Various Hospitals in County Armagh (1960-9)

	1960	1961	1962	1963	1964	1965	1966	1967	1968	1969
Carleton ..	694	717	781	833	948	957	948	961	972	962
Lurgan ..	448	480	502	576	600	630	729	764	842	965
Domiciliary ..	934	832	760	754	697	596	463	374	274	187
Daisy Hill ..	462	457	494	483	618	589	635	655	577	600

The selection of patients for delivery at home or in the general-practitioner unit is entirely a matter for the individual general-practitioner/obstetrician. The matron of the unit books the patient on referral by her general practitioner and no criteria are laid down for the selection of cases. The consultant obstetrician does not see each patient routinely during the antenatal period but is readily available for advice if requested by the general-practitioner/obstetrician. Antenatal clinics are not held at the maternity unit. The general-practitioner/obstetrician does not usually send his antenatal notes to the unit when the patient is admitted. There has hitherto been no restriction on any general practitioner attending the unit, but recently an obstetric list has been introduced. The general practitioner is normally present at delivery and if not available always

Armagh, Northern Ireland

J. OLIVER WOODS, M.D., M.R.C.G.P., General Practitioner