Age and the Electrocardiogram

M. Irené Ferrer, M.D.

Consultant in Cardiology Metropolitan Life Insurance Company
Professor Emeritus of Clinical Medicine, College of Physicians and Surgeons, Columbia University
Consultant Electrocardiographer, Presbyterian Hospital, Columbia Presbyterian Medical Center, New York, N.Y.

The electrocardiographic tracing most familiar to physicians is the normal adult electrocardiogram. This tracing is remarkably stable once it assumes its mature stage and indeed one can use the electrocardiogram as a clear identifier of any one individual since, in the absence of intervening disease, the tracing remains identical when repeated again and again. So stable is the electrocardiographic pattern for any one individual that it ranks with an individual's fingerprints as proof of identity.

The maturation of the electrocardiogram to its final stable adult status must be understood in light of the rather marked developmental changes that characterize it from early fetal life to adulthood. These will be reviewed. Finally the electrocardiogram in the elderly will also be discussed.

The Fetal Electrocardiogram

The normal pacemaker of the heart, the sino-atrial node, does not control the cardiac rhythm from the onset of cardiac development in the embryo. During the first 6-7 weeks of embryonic life there is no evidence of a discrete structure comparable to the definite mature sino-atrial node. The AV node also is not yet formed at this time. Only the primordia of the His bundle and its branches are differentiated from working myocardial cells as specialized conduction tissue.

Fetal electrocardiograms indicate that definable electrocardiographic complexes first appear between 10 and 12 weeks of embryonic age. These usually are only QRS complexes (i.e., no P waves) suggesting that the cardiac rhythm in early embryonic life originates in junctional tissue, probably in the main AV bundle of His. There are however considerable technical difficulties inherent in obtaining the fetal electrocardiogram. Initially the only methods providing any information were those recording the tracing from the infant's scalp via the mother's vagina but this could not be done until labor begins and the head descends. Fetal electrocardiograms recorded via the mother's abdomen have been unsatisfactory, because the electrical deflections are blunted and damped and usually only the QRS complexes can be defined. Recent studies have used 2-D real time and M mode echocardiography to record atrial and ventricular chamber motion, (i.e., atrial systole followed by ventricular systole). These recordings permit the diagnosis of cardiac rhythms and rates by analysis of the mechanical systoles of the atria and ventricles and this material will be able to provide serial observations of these in embryonic life in future.

At present one can only opine that normal sinus rhythm begins early in the middle trimester of gestation. When the embryo is about 8 weeks old a structure is first visualized which will develop into the mature SA node. By 10 weeks of age a discrete SA node with the tripartite cell population of the adult node is seen. The completed AV node can be recognized anatomically only after 10 weeks in humans and only at about midterm are definitive and mature bundle branches recognizable even though these specialized tissues are distinguishable at 6 weeks. Both the AV node and the bundles are of course needed to convey sinus rhythm to the ventricles. In summary then, by approximately 10 to 16 weeks of embryonic life the pacemaker and the AV conduction system function (sequentially) as they do in the adult heart.

Neonatal, Infant and Childhood Electrocardiograms

The age of the child is very important in evaluating the ECG tracing. Sinus rate in the fetus and the newborn is usually rapid and relatively unstable, probably due to unbalanced autonomic innervation. Early neural control is predominantly cholinergic. Adrenergic innervation appears later and is complete only some months after birth. Immediately after birth heart rate generally is between 135 and 140 per minute but may vary between 75 and 200 per minute. Sinus rates gradually decrease as the child ages, falling on the average to 85-125 per minute at age 2 years, 75-115 per minute at age 4 years, 65-105 per minute at age 6 years, and 60-100 per minute at age 8 years.

The form of the P wave changes continuously during early neonatal life. At birth it is tall, peaked and narrow in duration (0.03 to 0.08 sec.). Between one month and one year it becomes slightly wider in duration (0.04-0.08 sec.) and between one and three years is 0.06-0.09 sec. After this it widens further, reaching adult P duration (0.08-0.11 sec.) between 5 and 10 years of age. The height of P also decreases during early life.

The PR interval may be quite short at birth (0.08-0.10 sec.) and later lengths to 0.12 to 0.14 sec. by one to three years and to 0.12 to 0.17 sec. by five years or later. The PR interval varies somewhat with heart rate and this relationship appears in Table II of reference 7.

The duration of the QRS complex measures 0.08 to 0.09 sec. at birth and by the end of the second or third day of life decreases to 0.05 to 0.06 sec.

The voltage of QRS complexes overall is larger in full term children as compared to adults. In contrast, in premature infants the voltage of the QRS complexes may be quite low and does not reach the larger normal size until the first postnatal month.
The electrical axis of the QRS complex changes constantly in early life. There is right axis deviation (+90° to +110°) at birth. In the first half year of infancy the axis shifts leftward in the frontal plane and the adult axis orientation (+30° to +90°) is usually complete by age 6 to 8 months. It is thus clear that any degree of left axis deviation of the QRS (+31° to as far as –90°) never occurs in early life or indeed in teenagers. Positional left axis deviation (+30° to –44°) is not seen until after 16 years of age (see Fig. 5b, ref. 7). Left axis deviation of –45° to –90° of course, is due to an IV conduction defect—left anterior fascicular block.

The contour of the precordial V leads also is changing serially in early life. Predominance of the right ventricular mass over the left at birth is expressed as large R waves seen at birth in the right V leads. These large R waves decrease in size beginning around 6 months or one year of age and the decrease continues gradually over the first 4 to 6 years of life. Similarly, there are serial changes in T waves with growth. In the first 24 hours of life the T waves may be almost flat in contour in all 12 leads and then become larger in a few days. The T wave in the right V leads may by upright in the first 24 hours of life but after the first few days, or occasionally even as late as 3 weeks after birth, the T waves in the right V leads (V1-V3) normally become quite negative and remain so up to approximately one year of age. The T waves are positive in the left precordial leads (V4-V6). Although it is usual for the negative T waves in V1-V3 to become upright in the early years of life this change may not take place until the late teens or early twenties. This persistence of negative right sided T waves into early adulthood is called “the juvenile pattern” and implies no cardiac abnormality.

The Adult Electrocardiogram

The influence of age upon the adult electrocardiogram, once the tracing has developed from the childhood patterns and has stabilized, is really negligible. Recognizing as normal: (1) the possible persistence of the juvenile pattern of negative T waves in the right V leads, and (2) the frequency of elevations of the ST segment due to the early repolarization variant which is commoner in young persons but is sometimes seen in the third and fourth decades—the adult tracing can be described in fairly stable and consistent terms. The values for normal adults have been tabulated by the Criteria Committee of the N.Y. Heart Association. These normal values, and in addition the major abnormal patterns in adults, are reviewed recently by Fisch.

Although the age of the adult is not an influencing factor in reading electrocardiograms once the possibility of the juvenile pattern and early repolarization are acknowledged, knowledge of the body habitus of the adult may be a necessary piece of information in the interpretation of the tracing. Tall slender bodies and squat or fat bodies influence axis deviation. Persistence of right axis deviation beyond young age is seen in normal thin persons and left axis deviation often occurs in fat persons. Changes in axis orientation occur with changes in bodily position of the patient. Thus lying down is the routine posture used in recording tracings but if the individual is seated in an office lab or semi seated as in a propped up or gatched-up position in a hospital bed, changes in axis will occur and are medically meaningless.

Another normal variant that has not received enough medical emphasis is the effect of low potassium consequent to the taking of food shortly before the recording of an electrocardiogram. These potassium flux changes result in low T waves sometimes but not always accompanied by prominent U waves in most or all of the 12 ECG leads. Hence information on the possible effects of food intake near the time of the test is important.

The Electrocardiogram in the Aged

The average length of life in the United States has increased from 47 to 73 years but the maximal life span has not increased. For example, the number of persons reaching age 100 years or more has not changed in a 180 year period according to statistics in England. Nevertheless the number of persons surviving into the seventh, eighth, and ninth decades is growing. It is therefore very important to note that clear identification of changes in the cardiovascular system due to aging alone is difficult if not impossible and, although there may be certain changes observed at necropsy in the elderly, these produce no clinical dysfunction and are often listed as expected for advanced age or even considered normal. These changes are reported in pathological terms for the most part and do not generate cardiac or other dysfunction. They include alterations in the myocardium (brown atrophy, basophilic degeneration found in the sarcoplasm, focal amyloid deposits, increased subepicardial fat); possibly some slight reduction in the size of the left ventricular cavity and dilatation of the left atrium; fibrous thickening, occasionally with calcification, of the mitral and aortic valves; calcification of the mitral anulus; tortuosity and some mild dilatation (increased cross sectional area of the lumen) with some calcium deposits in the epicardial coronary arteries; dilatation and some vessel wall calcification of the aorta. None of these normal variations in the aged have any effect upon the electrocardiogram. Indeed the normal values listed in various sources as normal—particularly the values which are used in the programming of computer analysis of the electrocardiogram—citate only a lower level for adults—i.e., over a certain age (such as 25-30 years old) but have no ceiling, i.e., no separate values for persons older than that. In other words statistics for older persons are no different than the younger ones after a certain age is reached.

It is therefore quite clear that abnormalities noted in the electrocardiograms of the elderly cannot and should not be explained away as due to advanced age. The etiology of such abnormalities should be sought in the same way as in persons who are younger.

One particular error that is frequently made in the elderly is attributing bundle branch block (either right or left) to coronary disease just because the subject is aged. Other disorders can and do produce much conduction abnormalities and hence IV conduction defects are not bona fide markers for coronary artery disease.
With increasing detailed knowledge of disease as defined by current technologies, it is evident that the vital organs may function well into the ninth and tenth decades if spared from disease. The age of the organ system does not imply deterioration.

In summary, age is a crucial factor in reading electrocardiograms in neonates, infants and children, as well as young adults into their twenties. After that, the adult and the aged share the same normal values.

References


