The sequence of the electrical events which reflect cardiac excitation and comprise the background for recording the wave forms which together are called the electrocardiogram, has been evaluated in considerable depth in recent years. The greatest attention has been directed at the QRS complex and T waves, which reflect the ventricular event - and indeed much of the clinical information derived from the ECG springs from interpretation of these abnormalities of QRS-T. Nevertheless much valuable data can be obtained from careful evaluation of the P wave or atrial event. This part of electrocardiographic analysis has however been somewhat neglected except in the case of rhythm analysis.

SEQUENCE OF ELECTRICAL EVENTS IN THE ATRIA

A brief review of the atrial events occurring in sinus rhythm emphasizes the possible areas which will yield useful information. When the SA node sends out the excitation wave it generates, conduction of these impulses from the SA node throughout the atria, and between the SA node and AV node - contrary to the older theories of non-preferential diffuse radial spread of excitation over all the atrial myocardium - takes place via three specialized atrial tracts. These tracts, named anterior, middle and posterior, are composed of both Purkinje-like fibers and ordinary myocardial fibers and all three start from the SA node itself and end in the AV node. The anterior internodal tract, when it leaves the SA node, sweeps in front of the superior vena cava, divides in two and sends one branch directly over the roof of the right atrium to the left atrium. The second branch goes down the interatrial septum to enter the crest of the AV node. The anterior internodal tract leaves the posterior margin of the SA node, follows the crista terminalis to and through the Eustachian ridge until it reaches the inferior margin of the AV node. Most of these posterior tract fibers bypass the AV nodal crest which is where the physiologic delay or pause in the excitation process between the SA node and common bundle of His occurs. This AV nodal delay occupies about 0.04 second in time. Lateral extensions of the posterior internodal tract arborize over the dorsum of the right atrium and deliver the excitation signal to this atrial chamber.

Thus, special branches of the anterior and posterior internodal tracts carry the electrical activation directly and rapidly to both atria and the atrial chambers do not wait for this excitation to spread radially over the slower conducting myocardial avenues after leaving the SA nodal area. Once the electrical stimulation reaches both atrial chambers the excitation waves move through the atrial myocardium of the two cavities. The spread of excitation waves throughout the atrial myocardium on both sides is represented by the P wave of the clinical electrocardiogram. The electrocardiogram contains no visible electrical evidence of the firing of the pacemaker, the SA node itself, and the P wave does not begin to form until excitation waves have actually invaded the atrial myocardium. Recently, however, using very special electrical amplification and recording techniques and acquiring the electrical signals very close to the specialized tissues of the SA node by means of a cardiac catheter, it has been possible to identify the action potential spike of SA nodal activity. It is reasonable to expect that with more sophisticated circuitry it may be possible in the future to amplify the clinical electrocardiogram in such a way as to identify on this tracing the SA node spike and the His bundle spike as well. This latter, announcing the arrival of excitation in the area of the His bundle just beneath the AV node, is a valuable item for rhythm and conduction analysis. Healthy or diseased SA node function could be better analyzed if the pacemaker, or generator, spike plus the atrial
spread or P wave, were both available for analysis. First degree exit blocks from the SA node - delays between the firing of the SA node and the reception of excitation at the atrial myocardial level - could be diagnosed early in this way. At present only the more advanced stages of the SA exit block can be tagged. Failure of the generation of sinus rhythm or weakening of the generator function could also be assessed.

Following atrial excitation the mechanical event - atrial systole - occurs. The interval between the beginning of the P wave and the onset of atrial systole is 0.06 second for the left atrium and 0.07 second for the right atrium.

CHARACTERISTICS OF THE NORMAL P WAVES

In sinus rhythm the P wave is always upright in lead I and II and always negative in AVR. It may be upright, diphasic or negative however in lead III. As explained in Figure 1, leads II and AVR are best suited for recording the P wave. Lead V4 being over the right atrium, is also often helpful in P analysis. Indeed, in this lead the second, or later, half of the P wave has been accepted by some as the only electrocardiographic evidence of excitation of the left atrium as opposed to excitation of the right atrium as reflected in the first half of the P wave.

The P wave characteristics in children deserve special comment. It is interesting to note, since it is now possible to record the electrocardiogram of the unborn fetus, even identifying twins and triplets, that definable ECG complexes, albeit usually just the QRS complex, first appear in the fetal electrocardiogram between ten and twelve weeks of age. The P wave appears later, perhaps because the initial cardiac rhythm is not from the SA node but from a lower pacemaker such as the His bundle, or because the size of the P wave is too small for recording by the present methods. The P wave in newborns is narrow. Its duration varies between 0.03 and 0.09 second between birth and age three years. The P wave slowly becomes wider and between three and sixteen years measures between 0.05 and 0.11 second, and after age sixteen the normal range of P wave duration, 0.08 to 0.11 second is seen. The developmental variation in P width is probably related to the slowly increasing atrial mass as the heart grows. The PR interval is quite short at birth and during the first week of life (0.08 - 0.10 second) and then gets longer. One can see intervals of 0.08 second occasionally up to age three years but most are 0.10 - 0.15 second as the child grows, and by age sixteen years the adult PR interval is seen (0.12 - 0.20).

In adults the form of the P wave varies very little even in sinus tachycardia. It is not abnormal to find some notching on either the ascending or descending limb of the P wave.

INFORMATION OBTAINED FROM ABNORMAL P WAVES

The first two types of abnormal P waves occur in sinus rhythm. In atrial arrhythmias the form and vector orientation of the P wave is, of course, very different from the basic normal sinus rhythm and will be discussed subsequently.

1. Abnormal Width of P Waves: Whenever the duration of P in sinus rhythm is longer than normal (0.12 sec. or more) there is intra-atrial block present. This conduction defect of the atria can be seen on every sinus beat or, early in the disease processes generating the fibrotic, inflammatory or infiltrative pathology, it can be seen in only some P waves. This intermittent intra-atrial block later goes on to wide P waves in every sinus beat. It is not yet known if intra-atrial block occurs because of damage to the atrial conduction tracts or due to diseased atrial myocardium. Prolongation of the PR interval may take place solely due to a wide P wave, placing the defect as an intra-atrial block. If it is due to AV block alone, the PR segment, not the P, will be long.
Figure 2: Orientation of the P vector in normal sinus rhythm (N.S.R.) and slow atrial rhythm (S.A.R.) as shown on the frontal plane electrical axis diagram. The segment enclosing all P vectors in NSR lies in the positive sector in 100 cases (+90° to just beyond zero degrees) while the segment enclosing all P vectors in 39 cases of S.A.R. is quite negative (-30° to -107°). As far as P orientation is concerned there is a clear separation of the two rhythms.

Figure 3: An example of slow atrial rhythm, showing a rate of 75 # min. and negative P waves in II, III, AVF and positive P waves in AVR. The P vector is -79° (The twelve lead ECG is recorded with four sets of three simultaneous leads: I, II, III top to bottom on left, AVR, AVL, AVF next set to the right, V1-V6 in the third column and V4-V6 in the last column on the right.)
2. Abnormally Large P Waves: P waves in excess of 2.5 mm in amplitude during sinus rhythm have been attributed either to atrial hypertrophy or enlargement due to dilatation. The association between enlarged atria and tall P waves however, is by no means a constant one. The P is not always large when one, or even both, atrial chambers are enlarged and conversely, a tall P wave does not always indicate an enlarged atrial cavity. The second half of the P wave as recorded in V_{1}, if both wide (0.04 second or more) and deep (negative by 1.0 mm or deeper), suggests left atrial enlargement in a more reliable way but still is not completely satisfactory.

It is important to note that tall P waves in II, III, AVF (so-called P pulmonale) are seen in pulmonary emphysema but do not necessarily imply an enlarged right atrium. Rather they reflect the pulmonary disease since the vertical position of the heart, due to the abnormally low diaphragm, produces rotation of the right atrium bringing it nearer to the anterior chest wall. This occasions increased voltage of the P wave on a proximity basis only. Thinning of the chest wall with loss of body fat will do the same.

3. Change in Shape of P Wave: It has long recognized that changes in shape of P waves are seen in supraventricular arrhythmias. Notably, junctional rhythm (formerly called AV nodal rhythm) produces abnormally narrow P waves and an abnormal P vector (P is negative in II, III, AVF and positive in AVR.) Atrial tachycardias have a different shaped P from sinus rhythm as well as a different PR interval.

An arrhythmia recently recognized as not uncommon may be missed if the change in P shape is overlooked. This rhythm, called slow atrial rhythm, fires relatively slowly, at only 50 to 80 per minute, so that the rate alone does not alert the interpreter of the ECG to the presence of non-sinus rhythm. This slow atrial rhythm (S.A.R.) is now interpreted as a probable sign of SA node dysfunction since it arises only when sinus rhythm stops. As a rescue rhythm following SA node silence or sluggishness, this atrial escape focus takes over to support cardiac rhythm. Another rescue rhythm seen in the sick sinus syndrome arises in the AV junctional tissue and also fires at modest rates (45-55/min.) The orientation of the P vector - or frontal plane electrical axis of P - in S.A.R. is quite different from that of normal sinus rhythm, as can be seen in Figure 2. The negative P waves in II, III, AVF and positive P in AVR are seen in Figure 3. This negative position of the P vector in S.A.R. suggests that atrial excitation proceeds from the bottom to the top of the right atrium, arising as a low atrial rhythm, rather than moving from the upper right lateral wall or roof of the right atrium downward as occurs in sinus rhythm. Recent electrophysiologic studies however have shown that this "south-north" pathway in S.A.R. is a misconception.

Since there are specialized conduction bundles in the atria the exact site of origin of the S.A.R. is now almost always low in the right chamber and certainly is not always near the coronary sinus as was previously thought. Hence the term Coronary Sinus Rhythm has been dropped. S.A.R. probably can arise from a number of atrial sites and, depending on the pathway of atrial excitation and which, if any, atrial specialized conduction tracts are used to deliver the stimulus to atrial myocardium, the P wave shape and frontal plane axis will vary.

Atrial arrhythmias other than those mentioned so far are well known as far as P shapes are concerned. For example, the low, often barely visible, rapid oscillations seen in atrial fibrillation and the regular rapid biphasic flutter waves need no comments. It is worth noting that examination of the P waves (in sinus rhythm) in subjects with acute myocardial infarction may, on a rare occasion, show a somewhat esoteric finding, namely evidence of acute atrial infarction. Only about 80 percent of patients with ventricular myocardial infarction show the classical ST-T and Q wave abnormalities on the electrocardiogram. Only 12 - 15 percent of all patients with acute ventricular myocardial infarction also sustain acute atrial myocardial infarction. Atrial infarction thus is rare. If the infarcted area of atrial muscle is small no ECG evidence of it will appear. If the atrial necrosis is large there will be an elevated PR segment, analogous to the ST elevations in ventricular infarction. The P wave is the excitation wave of the atria as the QRS is the excitation wave of the ventricles. Currents of injury due to necrosis follow the excitation complexes - elevated PR segment after P wave and elevated ST segment after the QRS. It is a very rare phenomenon however to find the elevated PR segment on the ECG of acute infarctions.

Another instance where attention to P waves is valuable is in the discovery of the patterns of preexcitation. The form of the P wave, of course, is not abnormal in preexcitation but because of the short PR interval in both the L - G - L and W - P - W forms of the disorder, P waves appear very close to the QRS and this visual fact alerts the reader to the presence of preexcitation.

In Summary:

Careful attention to analysis of the P wave in electrocardiography can supply interesting, and crucial, information. In particular, intra-atrial block (wide P waves), atrial enlargements (large P waves) and arrhythmias (change in P shape and P direction) can be diagnosed and have their impact upon insurance ratings.