It has been observed, while studying routine electrocardiograms of air-force personnel, that although the QRS complexes of individuals remain mainly constant in the various leads, the P wave configuration varies considerably from one year to another. It is chiefly the P waves in standard lead III that vary from upright to biphasic to inverted. Consequently there is also considerable variation in the electrical axis of the P wave in the frontal plane, as well as accompanying amplitudinal variations of the P wave in standard lead II. It has moreover been observed that, within physiological limits, a relatively close relationship exists between the electrical axis of the P wave in the frontal plane and the heart rate; an increase in the heart rate is accompanied by a shift of the P axis to the right. It is the sole purpose of this study to account, by means of indirect evidence, for this axial shift on an increase in the heart rate.

**Survey of Literature**

It is generally accepted that the atrial depolarization wave originates in the S-A (sino-atrial) node, from whence it spreads radially to innervate the atria. It is further known that the S-A node is situated in the sulcus terminalis, just below the bifurcation formed where the upper surface of the right auricle meets the superior vena cava. As far as the origin of the impulse is concerned, the above-mentioned hypotheses are the only ones on which there is agreement. Even the anatomical size of the S-A node ranges, according to description, from 2-3 cm. in length and up to 2 mm. in width, to 5.5-7 mm. in length and 3 mm. in width.

Various workers found evidence that the excitation wave originates from within or near the head of the S-A node. Conversely, it has been determined through high-speed cinematographic studies that the contraction wave starts in the middle of the taenia terminalis, approximately 1 cm. to the right of the junction of the superior vena cava and the right auricle. This area would coincide with the central or caudal portion of the S-A node. In experiments during which thermal stimuli at a temperature in excess of, but very near to, body temperature are applied to the right atrium, an increase in the impulse discharge is found only when the area of the S-A node is stimulated. In this respect it is significant that although the whole of the S-A node is sensitive, the increase in the rate diminishes with the distance the point stimulated is removed from the head of the node.

Although the S-A node inherently possesses the quality of spontaneous rhythmical impulse discharge, in the intact animal it is mainly dependent on the autonomic nervous system. The negative and positive chronotropic effects are respectively dependent on vagal and sympathetic stimulation. Vagal stimulation causes a decreased period of depolarization, as well as hyperpolarization of up to as much as 33 mV more than the normal 50 mV in the pacemaker area of the heart of a frog. The negative chronotropic effect of vagal stimulation is exclusively ascribed to a suppression of pacemaker potential, while positive chronotropic effect is due to the accelerated rise in the action potential. It seems to be of special importance that when acetylcholine and epinephrine are administered locally, or when vagal or sympathetic stimulation is applied, a shift of the pacemaker area takes place.

**Material and Methods**

All electrocardiograms used for analysis were those of (as far as could be ascertained) healthy flying personnel between the ages of 17 and 55 years. The frontal electrical shift of the P wave axis during exercise was studied in 400 individuals. For this purpose 4 groups of 100 consecutive individuals each, within age-groups 16-25, 26-35, 36-45 and 46-55 were taken.

Exercises on a bicycle ergometer were carried out in a prone position, with all the electrodes in place. A heart rate increase of approximately 50% was effectuated in all cases. An 8-channel 'Heilige Multiscriptor' electrocardiograph, with a paper speed of 25 mm./sec. was used for recording the graphs. The P axis was determined according to the usual tri-axial reference system.

Oesophageal leads were used on 25 individuals (aged 16-25) with the aid of a polyethylene stomach tube, at the end of which six annular silver electrodes with a thickness of 1 mm. each were arranged at intervals of 5 mm., thus making it possible to take 6 unipolar leads simultaneously. The position of the oesophageal electrodes was determined by the complex configuration, and they were manipulated into the desired position. For instance, when the electrical axis of the P wave is ± 0°, the electrodes can be manipulated in such a way that the two lower electrodes show a mainly negative deflection; the two middle electrodes show a biphasic, and the two top ones a mainly positive deflection.

The A-V (atrio-ventricular) conduction periods were studied in various individuals, 254 showing sinus bradycardia and 58 showing sinus tachycardia when examined electrocardiographically while at rest. Sinus bradycardia was diagnosed when the heart rate was below 60 per minute, and sinus tachycardia when it exceeded 100 per minute. Since the initial deflections of the P wave, as well as the QRS complex, were iso-electrical or very near to the iso-electrical line in some leads, a more accurate measurement of the P-R (P-Q) intervals was obtained by studying different leads recorded simultaneously.

In addition, the heart rates of individuals displaying short A-V conduction times (0.07 sec. and less), and those displaying long periods (0.20 sec. and more), were determined. Apart from the specific manoeuvres referred to, various electrocardiograms of individuals subjected to other routine manoeuvres (e.g. the intravenous administration of 1·2 mg. of atropine and amyl nitrite inhalation) were also used for analysis.

**Results**

**Shift of P Wave to the Right During Exercise**

Physical exertion is accompanied by a shift of the frontal electrical axis of the P wave to the right (Fig. 1). The degree of this shift has been studied in 400 individuals, divided into 4 age-groups of 100 each. Table I reflects the mean P axis in the frontal plane before exercise, as well as the mean shift to the right immediately after exercise. Considerable individual variation is present, and the degree of shift ranges from a barely perceptible difference to as much as 100° (Fig. 2). In the case of higher ranges of shift, iso-electrical or negative P waves in standard lead III become positive deflections in this lead after exercise.
Other Manoeuvres Resulting in a P Wave Shift

The P wave shift is not observed during exercise only, but also during any condition causing an increase in the heart rate, including (a) the rapid phase of respiratory arrhythmia (Fig. 3), (b) tachycardia following on hyperventilation (Fig. 4), (c) administration of atropine or amyl nitrite and (d) inactivity during which a change in the heart rate takes place from time to time (Fig. 5).

![Fig. 1. Tracing taken (A) before and (B) immediately after exercise. The frontal P axis shifts to the right with an increase in heart rate as a result of exercise.](image1)

![Fig. 2. Histogram showing the degree of shift to the right of the P axis in the frontal plane with exercise of all 400 individuals. No marked difference could be demonstrated between the individual age-groups. It must be emphasized that the degree of shift is an approximation only, due to the low amplitudes of the P waves which make measurement inaccurate.](image2)

![Fig. 3. The tracing was taken during the slow and rapid phases of respiratory arrhythmia. Concomitant with the increase in heart rate a rightward shift of the frontal electrical axis of the P waves results. Note the decrease in atrio-ventricular conduction time during the rapid phase of the arrhythmia. The P-R intervals are indicated in hundredths of a second.](image3)

![Fig. 4. ECG taken (A) before and (B) during a period of sustained expiration after hyperventilation. Note the rightward shift of the P axis manifested during the resultant tachycardia.](image4)

<table>
<thead>
<tr>
<th>Age Group</th>
<th>P Wave Axis Shift</th>
<th>Number of Individuals</th>
</tr>
</thead>
<tbody>
<tr>
<td>16-25</td>
<td>54°</td>
<td></td>
</tr>
<tr>
<td>26-35</td>
<td>54°</td>
<td></td>
</tr>
<tr>
<td>36-45</td>
<td>50°</td>
<td></td>
</tr>
<tr>
<td>46-55</td>
<td>49°</td>
<td></td>
</tr>
</tbody>
</table>
P Wave Configurations in Simultaneous Unipolar Oesophageal Leads with Variations in the Heart Rate

Fig. 6 shows the slow and the rapid phases of respiratory arrhythmia. Simultaneously with the shift of the P axis in the frontal plane as observed in standard leads I and III, unipolar oesophageal electrodes spaced at 0.5 cm. indicate considerable changes in the configuration of the P wave, while the QRS complexes mainly remain constant. Before and immediately after exercise, and with oesophageal electrodes in a fixed position, similar changes are observed, viz. an inversion of the direction of polarity without marked changes in the complex amplitude.

Fig. 5. Electrocardiograms of the same individual taken in consecutive years. With a slight change in heart rate a shift in the frontal P axis is manifested.

The Relationship Between Heart Rate and the P-R Interval

Although this aspect may at a first glance appear to be out of context in the present discussion of the area of impulse origin, there is a popular assumption requiring further analysis. It is accepted that an increase in vagal tone normally has a negative dromotropic effect on the atrioventricular junctional tissue or, expressed differently, sinus bradycardia should be synonymous with a relatively long P-R interval. When the results in Tables II and III are studied, this assumption is disproved. If the normal mean P-R interval is taken as lasting 0.16 second, it becomes apparent that individuals with a sinus bradycardia, as well as individuals with a sinus tachycardia, more often (in 56% and 55% of the cases, respectively) show a relatively short A-V conduction period while they are at rest.

The Relationship Between the P-R Interval and Heart Rate

When this subject is approached from a different angle,
i.e., when the mean heart rate of individuals showing a relatively short P-R interval (0·12 sec. or less) and those showing a long P-R interval (0·20 sec. or more) is determined, it is found that no noticeable difference exists (Table IV).

### Table IV. Mean P-R Intervals and Heart Rates of Individuals Whose Routine ECG Showed Sinus Bradycardia and Sinus Tachycardia

<table>
<thead>
<tr>
<th>No. of individuals</th>
<th>Mean age in years</th>
<th>Mean P-R interval in seconds</th>
<th>Mean heart rate per minute</th>
</tr>
</thead>
<tbody>
<tr>
<td>Individuals with P-R interval = or &lt; 0·12 seconds</td>
<td>...</td>
<td>0·1·131</td>
<td>67·56</td>
</tr>
<tr>
<td>Individuals with P-R interval = or &gt; 0·20 seconds</td>
<td>...</td>
<td>0·2·146</td>
<td>67·65</td>
</tr>
</tbody>
</table>

### DISCUSSION

The increase in P wave amplitude in, *inter alia*, standard leads II and III during and after physical exertion, is commonly ascribed to sympathetic effects. When this is accompanied by a decrease in the amplitude of the P wave in standard lead I, this decrease is due to the common P vector shifting to the right because of the relatively low position of the diaphragm.²

Since this axial shift occurs in all cases of increased heart rate of S-A origin—e.g., after administration of atropine, inhalation of amyl nitrite, or when tachycardia occurs after hyperventilation, etc.—it seems improbable that the part taken in this by the diaphragm is at all important, if it does take any part at all. The exact role of the autonomic nervous system in the amplitude increase in the P wave in standard leads II and III cannot be determined easily, since, in the case of vagal stimulation, no,²⁰ or a very limited decrease, and in the case of sympathetic stimulation, a very small increase of action potential was found.¹⁹

The increase in the P wave amplitude has up to now been stressed in standard leads II and III. In this study, a decrease has been found in respect of the amplitude of the P wave in standard lead I, in conjunction with an increase in heart rate. Very seldom, no clearly perceptible difference or even an increase is found. It is difficult however to assess this, because the P wave in this lead is normally of low amplitude. On the one hand an amplitude increase is possible where the P axis changes from negative to positive, without the positive figure exceeding the negative, e.g., a change from -30° to +20°, and on the other hand, the increase may be due to excessive sympathetic influences. The latter influences are represented here because there is usually an increase in the amplitude of the P wave in standard lead I when the rate of a heart, already beating fast, is further increased, which increase is consequently accompanied by little movement of the P axis.

Because the facts mentioned above do not fully explain the shift of the P axis during heart rate increase, the possibility of a shift of the atrial impulse origin was considered. It was argued that if such a shift did take place, it had to be towards a relatively higher level in the atrium in order to explain the P axis shifting to the right, as well as the amplitude increase of the P wave in standard leads II and III, and the usual amplitude decrease in standard lead I.

This possibility was analysed by means of 6 unipolar oesophageal leads before and after heart rate increases, with the leads in constant position. With the electrodes in a constant position relative to area of impulse origin, a change in the P axis direction is, to a greater or lesser extent, probably due to a shift of the area of impulse origin. The direction of shift can be determined by means of the P wave configuration. Fig. 7 shows the proposed mechanism according to the oesophageal leads.

When the pacemaker area moves towards a relatively higher focus in the atrium, and in this case, in the S-A node, the distance between the area of impulse origin and the A-V node increases and this causes the depolarization wave to take more time to cover the distance. Unfortunately it is not known what influence the autonomic nervous system has on the atrial conduction speed or on the A-V junctional tissue in individual cases. Although vagal stimulation decreases the excitability of the A-V junctional tissues, with a consequent delay in A-V conduction,²¹ it is surprising to find a relatively short P-R interval in 56% of those individuals with a sinus bradycardia. Moreover, when it is taken into consideration that the speed of impulse conduction in the atrial muscle is approximately 1,000 mm./sec.,³ it means that a distance of 1 cm. is represented by as
short a period as 0.01 second. From this it follows that the pacemaker area can shift by as much as 1 cm., while practically no difference is observed on the electrocardiogram. This can serve to explain why a relatively short P-R interval may occur in the presence of sinus bradycardia, in spite of the negative chronotropic influence on the A-V node during an increased vagal tone.

Another aspect that calls for an explanation is the reason why, in dependable direct experiments, the pacemaker area has always been located in the head of the sinus node. A possible explanation that may be advanced is that all these experiments were carried out on animals subjected to mutilating operations. A normal physiological response of these mutilations is a tachycardia, with a consequent upwards shift of the pacemaker area—possibly to the area of the nodal head.

PRACTICAL APPLICATION

1. The electrical axis of the P wave in the frontal plane may be changed by alteration in heart rate and caution used in its interpretation.

2. 'Atrial or coronary sinus rhythm', diagnosed on grounds of P waves (negative in standard leads II, III and aVF and positive in aVR), is possibly sino-atrial origin when it occurs in the presence of relative bradycardia (Fig. 8).

3. 'Wandering pacemaker' diagnosed on grounds of variations in P wave configuration and fluctuations in P-R intervals, is encountered in healthy young persons. In these persons it is often based on a sudden heart rate deceleration after exercise or after any manoeuvre causing a transient tachycardia.

4. The electrocardiographic criteria for enlargement of the right atrium are usually based on the amplitude of the P wave in standard lead II, and the heart rate should be taken into account during its diagnosis.

SUMMARY

P wave variations in the different leads, with the consequent shift in the axis of the P wave to the right during heart rate increase, were studied. During exercise the shift of the P vector to the right ranges from a barely perceptible difference to as much as 100°. This shift does not occur during exercise only, but during any heart rate increase, and the degree of shift is closely related to the degree of heart rate increase.

When this shift of the P axis is studied by means of multiple unipolar oesophageal electrodes, it is apparent that the area of impulse origin has to change if the inversion of the direction of the depolarization wave in respect of the constant oesophageal leads is to be explained. It is also apparent that the area of impulse origin shifts to a relatively higher focus, in this case in the sino-atrial node on an increase in the heart rate.

Added to this, it has been found that in 56% of those individuals showing sinus bradycardia, the P-R interval is also relatively short and this is probably due to a shorter distance between the area of impulse origin and the atrio-ventricular node. Although sympathetic influences, as far as the increase in amplitude in standard leads II and III is concerned, may well have a role in the axial shift to the right during an increase in heart rate, this is probably a subsidiary role.

It is therefore suggested that, during conditions of rest, the area of impulse origin arises relatively distally in the sino-atrial node. The more distally it rises, the slower the heart rate and as the area of impulse origin moves proximally in the sino-atrial node, the higher the heart rate.

From this it would appear that although the negative and positive chronotropic influences are, respectively, dependent on vagal and sympathetic stimulation, the current heart rate is determined by the level in the sino-atrial node where this influence is exercised.

I should like to express my appreciation to the Surgeon General, Combt. Gen. E. C. Raymond, S.M. for permission to publish this article. I am indebted to Fd./Cts. R. A. Lampaecht and P. van Zyl, SAMNS; and to W.O. II P. Beek, SAMC, without whose assistance this investigation could not have been carried out.

REFERENCES


![Fig. 8](image-url) An initial 'atrial rhythm' diagnosed on grounds of negative P waves in standard leads II, III and aVF and positive in aVR. With an increase in heart rate there is a gradual change to a sinus rhythm.