THE EFFECT OF AMYL NITRITE ON THE ELECTROCARDIOGRAM OF NORMAL SUBJECTS

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The increasing use of amyl nitrite as a diagnostic aid in the investigation of cardiac murmurs has aroused interest in the pharmacological properties of this drug during the past few years. No literature on the ECG changes during, or immediately after, the administration of amyl nitrite could be traced. This is probably due to the fact that the precordial area is reserved for auscultation and phonocardiography during amyl nitrite administration and the resultant ECG changes are almost exclusively limited to the precordial leads.

METHODS

Amyl nitrite was administered to 100 consecutive pilots ranging in age from 17 to 25 years, after routine annual medical examinations had been carried out. As far as could be ascertained, the cardiovascular systems of all these subjects were normal. Following explanation of the procedure, the subjects inhaled amyl nitrite for a period of 20 - 30 seconds. During and immediately after the administration, recordings of standard leads I, II and III and precordial leads V2 - V6 were simultaneously made on an 8-channel electrocardiograph. Repeat examinations were carried out on 22 subjects after a period of one week.

In addition to the recordings after inhalation of amyl nitrite, similar recordings were carried out on 10 subjects after forced hyperventilation sustained for 10 seconds. All tracings were carefully scrutinized as regards rate, electrical axis of the QRS complex and T waves and T and U wave changes. The electrical axis was determined according to the usual tri-axial reference system.

RESULTS

With amyl nitrite administration, the following ECG changes were observed: increased cardiac rate of 10 - 116% with an average increase of 49% ; shift of the electrical axis of the P wave to the right associated with the increased heart rate ; a single atrial and ventricular extrasystole occurred respectively in 2 cases ; increased ventricular gradient occurred in 70 cases, decrease in 18 cases and no change in 12 cases (Fig. 1).

T and U wave changes could be classified in 4 groups:

I. General flattening of the precordial T waves in 62 cases (Fig. 2).

II. General flattening of the precordial T waves with prominent U waves in 19 cases (Fig. 3).

III. Inverted T waves in the precordial leads in 15 cases (Fig. 4).

IV. Combination of changes in I, II and III in 4 cases (Fig. 5).

There was no difference in the average increase of heart rate in the above 4 groups. The procedure was repeated on 22 subjects, representative of all 4 groups, without any change in the basic pattern. In order to exclude hyperventilation as a cause of T wave changes,[1] 10 subjects of groups III and IV were subjected to this manoeuvre without any significant T wave changes.

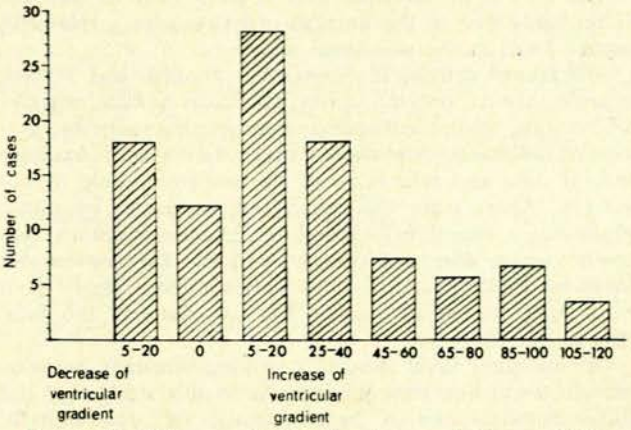

Fig. 1. The histogram depicts the degree of change of the ventricular gradient during and immediately after inhalation of amyl nitrite. In most cases the resultant change was brought about by inversion of the T wave in standard lead III.

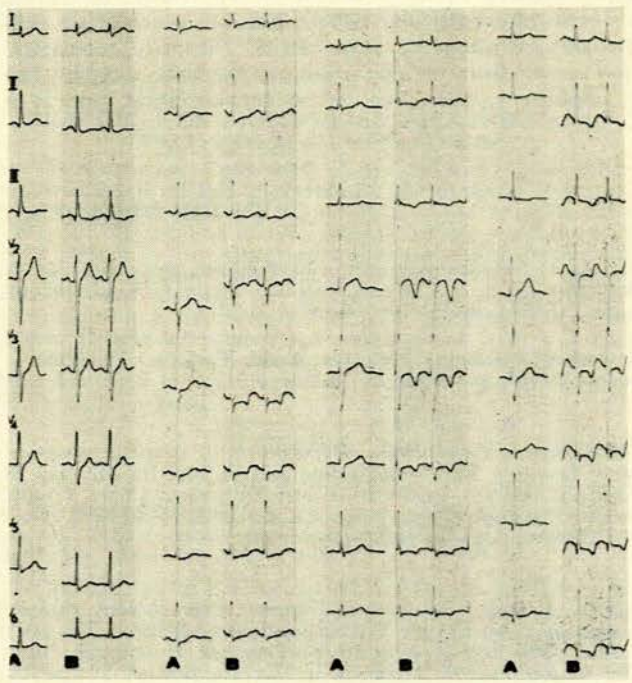

Fig. 2 *Fig. 3* *Fig. 4* *Fig. 5*

Fig. 2. Group I. A. Before. B. After inhalation of amyl nitrite there is flattening of all the precordial T waves.
Fig. 3. Group II. A. Before. B. After inhalation of amyl nitrite, flattening of all precordial T waves as well as prominent U waves occurs.
Fig. 4. Group III. A. Before. B. After inhalation of amyl nitrite there is inversion of the precordial T waves.
Fig. 5. Group IV. A. Before. B. After inhalation of amyl nitrite, flattening and inversion of precordial T waves as well as prominent U waves can be seen.

DISCUSSION

The increased heart rate associated with amyl nitrite administration is compensatory to the decreased peripheral vascular resistance and associated fall in systemic blood pressure.[2]

The shift of the electrical axis of the P wave to the right is probably due to the impulse originating in a relatively higher focus in the sino-atrial node.[3]

The factors causing the ventricular gradient and T wave changes are at present purely speculative. Both nitrates and nitrites inhibit adenosine triphosphatase activity, preventing adenosine triphosphate degradation with resultant loss of tone and relaxation of the smooth muscle of the vessels.[4] Apart from the vasodilatation caused by nitroglycerine, a raised myocardial oxygen consumption also occurs which does not comply with the haemodynamics involved.[5] Since changes in myocardial blood supply have been suggested as a cause of T wave changes,[6] this may play a role here.

On the other hand there is a striking similarity between the electrocardiograms of group II in this study and the ECG characteristics of hypokalaemia, viz. exceptionally high U waves (higher than T) and a simultaneous flattening of the T waves.[7]

Amyl nitrite causes a marked decrease in left ventricular stroke work secondary to a decrease in pressure work.[8] Thus another factor which must be considered is the lowered intraventricular systolic pressure which arises with the administration of amyl nitrite. Normally repolarization occurs from the epicardium to the endocardium; this is opposite to the direction of depolarization, and it is suggested that this phenomenon is due to the high intraventricular pressure to which the endocardium is subjected.[9] With a lowering of systolic pressure a change in the direction of repolarization can thus be considered. Complementary to this is the definite correlation between arterial pressure and U waves, as negative U waves in hypertensive patients become positive with control of the hypertension.[10]

SUMMARY

During and immediately after amyl nitrite inhalation in young adult males certain definite electrocardiographic changes occur, especially in regard to T and U waves, as well as changes in the ventricular gradient. The T and U wave changes can be classified according to 4 distinct groups. The probable mechanism effecting these changes is briefly discussed and includes differences in myocardial blood supply, a relative potassium deficiency and sudden intraventricular lowering of pressure.

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