The Effect of Hyperventilation on Various Arrhythmias

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The influence of hyperventilation on various cardiac arrhythmias is examined. Auricular and ventricular extrasystoles disappear temporarily, paroxysmal auricular and ventricular tachycardias as well as attacks of short paroxysmal auricular fibrillation are abolished by hyperventilation. An attempt is made to explain these phenomena.

ALTERATIONS of RS-T segment and of the T waves of the electrocardiogram have been described as a consequence of hyperventilation.\textsuperscript{5-13} This, however, is controversial, since such changes were not seen by other investigators,\textsuperscript{14} provided hyperventilation was performed in such a manner that a marked increase of the sinus rate was avoided and the respiration was not so shallow and rapid as to cause anoxia.

One of us observed a patient for several years who was subject to long paroxysms of auricular ectopic tachycardia. Hyperventilation in this patient was followed by a conspicuous reduction in the number of ectopic beats.\textsuperscript{12} There are no other studies known to us on the influence of hyperventilation on arrhythmias of various types. The present report deals with an investigation of the response of arrhythmias to hyperventilation. We do not propose to present a statistical analysis of the frequency of the changes since the series is too small; our report will be confined to the presentation of a few positive cases.

METHOD

Following the registration of control electrocardiograms the patients were instructed to hyperventilate by maximal inspiration and expiration. Care was taken to insure that the respiratory rate did not increase materially. If, as is often done, the patient is told to increase depth and rate of respira-

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tion "as much as possible"\textsuperscript{5} or to breathe "as rapidly as possible"\textsuperscript{14}, hypoxia of the myocardium is unavoidable since the volume of tidal air with this type of breathing is so reduced that the exchange of air involves mainly the dead space and does not contribute much to the exchange of gases in the lungs.

At the appearance of a Chvostek or Trousseau sign, normal respiration was substituted for hyperventilation. Electrocardiograms were repeated every minute during hyperventilation and for about 10 minutes thereafter. If significant alterations in the existing arrhythmias appeared during hyperventilation, the entire procedure was repeated whenever possible in order to exclude the possibility of a coincidence.

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Auricular Extrasystoles

E. S. was a 72 year-old woman. All records are reproduced from lead II. The top tracing (A) in figure 1 shows a sinus rhythm interrupted by short attacks of auricular fibrillation. In some areas the tracing resembles one of multiple auricular extrasystoles but the diagnosis of fibrillation is justified by irregularly formed F waves in several parts of the tracing. Hyperventilation was performed for five minutes before a Chvostek sign appeared. The sinus rate was unaffected and remained 90 to 100 beats per minute before and after hyperventilation. The disturbance of rhythm disappeared completely and a regular sinus rhythm prevailed (fig. 1B). Five minutes later auricular extrasystoles returned (fig. 1C) and short attacks of fibrillation recurred. After another five minute period of hyperventilation sinus rhythm reappeared (fig. 1D). Ten minutes after the second period of hyperventilation the electrocardiogram (fig. 1E) showed the same pattern as the control (fig. 1A).

In this patient hyperventilation abolished the extrasystoles and short attacks of auricular fibrillation without changing the rate of the basic sinus rhythm.
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FIG. 1. Lead II. (A) shows auricular extrasystoles and short attacks of auricular fibrillation before hyperventilation. Sinus rhythm appeared after hyperventilation (B). Five minutes later extrasystoles and fibrillation returned (C) but disappeared again after a second period of hyperventilation (D). (E) shows the return of the arrhythmias 10 minutes later.

Ventricular Extrasystoles

J. R., a 53 year-old male, suffered from coronary sclerosis and was hospitalized because of congestive heart failure. During the course of digitalis therapy ventricular extrasystoles appeared and exhibited characteristic, continuous changes of form (fig. 2A). The electrocardiograms shown were recorded in lead III.

Hyperventilation was performed until a Chvostek sign appeared. This led to complete disappearance of the extrasystoles (fig. 2B). In this instance, as in the previous case, the sinus rate was not affected by the hyperventilation and remained at 110 beats per minute. Within five minutes after the hyperventilation was discontinued the ventricular extrasystoles reappeared (fig. 2C), but renewed hyperventilation for five minutes again abolished them (fig. 2D). Two and one-half minutes after the second hyperventilation ceased ventricular extrasystoles of varying forms were again evident in the electrocardiogram (fig. 2E).

S. L., a 57 year-old male, was admitted because of coronary sclerosis. He had received digitalis for congestive heart failure. The tracings illustrated were recorded in lead III. Before hyperventilation the pattern of left bundle-branch block with multiform ventricular extrasystoles was recorded (fig. 3A). After four minutes of hyperventilation, which produced a Chvostek sign, a regular sinus rhythm occurred (fig. 3B). The rate was 60 before hyperventilation and 55 afterward. Following hyperventilation electrocardiograms were taken at one minute intervals. After seven minutes (fig. 3C) the same arrhythmia seen in figure 3A has recurred.

In one patient who had both auricular and ventricular extrasystoles, the ventricular ones dis-
FIG. 3. Lead III. (A) reveals multiform ventricular extrasystole with left bundle branch block. The extrasystoles disappear after four minutes of hyperventilation (B) and reappear seven minutes after the end of hyperventilation (C).

Paroxysmal Auricular Tachycardia

The hyperventilation experiment was performed on 16 patients with paroxysmal auricular tachycardia, but proved successful in only the one case which follows.

M. B., a 50 year-old male, was admitted with a long history of paroxysmal tachycardia, emphysema and rheumatoid arthritis. During one attack of paroxysmal tachycardia hyperventilation was attempted for eight minutes, but the patient cooperated poorly and a Chvostek sign failed to appear. No changes were observed in the attack and the patient refused to continue. He was discharged and returned in two months, claiming to have stopped many attacks by hyperventilation. When readmitted he had auricular tachycardia with a rate of 200 beats per minute (fig. 4A). The patient was actively hyperventilating for some time and could not be made to stop. About 20 seconds after figure 4A was taken, the attack stopped (fig. 4B). Only auricular extrasystoles were recorded. One minute later the attack recurred (fig. 4C), beginning with aberrant auricular extrasystoles. The patient could not be persuaded to hyperventilate again.

Paroxysmal Ventricular Tachycardia

S. V., a 72 year-old male, was admitted with the diagnosis of coronary sclerosis and auricular fibrillation. He had received moderate doses of digitalis. A ventricular tachycardia appeared which, because of the constant form of the ventricular complexes, could not be attributed, with certainty, to digitalis (fig. 5A). The rate of the tachycardia was 166 beats per minute. Hyperventilation lasting eight minutes led to the disappearance of the tachycardia (fig. 5B). Only a short series of extrasystoles appeared with a rate of 115. Three minutes after the end of hyperventilation the tachycardia had reappeared (fig. 5C). The rate was again 166. Hyperventilation, this time for nine minutes, again interrupted

FIG. 4. Lead II. (A) shows a regular paroxysmal auricular tachycardia. The attack stopped during hyperventilation (B) and only single auricular extrasystoles appeared. One minute later the attack recurred (C).
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FIG. 5. Lead III. Ventricular tachycardia (A) disappears after eight minutes of hyperventilation and is replaced by multiple extrasystoles with a slower rate (B). Three minutes after the end of hyperventilation the original tachycardia recurred (C) and it disappeared after a second period of hyperventilation lasting nine minutes (D). Multiform extrasystoles appear.

the tachycardia and multiform ventricular extrasystoles were recorded (fig. 5D).

F. H., a 52 year-old patient, was admitted because of an acute posterolateral infarction of the left ventricle. On examination the patient had a sinus rhythm with multiple ventricular extrasystoles. These became permanent within a few days and the pattern as seen in the three standard leads in figure 6 was continuously present. The ventricular rate was approximately 100 beats per minute. Figure 7A (lead I) was recorded after five minutes of hyperventilation. Regular groups of seven ventricular extrasystoles had appeared coupled to a sinus beat. Following 12 minutes of hyperventilation (fig. 7B) three ventricular extrasystoles followed each sinus beat. Thus, the number of extrasystoles had diminished markedly. A few minutes after the end of hyperventilation the sinus beats again became infrequent. Five minutes after hyperventilation ended the number of extrasystoles after each sinus beat had increased to six (fig. 7C), and after an additional 10 minutes normal sinus beats had disappeared and there was a continuous chain of extrasystoles (fig. 7D).

E. M., a 27 year-old male, was admitted because of a duodenal ulcer. The control electrocardiogram revealed a ventricular tachycardia with a rate of 84 beats per minute (fig. 8A and B). Very few sinus beats with a rate of 75 appeared; usually there was only one sinus beat, rarely three were encountered (fig. 8A). The chains of ventricular extrasystoles were so long because most of them were reversely conducted to the auricle and disturbed impulse formation in the sinus node. When this retrograde conduction ceased the sinus beats appeared. Combination beats were often seen because of the

FIG. 6. Paroxysmal ventricular tachycardia in the three standard leads.
Fig. 7. (A) shows Lead I of the same patient shown in figure 6 after hyperventilation lasting five minutes and (B), after hyperventilation lasting 12 minutes. Here only three ventricular extrasystoles follow each normal beat. Five minutes after the end of the hyperventilation (C) the number of extrasystoles increased and after 10 more minutes (D) there was again an uninterrupted chain of extrasystoles.

Simultaneous activation of a part of the ventricle by sinus impulse and ectopic impulse. Hyperventilation studies repeated 11 times in three weeks, always yielded the same results. In every experiment hyperventilation reestablished sinus rhythm within a few minutes. Figure 8C shows the continuous sinus rhythm four minutes after the beginning of hyperventilation and one minute after it had been discontinued. While the possibility exists that the sinus tachycardia at the end of hyperventilation prevented the appearance of the slow extrasystoles with long coupling, the tracing one minute after the end of hyperventilation (fig. 8C, second half) shows adequately long diastoles; therefore this explanation for the disappearance of the extrasystoles is unacceptable. Figure 8D shows the electrocardiogram six minutes after the end of hyperventilation with a sinus rhythm still persisting.

Fig. 8. Lead II. Attacks of ventricular tachycardia with only a few sinus beats between them (A and B). (C) shows a regular sinus rhythm after four minutes of hyperventilation; (D) taken one minute later shows still only sinus rhythm and so does the tracing in (E) obtained six minutes after the end of hyperventilation. The original arrhythmia had returned in (F) which was obtained shortly after (E).
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... This was never observed in this patient without hyperventilation but always appeared as a consequence of it. A few seconds later figure SE was recorded and showed the same pattern as the control electrocardiogram.

In one case of auricular fibrillation of several hours duration the fibrillation disappeared one minute after hyperventilation for eight minutes. It did not return until the patient was discharged three days later. A correlation between the hyperventilation and the disappearance of the fibrillation is not certain. In seven patients with auricular flutter hyperventilation was without effect.

DISCUSSION

The mechanism of these profound effects of hyperventilation upon the arrhythmias presented is not known. Two possibilities immediately appear as plausible explanations, namely, exercise and alteration in acid-base balance. Since our subjects breathed at approximately normal respiratory rates with little effort and since the heart rate did not increase during hyperventilation, exercise with its attendant change in sympathetic tonus can be ruled out. Changes of the blood pressure during hyperventilation in man are minimal.\(^6\)\(^,\)\(^8\)

Marked alterations in acid-base balance and in serum electrolytes have been noted in acute hyperventilation studies,\(^1\)\(^,\)\(^9\) of which variations in serum potassium and pH are most likely to affect cardiac impulse formation. Changes in serum potassium are minimal and cannot readily be expected to play a role. We know little, however, about the changes within the myocardial cells. A striking transient increase in the serum pH, occurring within two to three minutes after hyperventilation starts, persisting for approximately six minutes after it terminates and promptly returning to normal thereafter, has been substantiated in several studies.\(^2\)\(^,\)\(^4\) Thus it is possible that the mechanism involved, whatever it may be, depends upon alterations in the serum pH, since this change is significant and alterations in the form of arrhythmias are known to coincide with the change in pH. The importance of fluctuations in serum pH in the mechanism of the origin of extrasystoles, is well known, especially in regard to the effect on the supernormal phase of recovery.\(^7\)\(^,\)\(^12\) Since stimuli which are ordinarily subthreshold become effective or stimuli are formed during such periods of enhanced excitability, the supernormal phase has been assumed to have significance in the origin of extrasystoles, although it is not the sole factor involved. With these facts in mind, one can formulate the following tempting hypothesis. Voluntary overbreathing reduces alveolar carbon dioxide tension which, in turn, causes a reduction in the carbon dioxide tension of arterial blood and, consequently, an increase in the pH of arterial blood. This new pH lessens or abolishes the tendency to ectopic cardiac impulse formation by virtue of its effect on the supernormal recovery phase or through a change of the electrolyte balance. As a result the existing arrhythmia is either eliminated or markedly diminished. Upon cessation of overbreathing and rapid return of alveolar carbon dioxide tension and arterial pH to normal, the inhibition of ectopic impulse formation is released and the ectopic arrhythmia returns.

Extrasystoles initiated by aconitine disappear during asphyxia.\(^10\) Experimental analysis shows that this effect is not due to anoxia but is the consequence of hypercapnia; breathing of a mixture of 20 per cent carbon dioxide and 80 per cent oxygen abolishes the extrasystoles temporarily. These results are interesting since the appearance of dangerous ectopic ventricular arrhythmias and of ventricular fibrillation recently has been reported to follow rapid reduction of an existing hypercapnia.\(^3\)\(^,\)\(^11\)

SUMMARY

Instances of various arrhythmias which were temporarily diminished or abolished by voluntary overbreathing are presented.

The mechanism is unknown, but is believed to be the effect of increased pH of arterial blood upon the supernormal phase of recovery and perhaps changes of the electrolyte pattern.

SUMMARIO IN INTERLINGUA

Esseva studiate le influentia de hyperventilation super varie formas de arrhythmia cardiac. Esseva constatare que hyperventilation causa le disparation temporari de extrasystoles auricular e ventricular. Illo aboli tachycardias parox-
ysmal auricular e ventricular e accessos de breve fibrillation paroxysmal auricular.

Le mecanismo de iste effectos non es cognoscite, sed nos opinan que illo involve un augmentate pH del sanguine arterial e possiblemente un cambiate configuration del electrolytos.

REFERENCES

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