Seventy-two healthy young individuals were subjected to controlled, moderate hyperventilation with room air and monitored electrocardiograms. Of these, 60 percent exhibited flattening or inversion of T-waves in any one specific “normal” pattern. The incidence and magnitude of T-wave changes in a healthy young population was studied using this approach.

Hyperventilation (HV) has been shown to produce flattening or inversion of T-waves in the electrocardiograms of healthy individuals without heart disease. In most previous investigations, however, hyperventilation has been simulated by a short (15 to 30 sec) period of quick, very heavy breathing—a situation rarely encountered in routine clinical practice. The purpose of this study was to examine the electrocardiographic changes produced by controlled, moderate hyperventilation, in order to mitigate what occasionally occurs in the emotionally labile or reoperative patient. The incidence and magnitude of T-wave changes in a healthy young population was studied using this approach.

**REFERENCES**


**Hyperventilation-induced T-Wave Changes in the Limb Lead Electrocardiogram**

Grant S. Golden, B.A., Lawrence H. Golden, M.D., F.C.C.P.**, and Frederick R. Beerel, M.D., F.C.C.P.†

Seventy-two healthy young individuals were subjected to controlled, moderate hyperventilation with room air and with 4.9 percent CO₂ in air, and monitored electrocardiographically. Significant summed frontal T-wave changes with hyperventilation (ΣT₁₂₃ ≥ 1.5 mm) were observed in 12 patients. Six subjects (8.3 percent) showed T-wave depression. It was reversed in five patients by hyperventilation with 4.9 percent CO₂ in air. T-wave elevation, observed in six subjects, was reversed in four patients by hyperventilation with 4.9 percent CO₂. A short period of hyperventilation with an air mixture containing 4.5 percent CO₂ is suggested as a means of screening patients under suspicion of ischemic heart disease exclusively on the basis of ECG changes.

**Materials and Methods**

Seventy-two volunteers were chosen at random from the house staff and employees of the Millard Fillmore Hospital, Buffalo, New York. This sample population consisted of 34 men and 38 women, aged 18 to 40 years. The subjects gave a negative history of heart or pulmonary disease and had no physical complaints at the time of the experiment.

All investigative procedures were carried out in the sitting position. Using a Siemens monograph 34, a baseline limb-lead electrocardiogram (leads 1, 2, 3—recorded simultaneously) was obtained. V leads were avoided because of the lack of any one specific “normal” pattern for the right precordial leads, and because their application would have made routine screening more difficult. Resting arterial carbon dioxide tension, PaCO₂ was estimated by infrared analysis of end-tidal CO₂ using the Beckman LB-1 Medical Gas Analyzer. Throughout the procedures, conventional mouthpiece breathing was employed. The subject was asked to hyperventilate for three minutes with room air, at a rate specially chosen to reduce his estimated Paco₂ by 10-15 torr. In order to achieve this desired rate of breathing, the subject would voluntarily inspire and expire to the beat of a metronome, exchanging only a comfortable tidal volume throughout the trial period. Immediately after completion of hyperventilation, the ECG was again recorded. After a short rest interval, this hyperventilation procedure was repeated, at the same rate as previously established, using 4.9 percent CO₂ in air as the breathing medium. After three minutes of hyperventilation with this mixture, which normalized the Paco₂, the electrocardiogram was similarly repeated. The three tracings thus obtained were scanned for S-T segment alterations and analyzed for T-wave changes, using the summed frontal T-wave value (ΣT₁₂₃) as parameter for comparison. T-wave amplitudes were estimated to the nearest 0.5 mm, after measuring at least five consecutive complexes to obtain a satisfactory mean. For purposes of analysis and interpretation, certain arbitrary standards for summed T changes (ΔΣT₁₂₃) were adopted. A ΔΣT₁₂₃ of 1.5 mm was chosen as the lower limit for significant T wave change. Changes equal to or
Table 1—Condensed Experimental Data

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>( \Sigma T_{1,2,3} ) Resting (mm)</th>
<th>( \Sigma T_{1,2,3} ) HV (mm)</th>
<th>( \Sigma T_{1,2,3} ) HV 4.9% CO(_2) (mm)</th>
<th>PaCO(_2)* (torr)</th>
<th>( \Delta )PaCO(_2)* (torr) with HV</th>
<th>Resp Rate (breaths/min) during HV</th>
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<tr>
<td>Mean Population Data (72 patients)</td>
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<td>4.35</td>
<td>4.34</td>
<td>40.4</td>
<td>13.5</td>
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<td>( \pm 2.18 )</td>
<td>( \pm 2.21 )</td>
<td>( \pm 2.27 )</td>
<td>( \pm 4.3 )</td>
<td>( \pm 2.4 )</td>
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<td>#6</td>
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<td>9.0</td>
<td>10.5</td>
<td>32.9</td>
<td>11.9</td>
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<td>12.8</td>
<td>31.2</td>
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</table>

*As estimated from end-tidal CO\(_2\) values.*

greater than 1.5 mm but less than 3.0 mm were judged as "minor;" variations of 3.0 mm or more in \( \Sigma T_{1,2,3} \) were considered major changes.

RESULTS

The results of the study have been summarized in Table 1. Of the seventy-two subjects studied, a total of six, (8.3 percent) showed significant T-wave depression after hyperventilation. Each of these subjects was examined for the possibility of heart disease (history, physical examination, chest x-ray and electrocardiogram), and all were found to be without abnormalities. In every patient, the observed T-wave changes can be described as minor, and in five of six patients, they were reversible by HV with 4.9 percent CO\(_2\) (Fig 1). In the one odd instance, T-wave depression was intensified by the administration of 4.9 percent CO\(_2\) in air. In general, T-wave alterations were found to be transient in nature, spontaneously reversing themselves within one to three minutes after cessation of hyperventilation. In one patient, however, HV-induced changes persisted for 15 minutes after the completion of the experiment. All changes proved reproducible in subsequent testing seven to ten days after the original trials.

As a rule, T-wave changes occurred in the form of flattened upright or depressed negative T-waves; actual inversion of the upright T, a finding well documented for the mid and left precordial leads,\(^{1,2}\) was but rarely demonstrated on the frontal plane. S-T segment depression was observed in only a single case, and was also found to be reversible by hyperventilation with 4.9 percent CO\(_2\) (Fig 2).

A paradoxic increase in \( \Sigma T_{1,2,3} \) with hyperventilation was observed in six patients (Fig 3). These changes

CONTROL TRACING

AFTER HV, ROOM AIR

AFTER HV, 4.9% CO\(_2\) IN AIR

![Control Tracing](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20960/ on 06/26/2017)

**Figure 1.** T-wave depression induced by hyperventilation. Note: flattening of the repolarization wave in 2 and emergence of negative T in 3 directly after HV with room air. Similar changes were not observed after identical hyperventilation period with 4.9 percent CO\(_2\) in air.
results are consistent with the earlier observations of Christensen and Yu. The mechanism for the phenomenon is as yet obscure. In any case, it need not indicate latent heart disease, but is probably a benign, non-pathologic condition, present under the above described circumstances in about 10 percent of the population.

Since the restoration of T-wave voltages by HV with 4.9 percent CO₂ is reproducible in this normal group, HV with CO₂ might provide a simple screening procedure for apparently normal patients under suspicion of ischemic heart disease because of suggestive abnormal ECG changes.

Heightening of T-wave with hyperventilation, observed in the same frequency as T-wave depression, was an unexpected finding not previously described, to the authors’ knowledge. There is a possibility that these changes too, are induced by respiratory alkalosis, but the present evidence seems less conclusive, and subsequent testing appears indicated.

ACKNOWLEDGMENT: The generous assistance of Mrs. Frances Cirbus, chief technician at the Millard Fillmore Hospital Pulmonary Study Unit, is gratefully acknowledged.

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