Reflex Cardiovascular Responses to Cold Exposure of the Face or Foot

M.A.B. Frey, Ph.D., E.A. Selm, A.B., and J.W. Walther, Jr., M.D.

Summary

Six subjects performed a breathholding maneuver during facial cooling and immersed their foot in cold water, without drugs and after the intravenous administration of propranolol plus atropine (P+A). Cardiac interval (INT), mean interval for longest consecutive 5 cycles (L5INT/5); systolic time intervals including electromechanical systole (EMS), left ventricular ejection time (LVET), pre-ejection period (PEP), and PEP/LVET; and systolic (SP) and diastolic pressures (DP) were monitored during supine rest, during apnea with a plastic bag of ice water on the face, and from 16–30th and 46–60th sec of 1-min periods of foot immersion in 4°C water. P+A administration induced reduction in INT, L5INT/5, and LVET and increase in PEP, PEP/LVET, and DP. INT, L5INT/5, PEP, SP, and DP increased during facial cooling without drugs. Only the increases in INT and L5INT/5 were abolished by P+A and thus considered to result from reflexes mediated by vagal or sympathetic outflow to the heart. Reductions in INT, L5INT/5, EMS, PEP, and PEP/LVET at 16–30 sec of foot immersion without drugs were not observed after P+A; 46–60 sec responses neared resting values, however, with and without P+A. These results indicate an initial cardiac reflex response to foot immersion may be overpowered by the ventricular afterload and the baroreceptor response due to the increased arterial pressure.

Additional Indexing Words:
Face immersion Heart rate Cold pressor test Baroreceptors Systolic time intervals Vagus Left ventricular ejection time Sympathetic nervous system Pre-ejection period

EXPOSURE of the face to cold, usually cold water, and exposure of the hand or foot to a similar stimulus have been shown to elicit different cardiac responses. The mechanisms mediating these responses, however, have been only partially identified. Immersion of the face in cold water induces a
vagally-mediated bradycardia and peripheral vasoconstriction which is probably mediated by the alpha-adrenergic sympathetic system.\textsuperscript{1} The pre-ejection period (PEP) of systole is prolonged, but this occurs later in the face immersion than the bradycardia.\textsuperscript{2} It has not been established, however, whether this prolongation results from autonomic nervous system influences on the ventricle or from nonneural factors.

Hand or foot immersion in cold water initiates a different set of responses: prompt tachycardia followed by cardiac slowing\textsuperscript{3} along with increasing peripheral vasoconstriction including a significant decrease in forearm blood flow\textsuperscript{4}. Changes in duration of the pre-ejection period (initial shortening followed by a lengthening) which have been observed during limb immersion in cold water\textsuperscript{3,5} are also unexplained.

Our purpose in this investigation is to identify which of the components of cardiac response to face immersion and to limb immersion in cold water are neurally mediated. This would also help determine whether either of these stresses alone or both in concert could provide a comprehensive non-invasive cardiovascular test of autonomic function. Bennett et al.,\textsuperscript{6} after observing depressed response to face immersion in diabetic patients, suggested this as a longitudinal test of autonomic intactness.

\textbf{METHODS}

1. Subjects

Six subjects (5 male, 1 female) aged 22 to 31 years participated. All subjects were determined free from cardiovascular disease by history, physical examination, and maximum treadmill exercise test using the Bruce protocol. Physical characteristics of the subjects are enumerated in Table I. Resting blood pressures were determined at this visit by a trained observer using a mercury sphygmomanometer according to a standard procedure. The protocol was carefully ex-

<table>
<thead>
<tr>
<th>Subject number</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>Height (in)</th>
<th>Weight (lb)</th>
<th>Cigarette smoking</th>
<th>Peak workload (Mets)</th>
<th>Arterial pressure (resting, supine) (mmHg)</th>
</tr>
</thead>
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<td>M</td>
<td>32</td>
<td>74''</td>
<td>195</td>
<td>No</td>
<td>11.0</td>
<td>120/86</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>26</td>
<td>69''</td>
<td>185</td>
<td>1 pack daily</td>
<td>14.0</td>
<td>124/82</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>24</td>
<td>61-1/2''</td>
<td>120</td>
<td>No</td>
<td>9.0</td>
<td>118/68</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>22</td>
<td>68''</td>
<td>185</td>
<td>No</td>
<td>13.4</td>
<td>118/78</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>29</td>
<td>69''</td>
<td>200</td>
<td>No</td>
<td>12.6</td>
<td>130/84</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>26</td>
<td>68''</td>
<td>175</td>
<td>No</td>
<td>11.1</td>
<td>132/84</td>
</tr>
</tbody>
</table>
explained in detail to each subject, all questions answered, and consent obtained.

In addition to visiting the Cox Heart Institute for physical examination and treadmill test, each subject participated in 2 experimental sessions on different days separated by a week or less. At one visit subjects performed “face immersion” and foot immersion in cold water without drugs. On the other visit, propranolol and atropine were administered intravenously, to block chronotropic and inotropic influence of the beta-adrenergic and cholinergic systems, approximately 30 min before subjects performed these maneuvers.

2. Equipment and variables measured

Cardiac cycle duration, systolic time intervals, and arterial pressures were determined at rest and during these maneuvers at each visit.

Electrocardiogram (ECG), phonocardiogram, and carotid pulse contour were recorded simultaneously on an Electronics for Medicine (E-for-M) VR-12 photographic recorder at a paper speed of 100 mm/sec. Electrodes positioned on the chest to simulate a Lead II detected the onset of ventricular depolarization (Q wave), and a cardiotachometer provided continuous monitoring of heart rate during all experiments. An E-for-M PS-1-B microphone set to “sound” detection and connected through an AC/DC amplifier detected the phonocardiogram; and a similar transducer, adjusted for “pulse” detection and positioned at the point of maximum pulsation of the carotid artery, provided a recording of the pulse contour. Cycle duration (Q-Q), electromechanical systole (EMS), and left ventricular ejection time (LVET), are determined from these recordings, as shown in Fig. 1. Pre-ejection period (PEP) and PEP/LVET are calculated. The PEP, which includes the electromechanical delay and isovolumic contraction, is influenced by inotropic agents including sympathetic nervous system outflow; by ventricular filling (Starling mechanism); and by afterload which sets the pressure gradient the ventricle must overcome.7),8) PEP, especially the isovolumic contraction portion, is nearly independent of heart rate;9) LVET, however, is highly correlated with heart rate and is thus usually increased when cycle duration is increased.10),11) When the effect of heart rate is normalized, LVET has been shown to increase in duration as stroke volume increases.11) It is less influenced by arterial pressure or

![Fig. 1. Simultaneous recording of electrocardiogram, phonocardiogram, and carotid pulse contour showing how Q-Q INT, EMS, and LVET are determined for one beat. (Pre-ejection period = EMS-LVET.) EMS = electromechanical systole; LVET = left ventricular ejection time.](image-url)
inotropic influences than is PEP. The ratio PEP/LVET has been correlated with stroke volume and ejection fraction.\textsuperscript{12,13}

Arterial pressures were monitored, with cuff on the right arm, by a Narco automatic sphygmomanometer, with printed output through an E-for-M VR-6 recorder at a paper speed of 5 mm/sec, as shown in Fig. 2.

3. Procedures
(1) Control (without drugs)
After transducers were applied and subject had rested supine for a minimum of 15 min, cardiac variables were recorded for a 30-sec period on the VR-12 and blood pressure on the VR-6. Supine subjects then held their breath at end-inspiration while a technician supported a thin plastic bag filled with ice and water in contact with the nasal and maxillary areas of the face. This technique elicits the reflex, although responses may be attenuated.\textsuperscript{14} Subjects controlled the duration of the maneuver to their individual end points, indicating the initiation and the termination of breathhold by respectively closing and opening their eyes. Initiation and termination of the apneic-face cooling (AFC) period were marked on the recording. Data were recorded continuously from prior to breathhold until after resumption of breathing; only those data obtained during the actual face-cooling period, however, were analyzed.

After a recovery period of 5 to 10 min which allowed heart rate to return to pre-breathhold values, supine subjects immersed their left foot in a basin of water 4°C for 1 min or until withdrawal due to discomfort. Data were recorded during the intervals from 16-to-30th sec and from 46-to-60th sec of the “cold foot immersion” (CFI).

(2) “Reflex blocking” experiments
On this visit, after transducers were applied, propranolol-hydrochloride (0.2 mg/Kg) and atropine-sulfate (0.04 mg/Kg) were administered intravenously over 5 min. These doses of propranolol and atropine produce complete beta-adrenergic and cholinergic blockade, respectively, in man\textsuperscript{15} yielding an intrinsic heart rate.

Data were recorded for a 30-sec control period approximately 10 min after administration of the atropine, which followed the propranolol administration. Procedures for face cooling and foot immersion were identical to those on the “non-drug” visit. Subjects remained at the laboratory several hours after drug administration.

4. Data analysis
Variables were calculated on a beat-by-beat basis where applicable, using a

\begin{figure}[h]
\centering
\includegraphics[width=0.5\textwidth]{blood_pressure.png}
\caption{Sample recording of arterial cuff pressure with Korotkoff sounds superimposed. DP = diastolic pressure; SP = systolic pressure.}
\end{figure}
digitizer/computer system similar to that previously described.16) Mean values were determined for each subject in each of the conditions. Table II lists variables and conditions. The longest period of 5 consecutive cardiac cycles, or intervals, was identified during each condition and the mean of these 5 intervals determined (L5INT/5). This provides a measurement of the extent of short-term "face-immersion" bradycardia. Systolic and diastolic arterial pressures for each condition were read from the calibrated recording. Mean and standard error for the group of subjects for each variable in each condition were determined.

Resting control values for the variables in the propranolol-plus-atropine condition were compared with the "no-drug" condition by t test. Comparisons were made of the appropriate controls with values during apneic face cooling (AFC) and during the 16-to-30th- and 46-to-60th-sec periods of "cold foot immersion" (CFI), as well as between drug and no-drug conditions for each maneuver, by analysis of variance with p<.05 the level of significance. Where 0.05≤p≤0.10 is attained, however, that is so noted in the results herein.

**Table II. List of Variables Monitored and Experimental Conditions**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Abbreviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cycle interval</td>
<td>INT</td>
</tr>
<tr>
<td>Average interval of longest five-interval period during maneuver</td>
<td>L5INT/5</td>
</tr>
<tr>
<td>Electromechanical systole</td>
<td>EMM</td>
</tr>
<tr>
<td>Left ventricular ejection time</td>
<td>LVET</td>
</tr>
<tr>
<td>Pre-ejection period</td>
<td>PEP</td>
</tr>
<tr>
<td>Pre-ejection period/left ventricular ejection time</td>
<td>PEP/LVET</td>
</tr>
<tr>
<td>Systolic arterial pressure</td>
<td>SP</td>
</tr>
<tr>
<td>Diastolic arterial pressure</td>
<td>DP</td>
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</tbody>
</table>

Conditions

Supine, resting, no drugs

Apneic face cooling, no drugs

"Cold foot immersion", 16-30 sec, no drugs

"Cold foot immersion", 46-60 sec, no drugs

Supine, resting, propranolol plus atropine

Apneic face cooling, propranolol plus atropine

"Cold foot immersion", 16-30 sec, propranolol plus atropine

"Cold foot immersion", 46-60 sec, propranolol plus atropine

**RESULTS**

Data are summarized in Table III which lists mean, standard error, and number of observations for the 8 variables in all conditions.

1. Propranolol plus atropine administration

Blocking both the sympathetic and parasympathetic outflow to the heart decreased cycle duration (increased heart rate) including duration of the longest 5-interval period, and appeared to reduce variability among sub-
jects as evidenced by the smaller standard error. In these experiments, no change was observed in the duration of the systolic period, EMS, although the fractions of EMS were changed, with significant increases in PEP and PEP/LVET. Diastolic pressure was increased. These changes are shown in Fig. 3.

2. Apneic face cooling

Apneic face cooling induced a significant increase in cycle length (bradycardia). Without drugs the increase was 80 msec, whereas after the administration of the drugs it was reduced to 12 msec, which was reflected in a p<0.10 value for the interaction of the effects of the drugs and face cooling. Response as measured by L5INT/5 during the apneic face cooling was more dramatic, increasing from control to maneuver by 125 msec without the drugs and only 20 msec after drugs. The interaction here was significant at the p<0.05 level.

Neither EMS nor LVET were significantly changed by apneic face cooling; however, the PEP was increased with apneic face cooling, and the changes with and without the drugs were similar. Despite the reduction of heart rate, systolic and diastolic pressures were both increased, the effect on systolic pressure being less with cholinergic and beta-adrenergic blockade. These results are shown in Fig. 4.

3. Foot immersion in 4°C water

During the period from 16-to-30 sec of CFI without drugs, INT, L5INT/5, EMS, PEP, and PEP/LVET were all reduced, as shown in Fig. 5. None

<table>
<thead>
<tr>
<th>Variable</th>
<th>INT (msec)</th>
<th>L5INT/5 (msec)</th>
<th>EMS (msec)</th>
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<tr>
<td>Condition</td>
<td>Mean</td>
<td>S.E.</td>
<td>Mean</td>
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<tr>
<td>No Drugs</td>
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<tr>
<td>Rest</td>
<td>894</td>
<td>47.6</td>
<td>933</td>
</tr>
<tr>
<td>AFC</td>
<td>974</td>
<td>76.9</td>
<td>1058</td>
</tr>
<tr>
<td>CFI 15-30</td>
<td>783</td>
<td>69</td>
<td>821</td>
</tr>
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<td>N=5</td>
<td></td>
<td></td>
<td>N=5</td>
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<tr>
<td>CFI 46-60</td>
<td>893</td>
<td>60.6</td>
<td>931</td>
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<td></td>
<td></td>
<td>N=5</td>
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<tr>
<td>P+A</td>
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<tr>
<td>Rest</td>
<td>655</td>
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<tr>
<td>AFC</td>
<td>667</td>
<td>27.4</td>
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<tr>
<td>CFI 16-30</td>
<td>662</td>
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<td>666</td>
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<tr>
<td>CFI 46-40</td>
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N=6 unless otherwise noted.
Variables during All Experimental Conditions

<table>
<thead>
<tr>
<th>LVET (msec)</th>
<th>PEP (msec)</th>
<th>PEP/LVET</th>
<th>SP (mmHg)</th>
<th>DP (mmHg)</th>
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</thead>
<tbody>
<tr>
<td>Mean</td>
<td>S.E.</td>
<td>Mean</td>
<td>S.E.</td>
<td>Mean</td>
</tr>
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<td>280</td>
<td>7.9</td>
<td>91</td>
<td>7.2</td>
<td>.326</td>
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<td>N=5</td>
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<td>N=5</td>
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<tr>
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<td>104</td>
<td>6.9</td>
<td>.360</td>
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<tr>
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<td>N=5</td>
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<tr>
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<tr>
<td>270</td>
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<td>124</td>
<td>9.3</td>
<td>.460</td>
</tr>
<tr>
<td>N=4</td>
<td></td>
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<td></td>
<td>N=4</td>
</tr>
</tbody>
</table>

of these variables, however, were changed due to CFI after the administration of the drugs. On the other hand, systolic and diastolic pressures were elevated during the period 16 to 30 sec CFI about equally with or without drugs.

Between the measurements at 16-to-30 and 46-to-60 sec without drugs, the cardiac variables had returned to resting levels. EMS and PEP at 46-to-60 sec were longer than control in both no-drug and propranolol-plus-atropine situations. Reduction in number of subjects (N) from 6 to 5 or 4 was due to the premature foot withdrawal by 1 subject without drugs and 2 subjects in the drug session. Unfortunately, recording problems further reduced the number of blood pressure observations at 46-to-60 sec CFI. A sustained blood pressure increase during foot immersion in cold water has been previously observed3); therefore, we believe the lack of increase in pressure at 46-to-60 sec resulted from subject loss and was not physiologically significant.

**DISCUSSION**

In these studies using autonomic nervous system blocking drugs, we have identified which of the cardiac responses to "face immersion" and to foot immersion in cold water are neurally mediated and which result from other factors.

The responses we observed to intravenous propranolol plus atropine are very similar to those reported by others, where the variables have been pre-
Fig. 3. Cardiovascular effects of administration of intravenous propranolol plus atropine. Variables monitored supine at rest with no drugs and after the administration of P & A. N=6 unless otherwise noted. DP = diastolic pressure; EMS = electromechanical systole; INT = interval; L5INT/5 = longest 5-interval duration divided by 5; LVET = left ventricular ejection time; P+A = propranolol plus atropine; PEP = pre-ejection period; P_{P+A} = significance level of P+A; SP = systolic pressure.
Fig. 4. Cardiovascular effects of apneic face cooling. Variables monitored supine during apneic face cooling with no drugs and after administration of P & A. N=6 unless otherwise noted. Abbreviations same as Fig. 3 plus AFC=apneic face cooling; P_{AFC}=significance level of AFC; P_{INT}=significance level of interaction of drugs and AFC.

Previously monitored. Interval duration decreased from 894 msec (equivalent to a heart rate of 67 beats/min) to 655 msec (92 bpm) (refer to Fig. 3). This heart rate increase of 38% is similar to the 43% increase reported by Jose and
Fig. 5. Effect of foot immersion in 4°C water. Variables monitored supine at 16–30 and 46–60 sec of a 1-min foot immersion with no drugs and after administration of P & A. N = 6 unless otherwise noted. Abbreviations same as Fig. 3 and 4 plus CFI = cold foot immersion; P_{CFI} = significance level of CFI; P_{INT} = significance level of interaction of drugs and CFI.

Taylor, who also observed the abolition of sinus arrhythmia. A reduction in beat-to-beat variation in the recording of each of our subjects after drug administration confirms their observation. Intersubject variability in INT
and L5INT/5 also appeared to be reduced (Fig. 3). Arterial pressure increased from 124/70 (which calculates to a mean pressure of 88 mmHg) to 126/84 (mean pressure of 98 mmHg) which is approximately an 11% increase and is similar to the response reported by Jose and Taylor.15) Harris et al17) observed depressed ventricular function as indicated by a prolongation in PEP from 105 to 110 msec in their supine subjects after administration of propranolol. This is similar to, although slightly less than, the 15-msec change we observed. The total systolic period, EMS, is not significantly different in these subjects at rest on the day without drugs and the day on which sympathetic and parasympathetic blocking was accomplished. However, other data in our laboratory indicate EMS may be prolonged by the administration of these drugs.

Our technique of face cooling during breathhold induced the well-known “face immersion bradycardia,” bringing INT from 894 msec (equivalent to a heart rate of 67 beats/min) to 974 msec (62 bpm) (refer to Fig. 4). That the response is not as marked as reported by others is due not only to our method of facial cooling but also to the fact these figures consider all beats during the apneic period, and not just the period of maximum bradycardia which usually occurs late in the breathhold. If we compare our period of greatest bradycardia, L5INT/5, during facial cooling of 1058 msec (56 bpm) with the value for INT during control 894 msec (67 bpm) we have a decrease in heart rate of 16%. Heistad et al,1) whose seated subjects actually immersed their faces, observed a 23% decrease in heart rate during the last 10 sec of immersion.

After the administration of atropine (without propranolol), subjects performing face immersion in the Heistad et al study experienced a decrease in heart rate from 105 bpm resting to 103 bpm at 20 sec and 97 bpm at 30 sec of face immersion. Our subjects experienced almost no change in heart rate during apneic facial cooling after atropine and propranolol (from 655 msec [91 bpm] to 667 msec [90 bpm]). Together these results substantiate the reflex nature of face immersion bradycardia and its predominantly vagal mediation.

Responses of systolic time intervals to facial cooling (increase in PEP, without a change in LVET) agree with the previous results of Frey and Kenney.3) After autonomic blocking, all systolic intervals respond exactly as before blocking, that is, increases in PEP and PEP/LVET with no significant change in EMS or LVET, indicating that the changes induced by face cooling are not mediated by vagal or sympathetic outflow to the heart, although in some instances baseline (pre-face-cooling) values are changed. Two possibilities are suggested: (1) the increase in afterload during face immersion
is sufficient to prolong the isovolumic contraction period, and (2) there is a
decrease in venous return per beat, which puts the ventricular muscle at a
lower position on the Frank-Starling length-tension curve. This could be
due to a pooling of blood in the periphery during breathhold-face immersion,
but is considered less likely since the slower heart rate during face immersion
increases filling time.

Mean arterial pressures measured intra-arterially by Heistad et al,1) in
seated subjects were, at 77 mmHg, somewhat lower than our resting value of
88 mmHg; however, changes during face immersion to 92 mmHg are similar
to our observed changes to 100 mmHg. The increase in arterial pressure they
observed with face immersion after administration of atropine, from 82 to
95 mmHg, was slightly greater than that reported in this study after ad-
ministration of propranolol plus atropine. In fact, analysis of variance reveals
a significant interaction between the drugs and face cooling for systolic pressure
in the current investigation. This systolic pressure effect could be due to an
increase in stroke volume with increased filling at the lower rate heart at-
tained during facial cooling without drugs.

In general, it appears autonomically mediated effects during facial cooling
are limited to vagal chronotropic effects on the heart and alpha-adrenergic
sympathetic outflow to resistance vessels.

Early cardiovascular responses to foot immersion in cold (4°C) water
(that is, at 16-to-30 sec) indicate widespread sympathetic outflow to the heart
and vessels, probably accompanied by vagal withdrawal influencing chrono-
tropic responses (refer to Fig. 5). This is evidenced in the present study by
shortening of INT, L5INT/5, EMS, and PEP, and a decrease in PEP/LVET,
effects which are abolished after the administration of beta-adrenergic and
cholinergic blocking agents. Obrist et al18) also monitored heart rate responses
to foot immersion in cold water before and after administration of propranolol.
Comparing the Obrist et al study with the present study is difficult, since
Obrist averaged all data during a 90-sec immersion. However, the cardiac
acceleration they observed without drugs was approximately halved with
propranolol indicating a combination of sympathetic outflow and vagal with-
drawal during the immersion.

On the other hand, responses in arterial pressure are similar with and
without the drugs, thus this component probably results from an increase in
peripheral resistance due to stimulation of alpha-adrenergic receptors. Obrist
et al observed arterial pressure responses similar to those observed in this
investigation, which were also independent of propranolol administration.
Aizawa et al4) report reduced forearm blood flow which was minimal at
15 sec of a 35-sec immersion.
At the end of 1 min immersion, the cardiac responses are reversed; INT, L5INT/5, EMS, PEP, and PEP/LVET have returned to or exceeded control values. Furthermore, duration of PEP at 60-sec CFI exceeds control in both blocked and free-acting nervous system states—with the prolongation greater where there is no nervous system influence. This strongly suggests that the prolongation of PEP is due to the afterload, which is partly offset by sympathetic influence in the intact system.

Autonomic effects of CFI, thus, appear to encompass sympathetic outflow to the heart controlling both rate and contractility, in addition to modest vagal withdrawal affecting heart rate, and a sometimes powerful alpha-adrenergic influence on vessels. The fact that arterial pressure remains elevated while heart rate, initially elevated, returns toward control values is an evidence that the sympathetic nervous system effect on the vessels is of longer duration than the effect on the heart. The elevated pressure apparently provides sufficient stimulus for the baroreceptor reflex to overwhelm the cardiac response to the cold, while the vascular baroreceptor response is relatively slower developing due to the slower velocity of contraction and the hysteresis of the smooth muscle in the resistance vessels. In addition, the observation that INT and L5INT/5 are abbreviated at 30 sec CFI but are prolonged back to resting values by 60 sec CFI—after arterial pressure is increased in these young, healthy individuals—suggests CFI may provide a tool for measuring baroreflex responsiveness.

In this investigation we have shown that, while the bradycardial response to face immersion is mediated through the autonomic nervous system, the observed prolongation in PEP is not mediated by autonomic influences. We have also demonstrated that the initial autonomically mediated inotropic and chronotropic response to limb immersion in cold water is overwhelmed

<table>
<thead>
<tr>
<th>Table IV. Summary of Components Involved in Response to Each Stress</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Apneic face immersion</strong></td>
</tr>
<tr>
<td>Chronotropic cholinergic parasympathetic</td>
</tr>
<tr>
<td>Chronotropic beta-adrenergic sympathetic</td>
</tr>
<tr>
<td>Inotropic beta-adrenergic sympathetic</td>
</tr>
<tr>
<td>Vasoconstriction alpha-adrenergic sympathetic</td>
</tr>
<tr>
<td>Baroreceptor reflex (Secondary)</td>
</tr>
</tbody>
</table>
by the increased arterial pressure acting both as increased afterload for the ventricle and as stimulus for a baroreceptor reflex.

Thus, it appears that while neither face immersion nor cold limb immersion alone invoke all aspects of autonomic nervous system influence on the heart, in combination they may provide a tool to test cardiac/autonomic responses, as summarized in Table IV.

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