The Effect of Swimming on Patients with Ischemic Heart Disease

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SUMMARY  Swimming is frequently recommended for cardiac rehabilitation, but little is known of its physiologic consequences in ischemic heart disease. Eight males who had had a myocardial infarction 8-17 months before the study were exercised to exhaustion or angina with 10 W/min ramp on a cycle ergometer in sitting and supine positions. Oxygen uptake ($\bar{V}O_2$) was continuously measured to monitor the physiologic power requirement. All eight patients were taking $\beta$ blockers and four were taking digoxin. During sitting cycling, angina occurred in four and ST depression in five; during supine cycling, angina occurred in five and ST depression in six. $\bar{V}O_2$ was then measured while they swam at their own comfortable speed (mean 0.43 m/sec$^{-1}$) in a swimming flume at water temperatures of 25.5°C and 18°C. In six, the water speed was gradually increased until they were limited by symptoms. Comfortable swimming at 25.5°C was 87% (1.28 1/min$^{-1}$) and at 18°C 89% (1.30 1/min$^{-1}$) of sitting peak $\bar{V}O_2$, while heart rates were 92% and 91% respectively. The mean peak $\bar{V}O_2$ and heart rate did not differ significantly between bicycle and swim tests (peak $\bar{V}O_2$ sitting 1.49 ± 0.23, supine 1.42 ± 0.24, 25.5°C 1.60 ± 0.17, 18°C 1.52 ± 0.19 1/min$^{-1}$). Only two patients reported angina while swimming in warm water and one in cold water, although ST depression occurred in six in both swims. The subjective comfort and large muscle groups involved make swimming a good exercise, but the high relative energy cost and failure to identify ischemic symptoms indicate caution in cardiac patients, especially if their swimming skills are poor.

The Physiology of swimming in fit normal subjects and trained swimmers, both young and old, has been described, but the pathophysiology of swimming in patients with ischemic heart disease has been studied little. Our understanding of this condition raises theoretical concerns. Immersion in water and a horizontal body position both increase the central blood volume, which could stress the limited reserves of patients with ischemic heart disease. The large contribution of work from the arms, which results in a higher peripheral vascular resistance than leg work at the same work load, the compressive effect of the water on the extremities and the decreased skin blood flow, all increase the left ventricular afterload and thus might decrease the subjects' peak work level. The swimming flume, a kind of "swimming treadmill," offered a controlled and safe environment for evaluating such patients. Oxygen uptake ($\bar{V}O_2$) was used as an objective measure of the amount of work performed, and the ECG as an indicator of ischemic changes. The swimming results were related to the subjects' performance in the sitting and supine positions on a bicycle ergometer. Further, swimming tests were performed with water temperatures of 18°C and 25°C to assess the effect of different water temperatures.

Patients and Methods

We selected eight males, ages 49-66 years, from a cardiac rehabilitation program. They all had a myocardial infarction 8-17 months previously. Preference was given to patients with a history of angina or ST depression on a work test. All eight were taking $\beta$ blockers and four were taking digoxin. These were continued throughout the study. Further patient data are given in table 1.

A 12-lead ECG was obtained before the sitting cycling test and the six chest leads were recorded on a multichannel recorder during both cycling tests. Heart rate was determined by means of a cardiotachometer. During swimming, the ECG signal from a CMz bipolar lead was transmitted telemetrically to an FM receiver and recorded by an Elema Mingograph. Significant ST depression was assessed as greater than 1 mm ST depression 0.08 second after the J point.

In the first two swimming tests, $\bar{V}O_2$ was measured by the Douglas-bag method. In all other cycling and swimming tests, $\bar{V}O_2$ and other gas-exchange variables were measured continuously. In the continuous system, expired air passed through a wide-bore, low-resistance valve to a 6-l mixing box and was analyzed by a Centronic 200 MGA mass spectrometer. Argon was added just distal to the expiratory valve and also analyzed by the mass spectrometer so that ventilation ($V_E$) could be assessed by an argon dilution method. A respiratory pump was used to calibrate the $V_E$ monitoring system. The data from the mass spectrometer and the heart rate were recorded by a four-channel tape recorder. $\bar{V}O_2$, CO$_2$ output, $V_E$, heart rate and oxygen pulse could then be calculated off-line on a Hewlett Packard 9830 A calculator and graphed by an HP 9866 plotter. The following formulas were used:

$$V_E = \frac{k_{FAR}}{V_{AR}} = k_{BTPS} \times \frac{V_{AR}(0.9907-F_{AR})}{F_{AR}} \text{ l/min BTPS}$$

$$\bar{V}O_2 = k_{STPD} \times \bar{V}E(\bar{V}O_2 - F_{E\bar{O}_2}) \text{ l/min STPD}$$

$O_2$ pulse = $\bar{V}O_2$/HR (ml/beat)

where $F_{AR}$ = the difference of argon concentration
from air, \(F_{I\text{O}_2}\) = inspired oxygen, and \(F_{E\text{O}_2}\) = mixed expired oxygen concentration, and HR = heart rate. The peak \(\text{VO}_2\) was assessed by manually integrating the last 30 seconds of the continuous tracing.

On two different days within 3 weeks of the swimming test, the patients performed both sitting and supine work tests on an electrically braked cycle ergometer. The patients exercised until they could go no further or developed grade 6 symptoms of dyspnea or angina as judged by a Borg scale of 1-10. In the sitting test they sat for 3 minutes at rest, pedaled for 3 minutes at a load of 20 W and then against a work load that increased by 10 W each minute. During the supine test, the patients first rested for 2 minutes, had their feet on the pedals for another 2 minutes, and then started pedaling at an initial load of 20 W, which was increased by 10 W each minute.

The swimming studies were performed in the swimming flume of the G.I.H. in Stockholm on two separate days at water temperatures of 25.5°C and 18°C, respectively. On the first day, the subjects were timed as they swam two lengths of a 25-meter pool at their own comfortable speed. From this we calculated their water speed and will refer to this as their comfortable speed. Arterial blood pressure was measured before the swimming tests while the subjects were submerged to the level of the neck, but not during swimming. After standing for 2 minutes in the water, the patients swam at comfortable speed for 4 minutes. In the first two subjects in whom Douglas bags were used, the comfortable speed was continued for up to 7 minutes. Because in three of these four studies the two subjects were limited by symptoms before 7 minutes, these data were included in the analyses of the peak response. In the other six, after the first 4 minutes of comfortable speed, the water speed was increased by 0.05 m/sec until the subjects had to stop.

By their own choice, all subjects swim the breast stroke with their head out of the water. The breast stroke can load the oxygen transport system to approximately the same extent as front crawl, allows easy communication with the subject, and requires a relatively low swimming speed to elicit a maximal physiologic response. The subjects were not specifically trained in swimming nor was swimming a part of their rehabilitation program; swimming skills were not assessed before the test.

A defibrillator and dry plastic matting were kept at the side of the pool. One of the investigators was designated as the “dry” person. In the event of a cardiac arrest he was to remain dry so he could safely defibrillate the patient.

The data were first analyzed by Friedman’s nonparametric test for analysis of variance. Differences between exercises were then analyzed by the Wilcoxon’s rank sum test for paired data. Values are given as mean ± SD.

### Results

The swimming flume and mouthpiece were well tolerated by all subjects and there were no medical complications. The only arrhythmias that occurred were occasional ectopic beats on entering the water, a run of bigeminy in one subject in both swims and a short run of bigeminy after exercise in another subject.

The \(\text{VO}_2\) during comfortable speed swimming was 1.28 ± 0.12 l/min in warm water and 1.30 ± 0.20 l/min in cold water (table 2) \((p = NS)\). This was 87 ± 19% in warm and 89 ± 18% in cold water of the peak \(\text{VO}_2\) achieved during cycling. When the mean \(\text{VO}_2\) during comfortable speed swimming was compared with the highest peak \(\text{VO}_2\) obtained in any of the four tests, comfortable speed swimming required 81 ± 16% at 25.5°C and 83 ± 17% at 18°C of the peak \(\text{VO}_2\). In three subjects in warm water and an additional two in cold water, the comfortable speed swim required a peak \(\text{VO}_2\) that was the same as or higher than that during cycling (fig. 1A).

The peak \(\text{VO}_2\) during swimming was higher than or equal to the peak \(\text{VO}_2\) during cycling in the sitting position in seven of the eight subjects (fig. 1), but there

<table>
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<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>Angina</th>
<th>ASAT* (SIU)</th>
<th>Months from AMI</th>
<th>Medication</th>
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*Highest level recorded during acute myocardial infarction.
Abbreviations: ASAT = aspartate-arginine amino transferase; SIU = standard international units; AMI = acute myocardial infarction.
was no statistically significant difference between the mean peak VO₂ of 1.60 ± 0.17 l/min during swimming at 25.5°C and 1.52 ± 0.19 l/min at 18°C compared with the sitting cycling peak VO₂ of 1.49 ± 0.23 l/min (table 2). The mean peak VO₂ of 1.60 ± 0.17 l/min during warm water swimming was significantly greater than the mean peak of 1.42 ± 0.24 l/min (p < 0.01) during supine cycling (table 2, fig. 1).

The peak VO₂ during swimming increased linearly with the maximal water speed in the subjects who swam against progressively increasing water speeds (peak VO₂ = 0.79 ± 1.02 x water speed at 25.5°C, r = 0.89, p < 0.01). There was no clear relationship of the level of VO₂ to water speed during the comfortable speed swimming. The VO₂ could remain the same during the initial increase in water speed (fig. 2). This subject increased his speed from 0.35 to 0.75 m/sec before he had any increase in VO₂. This cannot be accounted for by a lag in the measuring system, for even with the large increase in demand at the onset of exercise, he reached his steady-state value in less than 2 minutes.

The maximal heart rate, ventilation and oxygen pulse did not differ between cycling and swimming (fig. 1, table 2).

There was no significant difference in the peak and comfortable speed VO₂ between the swims in warm and cold water (fig. 3). Most subjects swam for a shorter time and reached a lower speed in cold water, but this difference was not significant. The mean blood pressure while resting in the water was 113 ± 12 mm Hg before the cold water swim and 101 ± 6 mm Hg before the warm water swim (p < 0.05). Blood pressures during swimming were not obtained. The maximal heart rate did not differ between warm and cold water swims (fig. 3).

The commonest reason subjects stopped swimming was shortness of breath. When asked how they perceived the work of swimming in cold water as compared with warm water, five reported no difference, three felt the cold water swim was easier, and none felt the warm water swim was easier. Four subjects had angina and ST depression during sitting cycling, whereas five had angina and six had ST depression during supine exercise (table 3). The same six subjects

Table 3. Heart Rate at Onset of ST Depression

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<tr>
<th>Pt</th>
<th>Angina*</th>
<th>Heart rate (beats/min)</th>
<th>Angina*</th>
<th>Heart rate (beats/min)</th>
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<th>Heart rate (beats/min)</th>
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<td>ST↓</td>
<td>Supine</td>
<td>ST↓</td>
<td>Swimming</td>
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<td>5</td>
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<td>79</td>
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</table>

*Borg scale (1–10).12

Abbreviation: ST↓ = ST-segment depression.
had ST depression during both swims, but only two
had angina in warm water and one (a different patient)
in cold water. The heart rate at which ST depression
occurred was similar during swimming and cycling
(sitting 97 ± 16 beats/min, supine 93 ± 17 beats/min;
swim at 25.5°C, 96 ± 14 beats/min, swim at 18°C,
86 ± 15 beats/min, but only five had ST depression
during sitting cycling, compared with six subjects in
the other three tests.

Discussion

Knowledge of the physiologic power requirement is
essential in analyzing the effects of swimming in
patients with ischemic heart disease, for this gives
some idea of the relative stress of the exercise. Because
the power requirement of swimming cannot easily be
studied directly, we used VO₂ as a measure of the
energy cost and related this to the maximal VO₂ on a
cycle ergometer. This approach has been used to
assess the power requirement of other activities. By
this method, although comfortable speed swimming
only required a VO₂ of 0.85-1.47 l/min, it averaged
87% in warm water and 89% in cold water of the peak
VO₂ during sitting cycling. Similarly, in cold water,
the heart rate was 92% in warm water and 91% of the
maximal heart rate during sitting cycling. This high
percentage of the maximum response was partly
because our subjects' maximal exercise capacity was
reduced by their previous myocardial infarctions, but
was also related to the subjects' skill. For example,
the best swimmer, patient 1 (fig. 2), used only 68%
(VO₂ = 1.07 l/min) the first day and 54% (VO₂ = 0.85
1/min) the second day of his sitting cycling maximum $V_02$, while the worst swimmer, patient 3, used 108% ($V_02 = 1.47$ 1/min) the first day and 103% ($V_02 = 1.40$ 1/min) the second day of his sitting cycling maximum. This is a reflection of the wide range of mechanical efficiency during swimming which, in contrast to cycling, is highly dependent on the subject’s skill. The mechanical efficiency also varied considerably in individual subjects. An example is given in figure 3, where the subject doubled his swimming speed before he had any increase in his $V_02$ and then his $V_02$ increased rapidly with the increasing speed.

A major factor in swimming is the body build for this determines the sinking force and the drag force, both of which must be overcome during swimming. We could not reliably weigh our subjects in the water because they could not perform a full expiration while completely submerged, but they all floated easily and so the sinking force was probably not a major factor. Drag forces become increasingly important at higher speeds than those our subjects attained.

An unexpected finding was that the maximum $V_02$ obtained during swimming was at least as great as that during sitting cycling in seven of eight subjects. In studies in younger, untrained (for swimming) normal subjects, the maximum $V_02$ during swimming was about 10% less than cycling. Only elite swimmers attained their cycling maximum. In a study of healthy old men, a higher maximum $V_02$ was obtained during swimming than during stationary cycling, but the subjects all participated in a swimming program. Both of these studies concluded that only those subjects who were specifically trained in swimming would obtain equal peak $V_02$ during swimming and cycling. This cannot explain our results, for our subjects only performed light arm exercise during their rehabilitation program and had not swum for at least 1 year. Our subjects probably achieved equal mean peak $V_02$ during swimming and cycling because they were less limited by symptoms during swimming. This is supported by the fact that only those with evidence of ischemia had their highest peak $V_02$ during swimming.

Except in the one subject who achieved a higher peak heart rate during swimming, the increased peak $V_02$ was probably due to peripheral rather than central factors, because the peak stroke volume has been shown to be similar during swimming and other forms of exercise. This increased peripheral use of oxygen is probably not due to an increased intrinsic capacity for $O_2$ extraction, because the arms perform a major portion of the work of swimming and there is no need for postural muscles, making the total muscle involvement less than in cycling.

Our results differ from those of Heigenhauser et al., who found, in a group of patients who had had a myocardial infarction, that the peak $V_02$ during swimming was 21% lower than during cycling. This was due to a lower stroke volume during swimming than cycling. A possible explanation for the differences between their study and ours is that their subjects may have failed to reach a maximal effort during swimming. The ramp format protocol we used during swimming brings out a maximal effort in short time. Also, none of their patients had angina or ECG evidence of ischemia, which were present in six of our subjects. Fletcher et al. also found that swimming required a high $V_02$ compared with other exercises, but they did not compare peak values.
An important aspect of swimming in subjects with ischemic heart disease is the occurrence of signs and symptoms of exercise-induced ischemia. The same six subjects who developed ST depression during supine cycling had ST depression during both swims; and even though five had angina during the cycling test, only two had angina in warm water and one other subject had angina in cold water (table 3). Perhaps the sensation of dyspnea, which was the commonest complaint, overrode any perception of their more typical angina. Alternatively, either the water passing over the chest or the rhythmic arm movement may have acted as a counterirritant and blocked the subjects' perception of their angina. It is unlikely that the experimental situation itself distracted the subjects, for there was a defibrillator at the poolside and they were constantly asked how they felt. Although seven of the eight subjects expressed a preference for swimming over cycling, the failure to recognize symptoms must be kept in mind when patients with ischemic heart disease are being considered for a swimming program.

To understand the pathophysiology of angina in these patients, we must consider the three major factors that determine myocardial oxygen consumption:
heart rate, contractility and wall tension. The heart rate accelerated rapidly during swimming and reached over 90% of the maximal value even during comfortable speed swimming. Thus, the ST depression usually occurred in the first 2 minutes of swimming and although the rapid acceleration made the exact determination of the onset of ST depression difficult, it appeared to be similar during swimming and cycling (table 3). Because the maximal heart rates were the same during swimming and cycling, there was a similar effect on myocardial oxygen demand. Contractility could not be assessed in our study, but would also have been expected to be similar during swimming and cycling. Left ventricular wall tension would be expected to be higher during swimming than cycling for at least two reasons. Swimming work results in a higher blood pressure, a major determinant of left ventricular wall tension, at the same VO₂ as running. Cycling, in turn, produces changes in blood pressure similar to those with running. This results from decreased skin blood flow, the compressive effect of water, and the smaller muscle mass involved in swimming compared with running and cycling. Second, the increase in central blood volume that occurs during swimming would increase myocardial oxygen demand. The increased volume would also elevate left ventricular end-diastolic pressure in hearts that are noncompliant from previous myocardial infarction. This would in turn increase the pulmonary venous pressure and might explain why our subjects complained more dyspnea than angina.

There was no difference between the warm and cold water swims except for a shorter duration of the cold water swim and a moderately higher resting blood pressure in cold than in warm water. The potential importance of blood pressure is illustrated by one subject who had a resting blood pressure of 135/70 mm Hg and maximal heart rate during exercise of 115 beats/min in water at 25°C, but a resting blood pressure of 200/100 mm Hg and maximal heart rate of 105 beats/min in water at 18°C. He was also the only subject who had lower peak VO₂ during swimming than during cycling; his peak VO₂ was 1.83 during sitting cycling and 1.62 in warm and 1.60 l/min in cold water swimming. The elevated resting blood pressure in cold water was not associated with a lower peak VO₂, but, as a result, he reached his peak in a shorter time and at a lower heart rate.

Although we did not study this, water temperature close to body temperature could result in a lower diastolic pressure by causing peripheral vasodilation, and thus lead to decreased diastolic coronary artery perfusion and earlier ischemia during swimming, as has been found to occur in sauna baths. Thus, water that is too warm may be as detrimental, if not more so, than water that is too cold. All of our patients were taking β blockers and four were taking digoxin. We continued these medications for safety’s sake and because this is the state under which such patients will be exercising. Studies in normal subjects and patients with ischemic heart disease have shown that subjects with normal ventricles reach a lower peak VO₂ while taking β blockers, while subjects with reduced left ventricular function from ischemia can sometimes obtain a higher peak VO₂. There is also a decrease in the peak pressure-rate product. Although the limitation to peak VO₂ and cardiac output apply to any exercise, β blockers could reduce some of the greater blood pressure rise per work load that occurs during swimming and allow a greater peak VO₂ than without. The other important drug was digoxin, which does not affect the peak VO₂ but does make the ECG interpretation of ischemia less dependable. Of the four subjects taking digoxin, only two had ST depression with exercise, and this was associated in both with chest pain during sitting and supine cycling tests. We therefore considered it valid to interpret their ST depression as ischemic.

In conclusion, we found that in patients with a reduced exercise capacity due to ischemic heart disease, swimming, even at a comfortable speed, can require near-maximal effort. In contrast to normal subjects, good as well as poor swimmers achieved the same peak VO₂ during swimming as they did during cycling. There appeared to be a failure to identify ischemic symptoms. Swimming at 18°C and 25°C were equally well tolerated. Several recommendations can be made. Because there is a high aerobic cost, swimming can be an effective method of training the oxygen transport system, but because poor swimmers require a sudden large output from the oxygen transport system, poor swimmers with ischemic heart disease should avoid swimming as an exercise unless properly supervised. Because the heart rate at which ST depression occurred was similar during cycling and swimming, this heart rate can be obtained from a treadmill or cycle test and used as a guide when swimming is used in a cardiac rehabilitation program. With these guidelines, each patient must be treated individually and the role of swimming in a rehabilitation program must be judged according to the degree and type of disease and the patient’s skill and interest.

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