Use of a Transcutaneous PO_2 Regional Perfusion Index to Quantify Tissue Perfusion in Peripheral Vascular Disease

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In order to develop transcutaneous oxygen tension ($PtcO_2$) measurements into a practical method for assessing peripheral vascular disease, the relationships between extremity and chest wall $PtcO_2$ were examined in subjects with and without systemic atherosclerotic disease. The ratio of extremity to chest $PtcO_2$, or transcutaneous regional perfusion index (RPI) assessed limb oxygenation more reliably than did direct $PtcO_2$ measurement by obviating the effects of changes in systemic oxygen delivery upon local $PtcO_2$. The authors find that transcutaneous oximetry can be used during treadmill exercise testing and that the RPI is unchanged by exercise in all normal subjects. $PtcO_2$ and RPI were then measured during rest, position change, and exercise testing in patients with intermittent claudication. Whereas normal subjects maintain a constant thigh and calf RPI during exercise, patients with intermittent claudication consistently manifested large decreases in RPI in these areas when they were exercised until symptomatic. The authors find no overlap between the responses of normal subjects and patients with claudication; positive findings are, therefore, highly specific for exercise-induced limb ischemia. Since transcutaneous RPI exercise testing is easily performed and highly reproducible, it is well suited to clinical use in the diagnosis and documentation of intermittent claudication. Furthermore, since limb ischemia can be quantified, this method lends itself both to grading the severity of disease and to evaluating clinical progression of disease. It is suggested that such a quantitative approach to evaluation of intermittent claudication may allow refinement and extension of the indications for operative intervention in patients with intermittent claudication.

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THE FINAL common pathway of tissue injury in atherosclerotic peripheral vascular disease (ASPVD) is inadequate oxygen delivery to the limb.[1] Intermittent claudication occurs when the obstruction of arterial inflow to the leg is not so marked as to limit oxidative metabolism at rest, but progressively restricts the increases in oxygen delivery that are necessary during exercise. In the most severe cases, intermittent claudication may merge with the syndrome of rest pain or continuous ischemia.

Clinical measurements of tissue oxygenation were not feasible, however, until the development of miniaturized, heated Clark polarographic oxygen sensors that could measure the skin surface oxygen tension.[2] Transcutaneous oxygen tension ($PtcO_2$) directly and noninvasively measures local skin oxygenation,[3] but $PtcO_2$ is a complex function that reflects the local balance of oxygen supply and demand.[4,5] The heated electrode liquefies and disorganizes the solid crystalline structure of the stratum corneum, allowing rapid gas diffusion and a short sensor response time.[6] However, the heat has three other effects upon $PtcO_2$: dermal capillaries dilate; oxyhemoglobin dissociation is enhanced, increasing the local oxygen supply; and skin oxygen consumption is increased.[4,6] These effects compete, resulting in $PtcO_2$ values on the anterior chest that average about 80% of the PaO_2 in supine adults.[7] The authors[4] have previously demonstrated that the $PtcO_2$ is directly related to the total systemic oxygen delivery ($\dot{D}O_2$), defined as the product of cardiac index and arterial oxygen content, thus reflecting tissue perfusion. Recent publications examining limb $PtcO_2$ in ASPVD have found trends toward hypoxia[3,8] but as yet, wide variability and overlapping of the values seen in health and disease have limited clinical applicability. The authors suspected, however, that much of this variability might be due to variations in systemic $\dot{D}O_2$, and therefore designed a study to compare $PtcO_2$ on the extremity to $PtcO_2$ on the trunk, which is presumably a normally perfused location. The ratio of limb to trunk $PtcO_2$, or regional perfusion index (RPI) should therefore be independent of variations in O_2 delivery and reflective of local limb oxygen supply and demand relationships. Limb oxygenation was studied in claudication by measurement of RPI during exercise testing in normal subjects and patients with claudication.

This study reports the effects of position change, exercise, and recovery from exercise upon regional oxy-

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genation in patients with and without symptomatic ASPVD.

Materials and Methods

Subject Selection

Forty-seven limbs were studies in 24 patients, 12 of whom were asymptomatic and 12 of whom had a clinical diagnosis of intermittent claudication. Group 1 was formed by the 12 limbs studied in healthy, nonsmoking individuals between 25 and 35 years old. Group 2 was formed by 12 limbs studied in subjects between the ages of 55 and 65 who had no known lower extremity vascular disease. Half of these latter studies, group 2A, were performed in nonsmoking subjects with no known atherosclerotic disease. The other half, group 2B, were in subjects who were smokers and who had known coronary or cerebrovascular disease, but who had no symptoms, signs, or Doppler pressure evidence of lower extremity ASPVD. Group 3 was formed by 23 limbs in 12 patients, all of whom carried a clinical diagnosis of intermittent claudication and who had been admitted to Harbor–UCLA Medical Center for angiographic study. Their limbs were further divided into two groups; group 3A were the ten "asymptomatic" legs of claudicators and group 3B were the 13 limbs that were primary sites of the patients' chief complaints. The average age of patients with claudication was 61 ± 8 (SD) years. Ten patients were men; two were women. All were smokers and five had diabetes. Three patients had had prior vascular reconstructive operations; one had a femoral-popliteal bypass; a second had had a carotid endarterectomy; a third presented with recurrent claudication due to thrombosis of the axillary limb of a prior axillobifemoral bypass graft. All patients but two underwent angiography. In these two patients, angiography was not performed because their level of functional impairment as measured by treadmill testing did not warrant operative intervention.

Protocol

Transcutaneous oxygen sensors (Novametrics Inc., Wallingford, CT, and Kontron, Roche Inc., Everett, MA) were placed on the chest in the subclavicular position, medial distal thigh, medial mid-calf, and dorsum of the foot. Areas selected were not directly over any veins and, if possible, were on a slightly convex area so as to improve adhesion. The sites were shaved and then cleaned with an alcohol sponge. Electrodes were affixed using the double-sided adhesive rings and contact gel supplied by the manufacturer. Minimal amounts of contact gel were used since the use of more results in a higher frequency of sensor contact loss. Leads were taped to the leg 2 inches above the sensor and then led away from the subject through a belt worn at the waist. No other adhesive measures were necessary.

Electrodes were allowed to equilibrate with the subject in the supine position until stable values were achieved. This usually took from five to ten minutes. Tests were done in a room controlled at 72°, but equilibration times were nonetheless considerably shorter when a light blanket was placed over the subject. After equilibration, the simultaneous $PtcO_2$ values in the supine and standing positions were recorded.

The subject then started walking on a standard treadmill. Chest, thigh, calf and foot $PtcO_2$ values were recorded simultaneously during the exercise period and for five minutes thereafter. Asymptomatic subjects were walked at 2.5 mph on a 10% grade for ten minutes. Patients with intermittent claudication underwent a standard Bruce[9] low level treadmill protocol. An attempt was made to have all patients walk at 1.5 mph on a 4% grade. This level of exercise constitutes 4 metabolic equivalents or METs. In occasional patients who could not walk at 1.5 mph, the treadmill was slowed and the grade increased to maintain the 4 MET level of work. When typical symptoms occurred, the duration of exercise and the simultaneous $PtcO_2$ values were recorded. Subsequent measurements were made when the patient could walk no further. The $PtcO_2$ values were recorded and the treadmill was then stopped. Subsequent measurements were made for five minutes with the patient recovering in the standing position.

Data Analysis

The data for each group in each phase of the study were pooled. Intergroup variations were tested for significance by an unpaired Student's t-test. Intragroup variations between phases were analyzed using a paired t-test as appropriate.

Results

Asymptomatic Subjects

Chest $PtcO_2$. Chest $PtcO_2$ values in group 1 (youthful subjects) were stable throughout changes in position and activity (Table 1). Group 2 (older) subjects had significantly lower chest $PtcO_2$ values than group 1 in all positions and during exercise. In addition, group 2 chest $PtcO_2$ values were decreased ($p < 0.01$) in the supine position and rose with exercise ($p < 0.01$) when compared with those in the standing position.

Comparing Groups 2A (without atherosclerotic disease) and 2B (with atherosclerotic disease), group 2B had a significantly lower chest $PtcO_2$ values in the supine and standing positions. However, group 2B demon-

TABLE 1. *$PtcO_2$ in Normal Subjects*

Sensor Position	Subject Group	Supine	Standing	Maximal Exercise	Minutes After Exercise 1	2	3	4	5
Chest	1	78.8 ± 2.5	80.6 ± 1.7	80.8 ± 3.1	82.3 ± 2.9	84.0 ± 2.9	83.6 ± 2.9	82.8 ± 2.6	81.8 ± 2.7
	2	63.5 ± 2.7*	67.9 ± 1.8*	74.5 ± 2.0	75.8 ± 2.0	78.4 ± 2.6	78.0 ± 2.7	77.1 ± 2.8	76.0 ± 2.7
Thigh	1	73.8 ± 3.2	82.4 ± 2.5	81.5 ± 2.5	82.6 ± 2.4	83.8 ± 2.3	83.8 ± 2.3	83.3 ± 2.4	82.8 ± 2.4
	2	64.3 ± 1.9*	74.2 ± 1.3	79.8 ± 1.2	80.0 ± 1.4	82.1 ± 1.7	82.2 ± 1.8	81.3 ± 1.8	80.6 ± 1.8
Calf	1	70.0 ± 3.5	84.3 ± 3.1	84.3 ± 3.4	84.4 ± 3.1	86.1 ± 2.9	85.8 ± 3.2	85.3 ± 3.2	84.7 ± 3.2
	2	56.4 ± 2.8*	70.9 ± 1.8	78.2 ± 1.6	77.9 ± 1.8	79.7 ± 2.4	79.8 ± 2.6	79.1 ± 2.6	78.0 ± 2.7
Foot	1	69.8 ± 1.5	79.9 ± 1.7	72.3 ± 1.8	77.1 ± 1.2	79.6 ± 0.8	79.4 ± 1.3	81.3 ± 1.1	79.7 ± 1.0
	2	58.9 ± 3.0*	72.7 ± 1.5	73.6 ± 2.6	77.8 ± 2.1	79.8 ± 2.5	79.5 ± 2.6	79.3 ± 2.5	79.2 ± 2.5

* $p < 0.01$ by unpaired t-test for the difference between groups 1 and 2.

Variation of raw $PtcO_2$ in normal subjects with position, exercise and rest. Values are recorded simultaneously at all four levels. Group 1—age 20–35, N = 12. Group 2—age 55–65, N = 12. Results are in torr expressed as mean ± SEM.

strated greater increases in chest $PtcO_2$ during exercise, and thus group 2B during exercise was not significantly different from that of group 2A (Table 2).

Lower extremity $PtcO_2$. Upon standing, all limbs in all groups demonstrated increased $PtcO_2$ at all levels of the leg compared with supine values. Thigh and calf $PtcO_2$ in group 1 increased during exercise. In contrast, foot $PtcO_2$ significantly decreased during exercise in group 1. These values recovered rapidly at the end of exercise (Table 1). Group 2 had similar patterns of response to position and exercise at the thigh and calf, but consistently had lower $PtcO_2$ values than did group 1.

Standing thigh and foot $PtcO_2$ in groups 2A and 2B were similar. Group 2B had significantly lower standing calf values, as well as a significantly lower $PtcO_2$ throughout the leg in the supine position.

Transcutaneous regional perfusion index (RPI). Despite the observed differences in chest and limb $PtcO_2$, the ratio of limb to chest $PtcO_2$, or RPI is strikingly similar in all subjects. At all three locations RPI was 8–12% less in supine than in standing position ($p < 0.01$) (Fig. 1).

During exercise and recovery, group 1 thigh and calf RPI values were stable. Group 2, on the other hand, manifested decreases in RPI during exercise that, although small, were highly consistent from subject to subject and thus significant by paired t-test. The maximal decrease in calf RPI in group 2 was seen somewhat after the exercise was over.

Foot RPI fell significantly with exercise both in group 1 and group 2. In each case the decrease was about 10% ($p < 0.01$ by paired t-test). RPI rapidly returned to baseline in both groups; however, there was a suggestion that this occurred faster in group 1.

Although trends in RPI were remarkably similar in group 1 and group 2, values were somewhat higher in group 2 at the foot and thigh levels. These differences

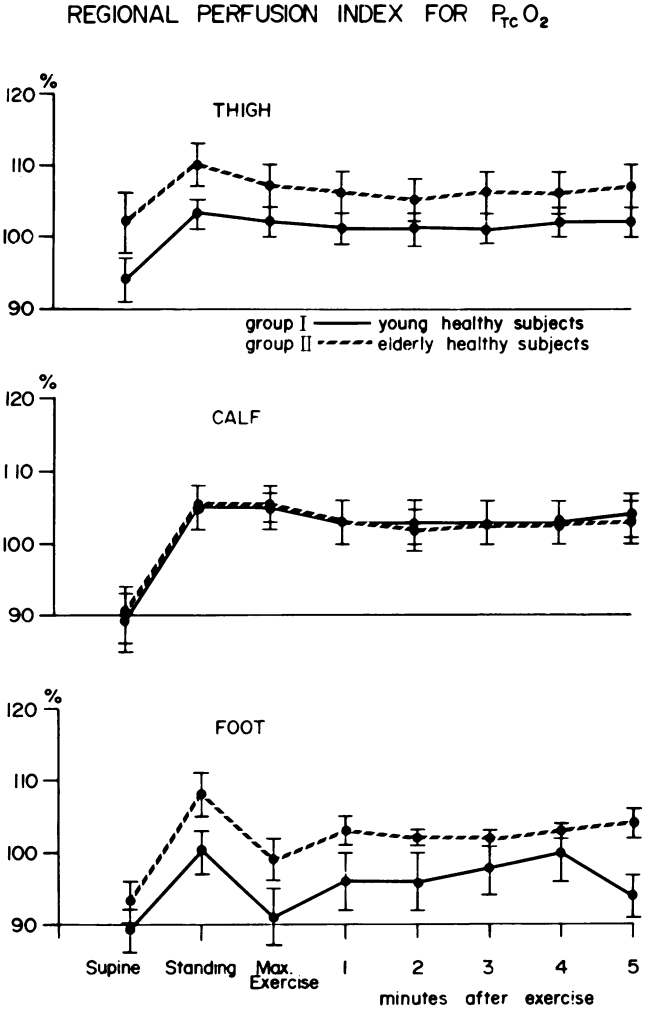

FIG. 1. Regional Perfusion Index in the thigh, calf and foot as determined by $PtcO_2$ relative to chest values. Values are mean ± SEM. Note the statistically significant difference in regional perfusion index values from supine to standing in thigh, calf and foot for both group I subjects (solid lines) and group II patients (dash lines) ($p < 0.01$ using the paired t-test).

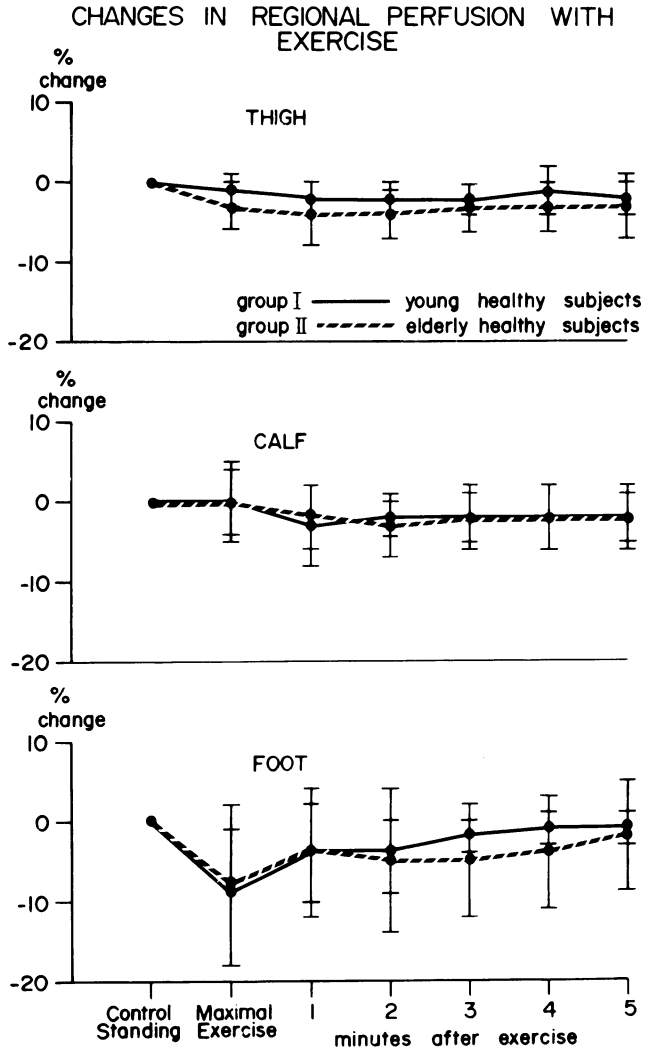

FIG. 2. Changes in RPI expressed as a per cent of the standing control values during exercise and recovery from exercise. Note that in this diagram, data are presented as mean ± SD. Solid lines represent group 1 subjects, and dashed lines represent group 2 patients.

did not, however, achieve statistical significance and were related mostly to the differences in chest $PtcO_2$.

Trend analysis of RPI. RPI trends during exercise, recovery from exercise, and positional change were examined using the standing RPI as a baseline. This revealed remarkable stability of local RPI throughout exercise and recovery at the thigh and calf levels (Fig. 2). Consistent small decreases in RPI were noted with exercise. Although exercise decreases were slightly greater in group 2, no significant differences between groups 1 and 2 or 2A and 2B were found.

Exercise induced significant changes in foot RPI in both groups 1 and 2; however, no significant differences between the changes in groups 1 and 2 were found (Fig. 2). All values returned to baseline within one minute after the termination of exercise.

Positional variations in RPI were no different in groups 1 and 2 at any limb level (Fig. 3). Foot RPI decreased more in group 2B than group 2A when subjects assumed the supine position (Fig. 3).

Patients with Claudication

Treadmill exercise testing. Treadmill energy expenditure needed to produce claudication was quantified by multiplying the achieved MET level by the exercise time in minutes. All asymptomatic subjects (groups 1, 2A, and 2B) were easily able to maintain 7.5 METs for ten minutes or 75 arbitrary treadmill work units. All claudicators achieved a 4 MET level of energy expenditure, although some required manipulation of treadmill speed and grade to achieve it. No patient with claudication was able to maintain the 4 MET level for ten minutes. Mean time to the onset of symptoms was 3.8 ± 1.3 (SD) minutes (165 yards at 1.5 mph). Mean time to inability to walk was 4.8 ± 0.7 (SD) minutes (210 yards at 1.5 mph). Mean total energy expenditure in claudicators was 19.2 ± 2.5 (SD) treadmill work units.

$PtcO_2$ measurements. Chest $PtcO_2$ values (Fig. 4) in claudicators (group 3) were indistinguishable from those in the elderly control group with systemic atherosclerotic disease (group 2B) (Table 2). However, group 3 chest $PtcO_2$ values were significantly lower at rest than those seen either in young (group 1) or elderly (group 2A) controls without atherosclerotic disease. These relation-

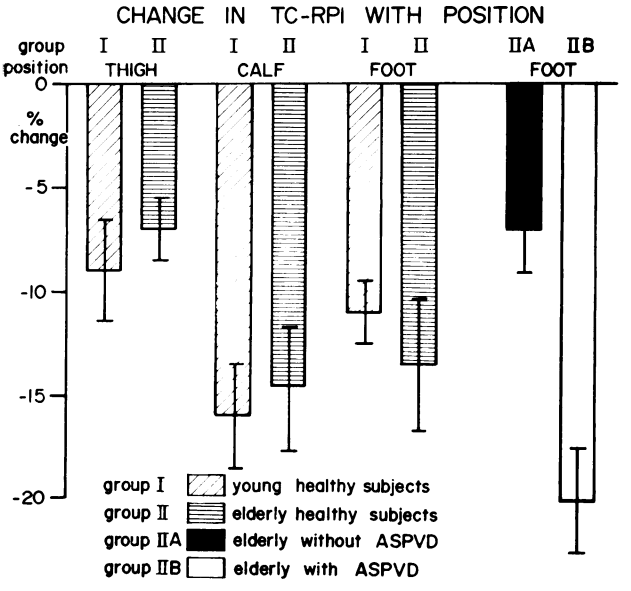

FIG. 3. Change in RPI from the standing to supine position. Bars represent mean ± SEM. Group 2B demonstrated a greater ($p < 0.01$, unpaired t-test) decrease at the foot than group 2A. Differences between groups 2A and 2B were not significant at the thigh or calf. AS signifies systemic atherosclerosis.

TABLE 2. *$PtcO_2$ in Normal Subjects Aged 55–65 Years*

Location	Group	Supine	Standing	Maximal Exercise
Chest	2A	68.3 ± 3.0	71.2 ± 2.3	76.5 ± 2.2
	2B	58.7 ± 3.6*	64.6 ± 2.2*	73.8 ± 3.2
Thigh	2A	68.0 ± 2.6	76.0 ± 1.9	79.8 ± 2.1
	2B	60.2 ± 1.7*	72.0 ± 1.6	79.8 ± 1.3
Calf	2A	62.0 ± 4.5	76.3 ± 1.2	82.2 ± 1.8
	2B	51.0 ± 2.9*	65.5 ± 1.2†	75.8 ± 1.9†
Foot	2A	66.8 ± 2.7	75.0 ± 2.5	78.2 ± 3.4
	2B	51.0 ± 2.5†	70.3 ± 1.1	69.0 ± 3.3*

Values are recorded simultaneously at all four levels. Group 2A—subjects with no history of smoking or atherosclerotic disease. Group 2B—subjects with prior history of systemic atherosclerotic disease but without symptoms of peripheral vascular disease. Results are in torr expressed as mean ± SEM. * $p < 0.05$. † $p < 0.01$. (unpaired t-test).

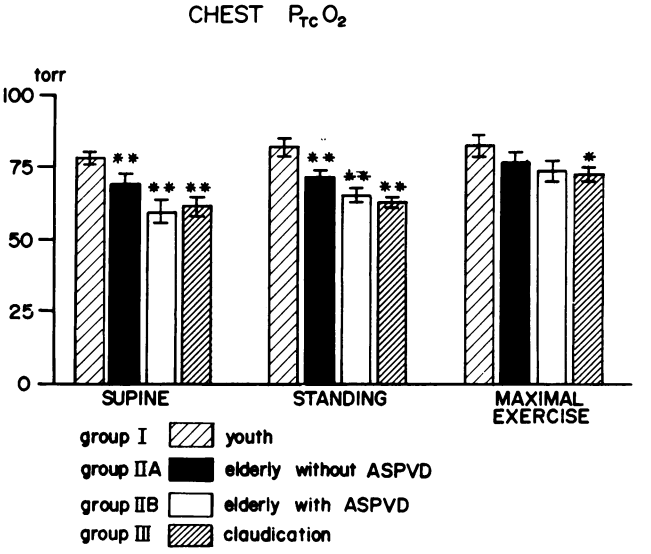

FIG. 4. Chest $PtcO_2$ in patients with intermittent claudication (group 3) is compared with young healthy subjects (group 1) and elderly subjects without (group 2A) and with (group 2B) systemic atherosclerotic disease. Results are shown as mean ± SEM. * = $p < 0.05$; ** = $p < 0.01$ for the differences of groups 2A, 2B, and 3 from group 1.

ships changed with exercise; the chest $PtcO_2$ value rose during exercise in patients with claudication. At maximal exercise tolerance, it was indistinguishable from chest $PtcO_2$ values of groups 2A and 2B as well as being much closer to the chest $PtcO_2$ in group 1 (Fig. 4). Thigh, calf, and foot $PtcO_2$ in claudicators (Table 3) were always significantly lower than those seen in young normals. When compared with group 2, however, claudicators only manifested significant differences during exercise or in foot $PtcO_2$ measurements taken in the supine position. Although differences between the groups were highly significant, the $PtcO_2$ of exercising claudicators occasionally still fell within two standard deviations of the mean value seen in group 2 (Table 3).

Transcutaneous regional perfusion index (RPI). Exercise decreased RPI in all legs of patients with claudication at all three levels. Decreases in RPI from standing controls averaged 18.2 ± 3.1 (SE) % at the thigh; 20.7 ± 4.0 (SE) % at the calf; and 19.5 ± 2.9 (SE) % at the foot (Figs. 5, 6). These changes were highly significant when compared with the responses of groups 1 and 2. Standard deviations were moderate and only rarely overlapped with values observed in nonclaudicators. Further analysis, however, revealed a biphasic response of RPI to exercise: symptomatic legs had markedly greater reductions than did asymptomatic legs; −27.6 ± 3.9 (SE) *vs.* −7.1 ± 1.5 (SE) % at the thigh. Similar differences in the RPI response to exercise were observed at the calf and foot (Fig. 5). Exercise-induced decreases in RPI in group 3A limbs were still significantly greater than those seen in groups 1 or 2 but manifested considerable overlapping of values. By contrast, group 3B (claudicating) limbs manifested changes that revealed no overlap with the values seen in any normal group. The decreases in thigh and calf RPI observed in claudicating limbs during exercise always exceeded mean normal variability by at least 3 SD (Fig. 5). Because the RPI changes observed in normal feet during exercise are larger, the discriminatory value of changes in foot RPI is not as good. Nevertheless, decreases in foot RPI in all claudicating limbs exceeded the variability of normal limbs by at least one standard deviation (Fig. 5).

Positional changes in RPI in patients with claudication. Group 3 RPI values were lower supine than standing at all levels. These differences, however, were no greater than those seen in any of the control groups (Table 4). There appeared to be a trend towards greater

TABLE 3. *Statistical Evaluation of leg $PtcO_2$ in Patients with and without Claudication*

		Supine	Standing	Maximal Exercise
Thigh	Group 1	73.8 ± 10.9*	82.4 ± 8.7	81.5 ± 8.8
	Group 2	64.3 ± 6.7	74.2 ± 4.6	79.8 ± 4.1
	Group 3	57.3 ± 8.9	66.2 ± 8.9	61.6 ± 13.9
Calf	Group 1	70.0 ± 12.1	84.3 ± 10.8	84.3 ± 11.8
	Group 2	56.4 ± 9.7	70.9 ± 6.3	78.2 ± 5.4
	Group 3	48.7 ± 9.3	61.7 ± 10.2	56.3 ± 13.8
Foot	Group 1	69.8 ± 5.3	79.9 ± 6.0	72.3 ± 6.2
	Group 2	58.9 ± 10.3	72.7 ± 5.1	73.6 ± 9.1
	Group 3	45.6 ± 12.4	61.3 ± 12.5	53.4 ± 14.5

* Mean ± 1 SD.

Group 3 patients with claudication have significantly lower $PtcO_2$ at all phases of the study, but in most instances standard deviations overlap with $PtcO_2$ values that would be considered normal or normal for age.

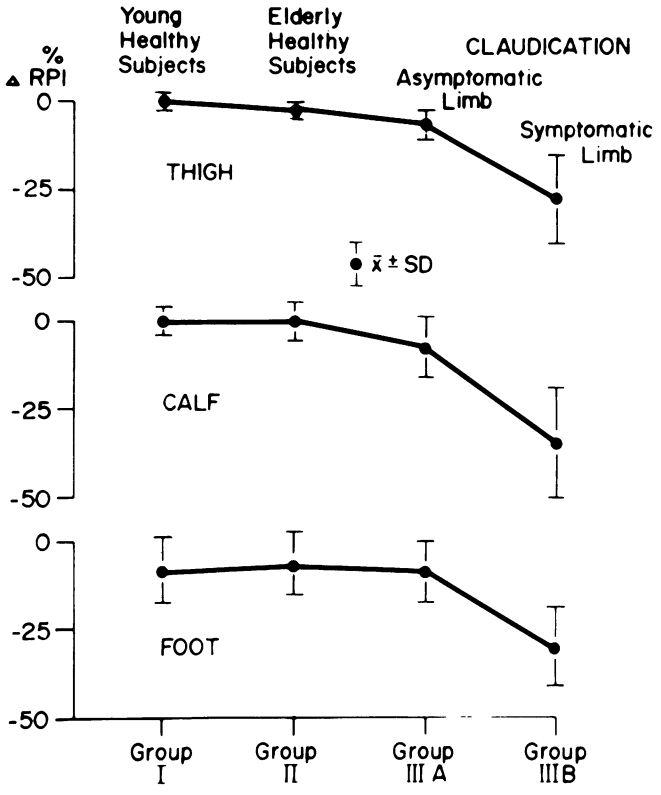

FIG. 5. Changes in RPI at the thigh, calf and foot level of young normals (group 1), subjects aged 55–65 years (group 2), the asymptomatic (group 3A) and symptomatic legs (group 3B) of patients with claudication when exercised. Values are depicted as mean ± SD.

differences between standing and supine RPI in diabetic patients; the greatest positional dependence was noted in the two limbs with ischemic skin lesions. The only significant positional difference between claudicators and young subjects was the greater decrease in supine foot RPI in claudicators. This change, however, was indistinguishable from that seen in group 2B subjects.

Perfusion of claudicating limbs subsequent to exercise. The time course of changes in RPI found in claudicating (group 3B) limbs is presented in Figure 6. RPI decreases are seen to be both marked and prolonged. At the thigh, three minutes of rest were required to recover half of the RPI decrease at maximal exercise. At the calf and foot, four minutes were required. Generally, no comparison of recovery time with a normal group was possible, since these groups did not manifest decreases in RPI during exercise. The decreases in foot RPI elicited by exercise in subjects without claudication always resolved within one minute.

It also has been noted that occasionally, maximal changes in RPI induced by exercise occur shortly after the end of the exercise period. In such cases, recovery of one half of the RPI decrease did not occur within the five-minute measurement period.

TABLE 4. *Positional Changes in RPI in Group 1 (young healthy subjects), Group 2 (elderly healthy subjects) and Group 3 (patients with claudication)*

	Group	Standing	Supine	% ΔRPI
Thigh	1	102.9 ± 2.3	93.5 ± 2.7	−9.0 ± 2.5
	2	110.0 ± 3.1	102.3 ± 3.7	−7.2 ± 1.5
	3	106.4 ± 2.5	94.8 ± 3.0	−10.9 ± 1.8
Calf	1	105.2 ± 2.8	88.7 ± 3.6	−15.8 ± 2.1
	2	104.9 ± 2.7	89.9 ± 4.4	−14.6 ± 2.8
	3	100.1 ± 4.5	81.8 ± 4.6	−18.8 ± 2.0
Foot	1	100.1 ± 3.3	89.0 ± 2.6	−10.8 ± 1.4
	2	107.6 ± 2.6	92.5 ± 1.9	−13.5 ± 2.4
	3	98.7 ± 4.3	75.7 ± 5.5	−24.8 ± 3.5*

Values are mean ± SEM. The right-hand column (% ΔRPI) is the per cent change from standing to supine in each group. Only the change at the foot in Group 3 was significantly (* = $p < 0.01$) different from the change noted in controls (Group 1).

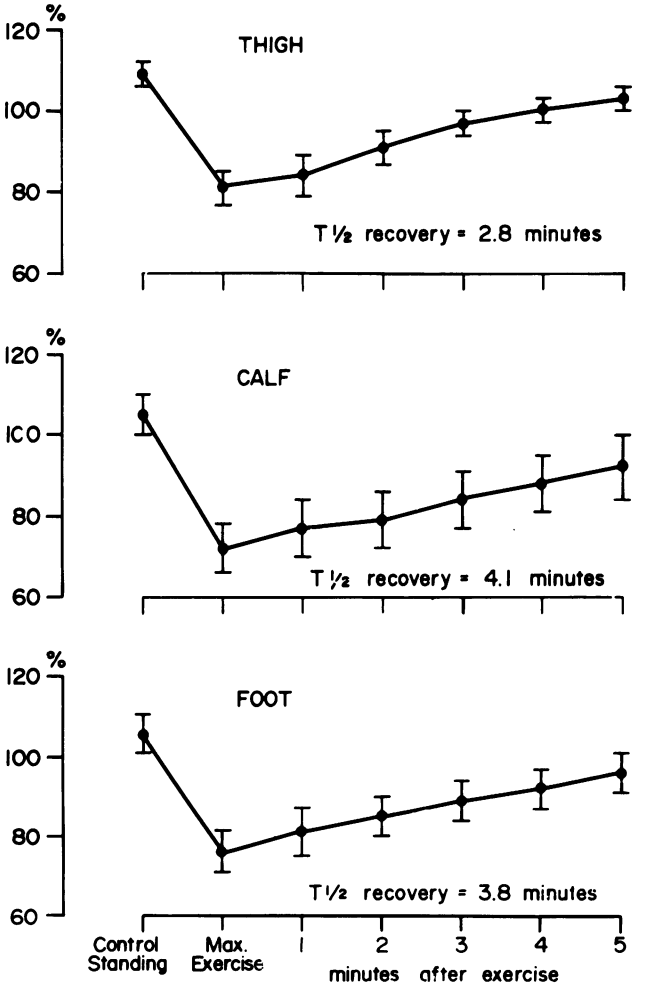

FIG. 6. Effect of exercise on RPI in claudicating limbs. Values are mean ± SEM. The $T_{1/2}$ recovery is the time required to recover half of the decrease in RPI caused by exercise.

Discussion

Changes in PaO_2 and cardiac output affect limb $PtcO_2$. Provocative tests of limb circulatory reserve, such as position change and exercise, also affect systemic cardiopulmonary function. The data clearly demonstrate that variability of systemic $\dot{D}O_2$ as well as of individual sensor locales with respect to capillary density, skin thickness, and temperature diminish the reliability of $PtcO_2$ in assessing ASPVD. Use of the RPI obviates the effects of cardiopulmonary function upon local $PtcO_2$, resulting in an accurate and usable index of local limb oxygenation. Provocative testing with exercise or position change obviates variability based on local factors.

Extreme stability of RPI indicates that local perfusion is well compensated during exercise in all asymptomatic limbs (Fig. 5). Oxygen supply exceeds demand in the vicinity of the sensor and therefore $PtcO_2$ is primarily a function of PaO_2. In claudicating limbs, oxygen supply to tissue in the vicinity of the sensor is decreased relative to oxygen demand during exercise. Tissue oxygen stores are small and so tissue PO_2 and $PtcO_2$ fall precipitously when local flow fails to increase in response to increased oxygen consumption.

Because $PtcO_2$ is mostly a function of PaO_2 until flow becomes critically limited,[10] even the nonsymptomatic legs of claudicators behave more like normal limbs than like claudicating limbs (Fig. 5). Thus, the specificity of large RPI decreases for claudicating limbs and the stability of RPI in normal limbs during exercise make this test unambiguous. The decreases in RPI in the contralateral limbs of claudicators were significant, but even though these limbs clearly must have had significant arteriosclerotic disease, the decreases in RPI were not nearly as large as those in claudicating limbs.

In essence, it was found that a markedly decreased RPI at the thigh or calf with exercise is a specific physiologic correlate of claudication. The data suggest preliminarily that a decrease in RPI must be 10% at the thigh or 15% at the calf using the described exercise protocol in order to be highly specific for claudication. The establishment of false-positive and false-negative rates at each level of change and location will require larger studies with angiographic confirmation.

Small changes may also indicate significant atherosclerosis, as was the case in the asymptomatic limbs of claudicators or the limbs of elderly controls who had systemic atherosclerosis. Frequently, these limbs will manifest mild discomfort or atypical symptoms during the protocol. Further experience with RPI measurement will determine to what extent small RPI change or the time course of their resolution may be used to diagnose such preclinical states.

The present experience with transcutaneous RPI measurement indicates that claudication can be objectively and accurately assessed both as to the patient's exercise tolerance and the degree of ischemia incurred during exercise. The test is easy to perform, reproducible, noninvasive, highly specific, and directly reflective of the underlying vascular pathophysiology. The authors feel, for these reasons, that it should be immediately applicable to the diagnosis and management of peripheral vascular disease. Furthermore, the ability to quantitate the previously subjective phenomenon of claudication in terms of limb RPI will make it possible to document the progress of such patients. This approach may refine the criteria for operative intervention in claudication, avoiding or delaying high risk procedures in stable patients and indicating prompt intervention in patients with deteriorating perfusion prior to the onset of life- and limb-threatening sequelae of ischemia.

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