CLINICAL RESEARCH

Transcutaneous oxygen tension during exercise in patients with claudication

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Abstract

Transcutaneous oxygen tension ($TcPo_2$) was monitored during maximal exercise in 10 patients with stable moderate to severe claudication. The $TcPo_2$ fell by 16% at the onset of claudication and 32% at the maximum walking distance. On resting this decrease reached a maximum of 66% roughly four minutes after exercise. This was followed by a steady recovery. The percentage changes were reproducible in each patient and were appreciably different from the $TcPo_2$ exercise profiles of normal healthy volunteers.

$TcPo_2$ monitoring during exercise is a simple, reproducible, cheap, and useful technique for assessing claudication and compares favourably with other techniques used to quantify this condition.

Introduction

As quantitative assessment of intermittent claudication is complicated, unreliable, and expensive it is difficult to assess objectively the clinical course of claudication or the effects of any treatment. The variety of techniques suggests their individual shortcomings. Invasive methods include the use of muscle pH and oxygen pressure electrodes and electromagnetic flowmeters, Doppler velocimetry, dilution techniques, and isotope clearance studies. Non-invasive methods include venous occlusion or impedance plethysmography and duplex Doppler scanning. In addition the arterial lumen may be visualised by angiography or digital subtraction angiography and the limb temperature, venous blood oxygenation, and foot systolic blood pressure measured¹⁻⁴; only the last of these methods has gained any widespread clinical acceptance.

Doppler systolic ankle pressures indexed against brachial blood pressure provide a quantitative assessment but are associated with some important problems. Arteries may be difficult to find and their walls may be calcified and incompressible. At rest there may be little arterial pressure gradient until stenosis reaches 70-80%,⁵ whereas minimal stenoses may cause claudication. As it is impossible to use a Doppler flowmeter while walking, pressures after exercise are sometimes measured instead.⁶ Although such measurements show characteristic changes, in practice it is difficult to measure brachial and ankle pressures simultaneously and as soon as the patient has stopped exercising. Even a few seconds' difference in the delay before the first reading may result in considerable inaccuracies.

We therefore studied the use of the transcutaneous oxygen monitor, originally devised for neonatal monitoring,⁷ in assessing patients with claudication. Although other groups have carried out similar studies in patients at rest,⁸ the use of the transcutaneous oxygen monitor during exercise seems more relevant in the assessment of intermittent claudication.

Patients and methods

In the study we used a Hewlett Packard model 78850A oxygen monitor. The transcutaneous oxygen tension ($TcPo_2$) probe is a modified Clark electrode: oxygen is reduced at the platinum cathode to produce a current proportional to the partial pressure of oxygen at the electrode surface.^{9,10} The probe temperature is thermostatically controlled at 45°C to produce maximal arterialisatation of the skin circulation and diffusion across it.¹¹ To use the apparatus the probe is attached to a double sided adhesive ring and the recording started. It is unobtrusive, causes no discomfort to the patient, and produces a clear, easy to read trace on graded paper both during exercise and at rest.

Measurements were taken in 10 patients with claudication (mean age 67 (range 52-72) years) whose symptoms had not changed for at least six months. All had a thigh to arm pressure index of less than 0.75 measured by strain gauge plethysmography. None could walk at a treadmill speed of 4.5 km/h and an incline of 10% for more than six minutes. Each patient attended the hospital twice, with a one month interval, to test the reproducibility of $TcPo_2$ measurements.

Recordings were made on the chest and 10 cm below the knee joint, over the tibialis anterior muscle (a position used by other workers^{12,13}). Measurements were made with the patient lying supine, sitting with the leg dependent, and standing (all readings being taken after the trace had

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stabilised). Patients were then exercised on a treadmill at an incline of 10% at speed individually adapted to their maximum walking distance (eight patients at 4.5 km/h and two at 3.0 km/h). They were then rested sitting until the pain had resolved and the TcPo₂ had once again reached a steady state. Control measurements were taken similarly from 10 volunteers matched for age (mean age 68 (range 53-81) years) with no symptoms, signs, or history of arterial disease.¹⁴

Reproducibility of the results was assessed by the correlation of corresponding values about a line of identity. Comparisons within the groups were carried out between points using the Wilcoxon matched pairs single rank test (significance at $p < 0.01$). Analysis between the groups at appropriate points was carried out using the Mann-Whitney U test (significance at $p < 0.01$).

Results

Both the control group and the group of patients with claudication produced characteristic exercise TcPo₂ profiles (fig 1). There was no difference between the resting values of either group, but the difference in the percentage change in TcPo₂ with exercise was highly significant (table I), giving a much better separation between the two groups (fig 2).

In controls (fig 3) the mean (SEM) sitting TcPo₂ increased on standing from 72 (2) mm Hg to 75 (2) mm Hg. At the start of walking there was a brief fall in the mean (SEM) TcPo₂ of 2 (3)% occurring after the first minute, which recovered rapidly. After exercise there was a rise in TcPo₂ to 84 (3) mm Hg before a return to resting levels. Control subjects experienced no pain or discomfort during exercise.

In the patients with claudication (fig 4) there was no significant difference between chest and leg TcPo₂ with the patient prone. On sitting, however, there was a rise from 59 (2) mm Hg to 71 (2) mm Hg, but there was no further

increase on standing. During exercise the TcPo₂ fell by 16 (4)% before the onset of pain. This fall was maintained to the point of maximum exercise, by which time the TcPo₂ had fallen by 32 (7)%. On resting the fall continued, reaching a minimum value with a total drop of 66 (6)% 3.5-4.5 minutes after exercise. This was followed by a steady recovery (with loss of pain 43 (9)% below resting levels) to levels slightly above those found before exercise 11-13 minutes after exercise.

FIG 1—TcPo₂ profile for controls and two types of profile for patients with claudication.

TABLE I—Comparison between control and claudicant groups

TcPo ₂ measurement	U	z Value	p Value
Standing	33	-1.28	NS
Sitting	43.5	-0.49	NS
After exercise	3	-3.55	0.0004
Minimum	0	-3.78	0.0002
Change at end of exercise (%)	2	-3.63	0.0003

FIG 2—Comparison of TcPo₂ values in controls (○) and patients with claudication (●). Percentage change in TcPo₂ with exercise gave a much better separation between the two groups than resting TcPo₂.

TABLE II—Reproducibility of TcPo₂ values

TcPo ₂ measurement	Correlation	t Value	p Value
Chest	0.19	0.545	>0.20
Lying	0.16	0.467	>0.20
Sitting	-0.13	0.360	>0.20
Standing	0.02	0.065	>0.20
Change at pain onset (%)	0.86	4.745	<0.01
Change at end of exercise (%)	0.79	3.609	<0.01
Minimum	0.75	3.164	<0.02

The reproducibility of resting values in patients with claudication was poor. There was, however, good correlation between monthly readings of percentage changes in TcPo₂ during exercise, and even the greatest change in TcPo₂ correlated significantly at 0.75 (table II).

The traces for patients with claudication appeared to be of different types (fig 1). In one type the TcPo₂ fell at the start of walking and continued to fall steadily throughout exercise, reaching a minimum after exercise. At the other extreme the TcPo₂ fell at the start of exercise but then flattened out to give a plateau until the end of exercise. On rest it again fell to give a minimum after exercise followed by recovery.

Discussion

The main objection against the use of the transcutaneous oxygen monitor in occlusive arterial disease has been that it is affected by

FIG 3—Mean (SEM) walking TcPO₂ values for controls on time point scale. Transient changes are seen with exercise.

FIG 4—Mean (SEM) walking TcPO₂ values for patients with claudication on a time points scale. There is a highly significant fall during exercise, which is maintained on resting giving a minimum after exercise.

too many variables.¹⁵ The TcPO₂ value depends principally on the skin blood flow and the oxygen saturation of the blood.¹⁶ During walking there is a diversion of available blood flow to exercising muscles sufficient to cause a fall in the ankle systolic pressures in patients with restricted inflow.¹⁷ In normal subjects the intramuscular oxygen pressure rises with exercise whereas in patients with peripheral vascular disease it falls.¹⁸ This steal of flow and

increased extraction of oxygen will result in a reduced oxygen delivery to the skin and therefore a drop in TcPO₂.

In this study resting values and postural changes in TcPO₂, both in controls and patients with claudication, agree with published data.¹³ The drop in TcPO₂ on exercise in the control group was probably caused by a brief diversion of flow that was quickly compensated by autoregulatory mechanisms. After exercise the

increased flow persisted for a short length of time giving a temporary rise in $TcPo_2$. In the group of patients with claudication the same process began at the start of walking. The increase in flow, however, was insufficient to compensate for the increased oxygen extraction, and there was a steal of flow from skin to muscle resulting in a persistent fall in $TcPo_2$.

Considering the pattern of change in the patients with claudication, there appeared to be a spectrum between the two types of trace shown in figure 1. The first type of response may represent the initial fall of the second in patients unable to walk long enough for a plateau to occur. (Indeed, in one patient whose claudication distance subsequently improved dramatically the shape of the trace changed from the first to second type.) In the second type of trace an equilibrium seemed to be reached. The blood flow may be limited by arterial compression within the exercising muscle, which is relieved on rest to allow maximum steal.

The total duration of plethysmographic hyperaemia after occlusion, even in patients with severe claudication, is roughly three to four minutes,²⁰ but the time to recovery of $TcPo_2$ after exercise is more than 10 minutes. This suggests that although the total segmental limb flow returns to resting values quite quickly, the flow distribution and oxygen extraction remain abnormal for much longer. Hence $TcPo_2$ measurements seem to give a better indication of the true duration of the pathophysiological abnormality.

There was no direct or reproducible relation between $TcPo_2$ and the onset and loss of claudication pain. It was apparent, however, that the patients with the worst performance at exercise, and therefore presumably the severest disease, showed the greatest percentage drop in $TcPo_2$ and the longest recovery time.

The monitoring of transcutaneous oxygen at rest has not been a useful assessment of arterial disease, and the overlap between values for controls and those for patients with claudication has restricted its use to the consideration of amputation sites.²⁰ The use of $TcPo_2$ monitoring during exercise, as described in this study, improves reproducibility and gives a good separation between normal and abnormal responses. Exercise $TcPo_2$ is a relevant, reliable, and

useful test in patients with claudication, which, in addition to the cost, convenience, accuracy, and ease of operation of the equipment, makes it an attractive method of quantifying intermittent claudication.

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100 YEARS AGO

Dr Holmes has met with an exceptionally warm reception in this country. Seldom has any visitor to our shores received the homage of a more universally affectionate and respectful greeting from every class of society. There are few men in our generation who have ever received a more flattering welcome from all the noblest and best elements of English society. Since the visit of Longfellow, perhaps no other American has been received with such unanimous and affectionate enthusiasm. He has seen the best and most brilliant side of London society, and has made the personal acquaintance of all that England can boast that is most representative in rank, wealth, intellect, statesmanship, and humanitarian energy. Dr. Holmes has reached an age when common prudence compels a man to husband his resources, and to limit the tax upon his physical energies; and he has not been able, therefore, in the overwhelming calls upon his time, and the flattering offers of hospitality from all sides, to fulfil more than a tithe of the opportunities that were offered him of studying the institutions, and extending his acquaintance with all the classes of the country, in the limited time at his disposal. Seeking rest and recreation, and declining public receptions, he has, nevertheless, availed himself of various opportunities of identifying himself with the profession which he adorns, and which is proud to claim him among its members. He has been seen at more than one medical house, and has made the acquaintance of most of the leading medical men in London during his short stay here. On Wednesday last, after dining with Sir Henry Thompson, who had assembled to meet him a small party, including Mr. Gladstone, Mr. John Morley, Mr. Robert Browning, Sir James Hannen, Sir James Paget, Professor Masson, Mr. Alma Tadema, Mr. Du Maurier, Mr. Knowles, and Mr. Ernest Hart, Dr. Holmes attended the *soirée* at the Royal College of Surgeons, and paid his respects to the President, Mr. Savory. He took the opportunity of going through parts of the museum in which he was particularly interested, under the guidance of Sir James Paget. Dr. Holmes expressed not only his delight and high admiration at this magnificent museum, and the unparalleled scene it

presented, but was particularly interested, and especially admired, the complete typical osteological specimens arranged by Professor Flower for the purposes of anatomical study. Dr. Holmes, although advanced in years, retains all the vivacity and sympathetic flexibility of mind which are characteristic of the fullest vigour and freshness of mind. It is fifty years since he had visited London. He then made the acquaintance of Lawrence, Brodie, Tyrrell, MacMurdo, Abernethy, and some of their contemporaries. He makes this week a visit to Lord Tennyson, after which he will go to Oxford and Cambridge, and probably to Edinburgh, to receive the honorary degrees which those Universities propose to confer on him. In receiving Dr. Holmes with such unanimous enthusiasm, English society has done much to vindicate its claim to sound catholicity, and sincere appreciation of a most wholesome and penetrating genius. More than one Royal and illustrious visitor has been present in London during the season. None has received so warm, unforced, and universal a welcome as the "Autocrat of the Breakfast Table." (*British Medical Journal* 1886;i:1121.)

Her Majesty having signified to the Countess of Dufferin her intention of presenting medals to the most distinguished female licentiates in the medical schools of India, the central committee propose awarding them in the following manner. A gold medal will be offered for annual competition in each of the four Indian Universities, Bengal (Calcutta), Bombay, Madras, and Punjab (Lahore); students at the Agra Medical School being allowed to compete at Lahore. These medals will be called the "Queen-Empress Medals," and will only be awarded to candidates who attain a high standard of proficiency. His Excellency the Viceroy has also placed five silver medals at the disposal of the National Association; and the central committee propose offering them for competition among the female students of the hospital assistant class, one to each of the medical schools of Agra, Bombay, Calcutta, Lahore, and Madras. (*British Medical Journal* 1886;i:409.)