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Prevalence and Causes of Normal Exercise Oximetry in the Calf in Patients with Peripheral Artery Disease and Limiting Calf Claudication

I. Signolet [a], S. Henni [a], C. Colas-Ribas [a], M. Feuilloy [b,c], J. Picquet [d,e], P. Abraham [a,e,*]

[a] Department of Sports Medicine and Vascular Investigations, University Hospital, Angers, France
[b] Ecole supérieure d'électronique de l'Ouest, Institute of Science & Technology, Angers, France
[c] LAUM - UMR CNR6613, Angers, France
[d] Department of Vascular and Thoracic Surgery, University Hospital, Angers, France
[e] UMR INSERM 1083/CNRS 6214, Angers University, Angers, France

WHAT THIS PAPER ADDS

An ankle brachial index (ABI) of < 0.90 does not necessarily prove that claudication is of arterial origin. Transcutaneous oxygen pressure (Ex-tcpO_2) can provide evidence of exercise induced ischemia. Most patients with normal calf Ex-tcpO_2 despite ABI < 0.90 have non-calf ischemia or a non-vascular limitation. Treadmill testing is important in patients suffering from claudication, even with confirmed underlying arterial disease. Comorbidities should not be underestimated as co-factors in walking impairment.

Objective: In patients with claudication, an ankle brachial index (ABI) under 0.90 is considered to be abnormal and a sufficient argument for the arterial origin of exercise induced pain. Exercise transcutaneous oxygen pressure (Ex-tcpO_2) can provide evidence of exercise induced regional blood flow impairment (RBFI) and confirm the arterial origin of walking induced pain. The frequency with which calf Ex-tcpO_2 remains apparently normal in patients with claudication and abnormal ABI is unknown. Causes of these discrepant results have yet to be analysed.
Methods: A retrospective analysis of 4575 Ex-tcpO_2 tests performed on 3,281 patients was conducted. The focus was on patients with a history of calf claudication and ABI under 0.90. Duplicate or non-standard tests were excluded, as were patients with no pain or those able to walk more than 15 minutes (on a treadmill). Searches were conducted for possible explanations of normal calf Ex-tcpO_2 in the selected patients.
Results: Cardiorespiratory limitation was identified in 50 patients and isolated non-calf ischemia in 36 of the 106 patients selected. There was no obvious explanation during Ex-tcpO_2, but clinical improvement after non-vascular treatment or total absence of improvement after a technically successful revascularisation was noted in 12 patients. Four patients were lost on follow up. Four patients improved after revascularisation, which suggests that the Ex-tcpO_2 result was false negative.
Conclusions: Ex-tcpO_2 is negative in more than 20% of tests performed in patients with an ABI under 0.90 and a history of calf claudication. In most cases, when excluding re-tests and non-limiting or non-calf claudication on the treadmill, non-calf ischemia or a non-vascular limitation occurring during the test were observed. This observation supports both the value of treadmill testing in patients with calf claudication assumed to be of arterial origin (ABI$<$0.90) and the use of Ex-tcpO_2 to detect non-calf ischemia.
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INTRODUCTION

The ankle brachial index (ABI) at rest is commonly used to diagnose peripheral arterial disease (PAD). A low resting ABI (ABI$<$0.90) in patients with claudication is usually seen as proof of PAD[1] and by extension, of the vascular origin of walking induced pain and walking limitation.[2—4] Nevertheless, comorbidities may limit walking ability and occasionally induce lower limb pain, which calls into question the role of PAD as the sole cause of walking limitation, even in patients with a low ABI.

Transcutaneous oxygen pressure (tcpO_2) during exercise (Ex-tcpO_2) is useful in providing evidence of exercise induced regional blood flow impairment (RBFI) during exercise and confirming the arterial origin of walking induced pain.[5]

* Corresponding author. University Hospital, 49933 Angers, Cedex 09, France.
E-mail address: Piabraham@chu-angers.fr (P. Abraham).
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Given that $tcpO_2$ is different from ABI and can be measured throughout exercise and recovery, it is of interest to provide an objective argument for calf exercise induced ischemia underlying calf pain. Ex-$tcpO_2$ tests have been used in the study department from 2000 for all patients referred for claudication.[5,6] Ex-$tcpO_2$ is of interest given that it is insensitive to arterial stiffness, is highly accurate and reliable for measuring RBFI during exercise, and can help to better select patients requiring referral for radiological aterial imaging.[5,7] Ex-$tcpO_2$ is not a widely known technique, but it is currently used in university hospitals in France and abroad.[8]

It was hypothesised that even in patients with limiting calf claudication and a consistent abnormal ABI at rest, calf ischemia should be questioned as the sole cause of walking impairment. Patients with limiting calf claudication and ABI under 0.9, as well as seemingly normal Ex-$tcpO_2$, have not been studied previously. Whether such discrepancies are caused by a lack of sensitivity of Ex-$tcpO_2$ or by limitations other than calf ischemia remains an unanswered question.

METHODS

Study sample

A retrospective review was made of the database of all Ex-$tcpO_2$ tests performed in the study department since 2000. The study conformed to the principles of the Declaration of Helsinki.

Initial assessment

As per routine, all patients completed a standard questionnaire on symptoms of claudication (the San Diego claudication questionnaire until 2008, followed by the Edinburgh claudication questionnaire thereafter). All patients were also asked about previous vascular interventions for lower limb peripheral arterial disease. Gender, diabetes, smoking habits, and statin (or other cholesterol lowering drug) use were determined from patient hospital databases and with the use of standard questionnaires. Thereafter, trained vascular laboratory nurses conducted non-invasive vascular examinations, which included bilateral ankle and brachial systolic blood pressure measurement with the Doppler method.[9] For the present study, the ABI of each leg was calculated as the ratio of the highest ankle pressure to the highest arm pressure. The ABI for each subject was the higher ABI of the two legs. Finally, body mass, stature and walking speed over 10 m were measured.

Treadmill test

A standard procedure was used for all patients who could walk the 10 m of the laboratory corridor in less than 15 seconds. Other patients were excluded from the study. Standard treadmill tests were performed at a 10% gradient and a speed of 3.2 km/h until claudication or non-vascular symptoms led the patient to stop, or for a minimum of 15 minutes in the case of non-limiting symptoms or absence of symptoms.[6] During the treadmill test, patients were encouraged to report any vascular or non-vascular symptoms (e.g. angina pectoris, general asthenia and dyspnoea). All patients had a 12 lead electrocardiogram throughout the test and the test was stopped in cases of severe dysrhythmia or symptomatic or asymptomatic significant ($>$ 2 mm) horizontal or descending ST depression. The maximal heart rate was recorded during the test. Saturation was recorded throughout the test with a frontal probe. Significant desaturation was defined as a pulse oximetry under 90% with satisfactory heart rate estimation (to avoid abnormal signal detection as a cause of a falsely low saturation).

Exercise $tcpO_2$

Since initial development in the laboratory, the Decrease from Rest of Oxygen Pressure (DROP) index has been used for analysis of Ex-$tcpO_2$ results. The DROP is calculated as the absolute $tcpO_2$ change from rest of the limb minus the corresponding absolute $tcpO_2$ change of a chest electrode. Although absolute $tcpO_2$ is not a reliable measurement, by definition, the DROP becomes independent from absolute starting value. The study of exercise induced changes with the DROP calculation has therefore been shown to be accurate and reliable. The DROP is, by construction, equal to zero at rest. It remains mostly unchanged during exercise in normal subjects, whereas it decreases from zero during exercise and returns to zero in the recovery period in PAD patients with arterial claudication. During exercise tests, the lowest value for DROP (DROPm) is used to argue for a RBFI. It was found that a DROPm below -15 mmHg was highly predictive of the presence of exercise induced RBFI. This cut off point has been shown to be highly accurate compared with the 'gold standard' of angiography[5,6] and has good reliability in test-retest recordings.[10] The standard technique consists of using at least five $tcpO_2$ probes, one on each calf, one on each buttock, and one on the chest (TCM400 Radiometer, Copenhagen, Denmark). When available, six probes were used with the additional probe positioned on the lower back, thigh, or feet. Calibrations and procedures for the $tcpO_2$ recordings have been described extensively elsewhere.[5,6,10,11] The DROPm used for the analysis was the lowest right or left leg value.

Selection of patients for the analysis

From the initial database, patients were selected with an ABI under 0.90, who underwent a standard treadmill protocol, with a history of calf exercise induced pain, regardless of the presence or absence of associated proximal (buttock, hip, thigh) or very distal (foot) claudication, with normal $tcpO_2$ results on both calves (DROPm ≥ -15 mmHg), with calf pain on treadmill, and who stopped the test (for pain or medical reasons) before minute 15.

Lastly, for patients who had undergone multiple tests, only the results from their first visit were analysed.

Data analysis of selected patients

From the recorded data, a negative calf Ex-$tcpO_2$ despite calf pain both in the past and on the treadmill, and an ABI

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