Laser Doppler flowmetry in the diagnosis of chronic compartment syndrome

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Chronic compartment syndrome (CCS) is usually considered to be due to ischaemia of muscle. We have attempted to use the direct measurement of muscle blood flow for diagnosis since the assessment of intracompartmental pressure does not provide accurate knowledge of the vascular state. We recorded simultaneously continuous measurements of the laser Doppler flow (LDF) in muscle and the intracompartmental pressure (ICP) after exercise in seven patients with CCS, and in seven control subjects.

The mean ICP was 74.1 ± 4.4 mmHg in CCS patients and 24.2 ± 3.4 mmHg in control subjects one minute after exercise, decreasing to 34.6 ± 2.3 mmHg and 15.0 ± 1.6 mmHg at 20 min, respectively. The LDF was 0.80 ± 0.11 arbitrary units (AU) in control subjects and 1.09 ± 0.14 AU in CCS patients one minute after exercise, and 0.41 ± 0.11 AU and 0.27 ± 0.04 AU, respectively, at the end of the recovery period.

The ICP showed a progressive decrease over time in both groups. The LDF decreased sharply during the first minutes of recovery in control subjects, but in patients with CCS there was a delayed hyperaemic peak with blood flow reaching 0.84 ± 0.10 AU at nine minutes as against 0.33 ± 0.06 AU for control subjects (p < 0.01). The ICP increased in both control subjects and CCS patients after exercise with no clear cut-off point between the groups. By contrast, changes in muscle blood flow over time were clearly different between control subjects and patients with CCS.

For this reason, LDF should be investigated further as a technique for the diagnosis of CCS.

Chronic compartment syndrome (CCS) below the knee is seen mainly in trained athletes after prolonged or intensive exercise. The intracompartmental pressure (ICP) is normally high at the end of exercise, and usually returns to a resting value within 40 minutes. The vascular origin of the syndrome is debated, but many studies have shown that high tissue pressure affects the microcirculation of muscle, leading to ischaemia when the metabolic demand is increased by exercise. There is a physiological increase in ICP during exercise in normal subjects and there is no consensus as to the level of pressure which is diagnostic of CCS.

Attempts have been made to measure muscle blood flow after exercise using clearance methods, but such research methods are not generally available and are technically demanding. The long half-life for the wash-out of radiolabelled molecules means that continuous measurements cannot be made during recovery when the muscle blood flow changes rapidly. Recent studies have used MRI or scintigraphy to measure muscle perfusion or metabolism, but both techniques are too expensive for routine use.

We have used laser Doppler flowmetry (LDF) with commercially available implantable optic fibres to record microcirculatory impairment due to high intracompartmen- tal pressure in patients with CCS and in control subjects.

We have studied the pressure-flow relationship in hyperaemic muscle after exercise to discover whether direct measurement using LDF could help to define normal and abnormal pressure and the flow responses to exercise.

Patients and Methods

We studied 13 patients suspected of having CCS. All gave fully informed consent and the protocol was approved by our local ethical committee. We provisionally defined CCS as a compartment pressure greater than 40 mmHg one minute after exercise. On the basis of clinical symptoms and pressure recordings after exercise, we diagnosed CCS in seven patients. The other six were used as controls along with one other who had had a fasciotomy for CCS 1.5 months previously. In all except one case we obtained follow-up information from a questionnaire sent to the patient or their usual physician at six to 36 months. Of the
seven control subjects, two had spontaneous resolution of their symptoms, one was diagnosed as having the adductor canal syndrome, one had a stress fracture and another an atypical L4-L5 sciatica.

No measurements were performed at rest to avoid pain induced by the catheters during exercise. The subjects exercised on a treadmill with no slope. A warming-up period of 10 to 30 minutes was used and then exercise was started at 6 km/hour and increased by 2 km/hour every five minutes until pain appeared or the patient was exhausted. At the end of the test, the subjects lay supine with their knees extended and feet in a neutral position. Recordings were started within one minute and stopped at 20 minutes.

**Technique.** A catheter completely filled with saline with no air bubbles was connected by a three-way stopcock to a side-ported needle (Stryker, Kalamazoo, Michigan), an electric syringe and a disposable pressure transducer (Mal- linckroot, Petten, The Netherlands). The needle was inserted into the muscle at a 30° angle to the long axis of the leg. In 12 patients it was placed in the anterior compartment and in two in the superficial posterior compartment. A volume of 0.3 ml of sodium chloride USP 0.9% was injected slowly to a similar pressure to that of the interstitial fluid and then infusion was continued at a rate of 1.5 ml per hour. The ICP was recorded through a digital monitor (Siemens, Germany) on a personal computer. Direct pressure on the muscle compartment produced a brisk response on both recorders, confirming the correct position of the needle. The pressure transducer was zeroed before the puncture at the level of muscle measurements.

The microcirculatory muscle blood flow, expressed in arbitrary units (AU), was recorded using a laser Doppler flowmeter (PF4000; Periflux, Perimed, Sweden). A sterilised flexible optic fibre 10 to 15 cm long was introduced into the muscle through an 18-gauge needle, close to the site of pressure measurement at a maximal angle of 30° to the muscle axis. The needle was then carefully withdrawn, leaving the fibre in position in the muscle. Signals were recorded on the computer simultaneously with the pressure recordings. Non-circumferential tapes were used to fix both the pressure needle and the optic fibre.

We used continuous recording, but averaged each 30-second period to reduce the instantaneous variability of the signals. Non-linear regression analysis of the relationship between the LDF and ICP was performed on the Systat program using the least square method (Systat Inc, Evanston, Illinois). We assessed intergroup differences using Mann-Whitney tests, and one-minute to end-of-recovery differences using a Wilcoxon test. Correlation between the LDF and ICP was assessed by the Pearson correlation coefficient. A p value of less than 0.05 was regarded as statistically significant, and the results were expressed as the mean ± SEM.

**Results**

There were no immediate or delayed general or local complications. Typical post-exercise recordings from a
A typical example of raw data for intracompartmental pressure (ICP) (a), muscle blood flow (LDF) (b) and subjective score for pain (c) in a patient with compartment syndrome.

Muscle blood flow (LDF) and intracompartmental pressure (ICP) in CCS patients (squares) and control subjects (circles) during the first 20 minutes of recovery from incremental maximal exercise.
minutes the LDF was 0.27 ± 0.04 AU in controls and 0.41 ± 0.11 AU in the CCS group. The mean LDF remained high during the first minutes of recovery in the latter group. There is an exponential relationship between the mean ICP and the mean LDF in control subjects ($r^2 = 0.997$), but it is more complex in subjects with CCS, with a plateau during the first minutes followed by a continuous decrease to the end of recording (Fig. 4).

**Discussion**

The diagnosis of CCS can sometimes be made on clinical examination alone, but before operation is advised most authors agree that it should be confirmed by measurement of the ICP, although this provides no direct information on the impairment of muscle blood flow. Several methods of measuring the ICP have been described, and most provide easy, reliable and accurate measurement of the intramuscular pressure.\(^{17,18}\) Despite this, because the pressure increases in normal subjects during exercise\(^{7,8,10}\) there is no consensus as to the diagnostic criteria. The abnormal limits for the ICP have been variously defined as greater than 85 mmHg during exercise,\(^{19}\) 30 mmHg,\(^{8,10}\) 40 to 50 mmHg,\(^{20}\) and 75 mmHg at the end of exercise.\(^{21}\) The increase over resting pressure has also been used,\(^{8}\) with values of 20 mmHg,\(^{8}\) 40 mmHg at five minutes of recovery,\(^{21}\) or 15 mmHg after 15 minutes of recovery.\(^{9}\) The assessment also depends on the compartment studied. This considerable variation emphasises that simple pressure recording provides no direct estimation of the disorder of blood flow.

Styf, Körner and Suurkula\(^{22}\) attempted to measure blood flow in CCS using $^{133}$Xe clearance; they found a decrease of blood flow in the first minutes after exercise in the CCS group compared with normal controls. We found no significant difference between CCS and control patients during the first minute of recovery, but our failure to find a difference may be due to the relatively long delay between the end of exercise and our recordings. There are known to be very rapid changes in blood flow in the early recovery period after exercise and we probably missed the maximal LDF in the control subjects. The findings shown in Figure 3, however, allow us to hypothesise that the blood flow had reached a plateau and was not higher than that recorded during the first ten minutes of recovery in CCS patients. We did not measure resting blood flow, but the mean LDF soon after exercise was increased compared with that at the end of the recovery period. This is consistent with the findings of Qvarfordt et al\(^1\) who used Xe clearance in CCS patients to show a tenfold increase in muscle blood flow compared with resting values. This increase was significantly lower than that seen in the same patients after fasciotomy.

Amendola et al\(^2\) studied muscle metabolism by MRI and found prolonged periods of abnormal muscle metabolism in CCS subjects compared with normal controls; they suggested that the pathophysiology of the compartment syndrome did not appear to be related to ischaemia. We found delayed hyperaemia in CCS patients, which seemed to coincide with the relief of acute pain, although some had continued discomfort for hours after the study. This is consistent with recent reports using infrared spectroscopy\(^{23}\) and suggests that the compartment syndrome is related to ischaemia. It may be that the early symptom-producing effects of ischaemia have a more prolonged effect on muscle cell metabolism.

Previous clinical studies have shown that muscle blood flow may be impeded by externally applied pressure at rest and that there is an inverse relationship between transmural pressure and calf blood flow.\(^3\) Ashton\(^7\) also showed that the transmural pressure at which flow ceased depended on adrenergic vasomotor tone. Many animal models have been developed and studied\(^4,24\) leading to different physiopathological hypotheses for the cause, such as the critical arteriolar or capillary closing pressure\(^3\) or the waterfall phenomenon\(^25,26\) but some of these are debatable,\(^5\) and were defined from animal experiments at rest and not in hyperaemic human muscle.

Our study has confirmed that although individual differ-
ences in the effect of increased tissue pressure may account for differences in pressure tolerance, there is a threshold after which the microcirculation is altered, although muscle blood flow does not completely cease. Figure 3 shows that patients with CCS maintain their maximum flow for a longer period. The delayed LDF peak found in the CCS group suggests that the flow in the first minutes of the recovery period is not maximal, but limited by the effect of increased tissue pressure.

**Conclusions.** Our study shows that although there is no consensus on what is an abnormal pressure increase after exercise there is a different time course in the LDF findings in control subjects and patients with CCS. Pain relief in CCS patients coincides with the delayed peak of hyperaemia, supporting the hypothesis of the vascular origin of CCS. High intracompartmental pressure appears to limit the maximal blood flow resulting from metabolic hyperaemia of muscles after exercise.

Laser Doppler flowmetry provides an interesting method for the direct measurement of the circulatory disorder in CCS, and can be used to help in the diagnosis. The sensitivity and specificity of the method need analysis in a larger population, but the technique shows promise for further studies of the physiopathology and the diagnosis of chronic compartment syndrome.

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**References**


