Cent. Eur. J. Med. • 9(2) • 2014 • 235-239
DOI: 10.2478/s11536-013-0262-6

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Central European Journal of **Medicine**

Does a pressure increase translate into an adjacent compartment? A cadaver study

Research Article

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Received 7 August 2013; Accepted 14 October 2013

Abstract: Objectives: The influence of local pressure elevation on the adjacent compartment of the lower limb is poorly described. We examined these effects in a non-fractured cadaver model. Methods: Paired legs of unfrozen specimens were used. The pressure in the anterior and in the peroneal compartment of the contralateral limb was raised by bolus injections of saline (5cc bolus/60 seconds. Pressure changes in the adjacent compartments were measured. Results: Pressure increase in the anterior and peroneal compartment, up to 100 mmHg did not affect the mean maximum pressure changes in the other compartments. Exceeding 100 mmHg in the anterior compartment, the peroneal pressure was 13.67 mmHg(range 8-20 mmHg), the deep posterior 7.50 mmHg(5-13 mmHg) and the superficial compartment pressure was 6.67 mmHg(4-9 mmHg). In cases of raised pressures in the peroneal compartment above 100 mmHg, the anterior, deep and superficial posterior compartments showed pressures of 12.5 mmHg(11-15 mmHg), 7.5 mmHg(4-11 mmHg), and 7.5 mmHg(4-12 mmHg). Conclusions: Our data suggest that increased pressures in the anterior tibial or peroneal compartments do not directly influence the neighbouring compartments. It appears that in an intact compartment, the fascia seals pressure elevations in the range that is clinically relevant. Whether or not this may have clinical impact on the indications for single versus multiple compartment fasciotomy should be subject to further studies.

Keywords: *Compartment syndrome • Compartment pressure measurement • Tibial fasciotomy • Adjacent compartment*

1. Introduction

Acute compartment syndrome (ACS) can result in local ischemia of muscle, vascular or neural structures and represents a surgical emergency [1-3]. No major change in the theory of the underlying pathophysiology has been observed in the last decades [4-8]. It may be caused by either a volume increase within a closed fascial space, or externally applied pressure that compresses muscular structures8. It is crucial to assess the particular history of the patient. Ulmer reported that the sensitivity of clinical findings is generally poor [9]. Shuler suggests the need for more reliable means of assessing intracompartmental pressure [10]. The assessment and clinical findings of ACS are provided by intrafascial needle pressure measurement [11-13].

Direct trauma, haematoma, post ischemic reperfusion or other aetiology may induce an isolated ACS in a local compartment (LCS) [14-19]. Operative treatment by fasciotomy, if needed, was limited in these reports to the anterior and peroneal compartment. The pathophysiology of an isolated compartment ACS on the adjacent compartment is poorly described. Preliminary reports described the potential effect of the ACS in the deep posterior compartment of the lower leg on its superficial compartment inducing rising pressures [20]. There are no reports describing this potential effect induced by the anterior or peroneal compartments in human limbs. We hypothesize that an isolated pressure increase in this mentioned compartment without fractured bones may affect the adjacent compartments.

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2. Materials and methods

2.1 Specimens

In this study adult, fresh and unembalmed cadavers were used. Inclusion criteria: no lower leg surgery, no fracture, no skin damage or any other operations of the extremity. In supine position the heel was raised 2 inches and positioned release resting pressure effects at the posterior compartments. All experiments were performed at room temperature.

2.2 Technique of pressure measurement

All limb compartments were assessed by intracompartmental needle pressure measurement (Stryker Instruments, Michigan, USA). The compartments were broached in the midshaft area using four slit catheters, one in each compartment, after calibration at atmospheric pressure.

2.3 Technique of volume increase (pressure induction) and Assessment of pressure elevations

A cannula (2 mm diameter) was placed in the anterior or peroneal compartment to raise the pressure stepwise by infusion of 5cc of 0.9% sodium chloride solution per 60 seconds, respectively. In this volume-controlled model 100cc saline over 20 minutes was infused. This results in a gradual rise in pressure. The resultant pressure changes in the anterior or peroneal, and in the deep posterior and superficial posterior compartments were recorded for 20 minutes continously. The maximum pressure, documented in this study, was 200 mmHg. This corresponded to the upper pressure limit. The time points of pressure measurements were briefly before every bolus injection.

2.4 Statistics

To determine the correlation of the intracompartmental pressure (ICP) and the infused saline, we calculated correlation coefficients. Further statistical analysis was performed to determine whether there was a clear difference in the neighbouring ICP before (≤30 mmHg) and after simulated LCS (≥50 mmHg) increased at levels of significance. Hereby we excluded the inconclusive pressure measurement between 30 and 50 mmHg that imply re-evaluation. In addition, to simulate the comparison of an injured to un-injured limb, the ratio of mean pressure of the LCS over mean pressures of the summarized adjacent compartments at ≤30 mmHg vs. ≥50 mmHg, were calculated. Statistical analyses were performed using MedCalc, version 9.5.0 (MedCalc Software, Belgium).

3. Results

In this cadaver study we included six pairs of fresh, unembalmed lower legs cadavers. The summarized results of the measurements are shown in Figure 1, 2.

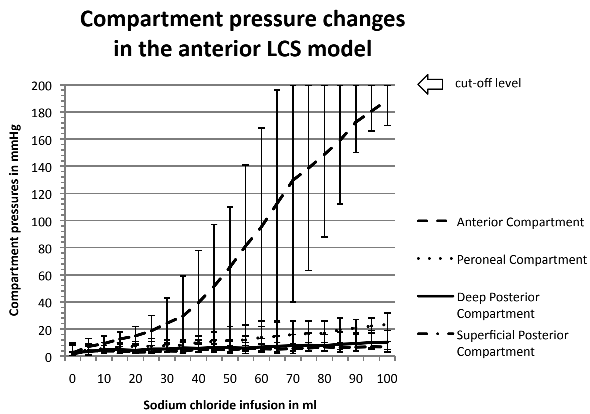

Figure 1. Pressure changes in all compartments simulating an anterior local compartment syndrome (LCS) in six cases.

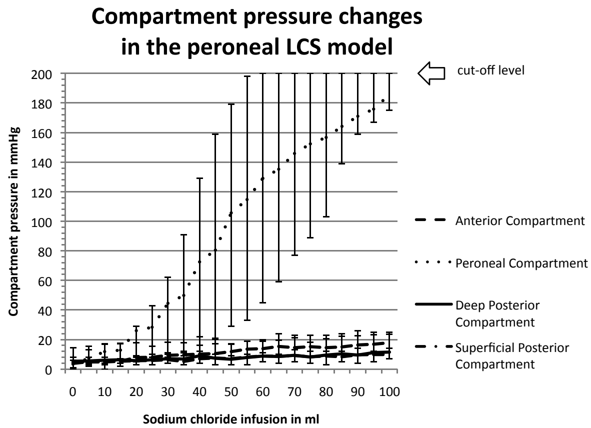

Figure 2. Pressure changes in all compartments simulation a peroneal local compartment syndrome (LCS) in six cases.

The mean volume of sodium chloride solution at 150 mmHg in the anterior compartment was 75,5 ml (range 55-95 ml) and in the peroneal compartment 70,5 ml (range 45-92 ml). No leakage of the set up was detected in any case and the raised heels ensured unaffected posterior compartments. In all cases the pressure increase in the anterior compartment showed a disproportional behaviour when the pressure increases raised beyond 100 mmHg. The other compartments showed elevated pressures up to 29 mmHg, but never exceeded 30 mmHg. After raising the pressure in the anterior compartment at a level of 100 mmHg, the mean maximum ICP increase found in the peroneal, deep, and superficial posterior compartments was 13.67 mmHg (range 8-20 mmHg), 7.50 mmHg (range 5-13 mmHg) and 6.67 mmHg (range 4-9 mmHg). Simulating a peroneal

LCS at a pressure of 100 mmHg the mean maximum ICP increase resulted in an anterior, deep and superficial posterior compartment pressure of 12.5 mmHg (range 11-15 mmHg), 7.5 mmHg (range 4-11 mmHg), and 7.5 mmHg (range 4-12 mmHg). The further compartments of the posterior aspect of the lower limb were not affected by the anterior or peroneal pressure changes. Pearson's correlation coefficients were obtained for ICP and the amount of infused saline in each compartment. All compartments showed a significant correlation to the volume change in the anterior or peroneal compartment model (Table 1). The average correlation coefficient in case of the anterior LCP was 0,989 and peroneal 0,966. Using the t-Test in case of the anterior tibial LCS the mean intracompartmental pressure (ICP) of adjacent compartments showed statistically significant ($p<0.05$) increase (Table 2), but did not exceed 30 mmHg.

Table 1. Pearson's correlation coefficient.

Correlation between compartmental ICP [mmHg] and infusion [cc]				
a. Anterior LCP				
Anterior	Peroneal	Deep posterior	Superficial posterior	Average
0,980	0,989	0,975	0,931	0,989
b. Peroneal LCP				
Anterior	Peroneal	Deep posterior	Superficial posterior	Average
0,9812	0,9893	0,9352	0,9587	0,966

Table 2. t-Test in case of anterior LCS model.

Compartment	n	Mean	Std. dev.	p-Value
Anterior Compartment				
ICP ≤ 30 mmHg	50	14,44	9,07	0.001
ICP > 50 mmHg	70	146,91	54,18	
Peroneal Compartment				
ICP ≤ 30 mmHg	50	6,24	3,04	0.001
ICP > 50 mmHg	70	18,17	6,20	
Deep Posterior Compartment				
ICP ≤ 30 mmHg	50	4,76	2,76	0.004
ICP > 50 mmHg	70	9,65	4,92	
Superficial Posterior Compartment				
ICP ≤ 30 mmHg	50	3,52	1,72	0.015
ICP > 50 mmHg	70	6,58	2,41	

Similar relationships were seen in peroneal local compartment syndrome; the adjacent anterior compartment pressure increased significantly ($p=0.002$) but did not pass over 30 mmHg. The posterior compartments did not develop any significant pressures changes (Table 3). The ratio of mean pressures of the simulated LCS over mean pressures of the summarized adjacent compartments at ≤30 mmHg vs. ≥50 mmHg, was calculated. In case of the provoked anterior as well as peroneal LCS the ratio showed statistically high significance ($p<0.001$) between the level of normal (<30 mmHg) and increased (>50 mmHg) intracompartmental pressures.

4. Discussion

The history of injury is the first evidence that a patient may be at risk for development of an acute compartment syndrome (ACS). The more the severity of the initial soft-tissue injury, the grater the jeopardy of soft-tissue related complications [21]. Clinical assessment of the lower limb in suspected ACS is crucial for decision-making [4,22]. The absence of fracture may result in a delayed diagnosis and increased risk of muscle necrosis [10,29]. The needle compartment pressure measurement is the golden standard to objectify raised compartment pressures [7,8,12], especially in case of constrained mental state of the patient. The most common mechanism is a closed tibal shaft fracture [23]. The incidence of a compartment syndrome of the lower limb associated with fracture and blunt trauma varies from 3 to 17% [24]. Also, overlooked compartment syndrome is a major liability risk for the treating physician. The question of fault is influenced particularly if there was failure to recognize or diagnose the compartment syndrome. Templeman et al. estimated the costs and lawsuits for awards after overlooked compartment syndrome at $280,000.00 [25]. Therefore exact understanding of elevated compartment pressures is important.

Our non-fractured model shows, that there seems to be little influence of pressure changes on the adjacent compartments in case of raised anterior or peroneal intracompartimental pressures. Simulating anterior compartment syndrome, even at pressures over 150 mmHg, the neighboured peroneal compartment showed no increase above 30 mmHg. This is unexpected, because the peroneal compartment's limiting structure, the muscle septum, is thinner that the surrounding fascia. The behaviour of the adjacent compartments in case of the raised peroneal pressure change was equally limited. In any compartment there was a close correlation with the amount of saline injection. Summarising the statistics there was a significant volume change in the most of adjacent compartments but none of them were pathologically relevant.

Despite the minor number of cases the measurements show distinct results of pressure changes constrained to the affected compartment. Our cadaver model is limited, because pathophysiological mechanism such as oedema, bleeding, or muscle contraction is not simulated. Nevertheless, the physical behaviour of the compartments concerning their dissemination and the pressure changes are investigated. Moreover, the pressure changes were not investigated in case of a fractured model simulating a local ACS.

In the absence of fracture compartment syndrome may develop in single compartments due to muscle rupture [26] or other (non-) contact injuries [18,19]. The impending compartment syndrome of a single compartment is at risk to be missed. Some authors described pathologically elevated single compartment pressures not effecting the adjacent compartment [26]. Other authors illustrated that an LCS can exert external pressure on the adjacent compartment and may induce an ACS [20]. Rorabeck et al. summarised patients with chronic compartment syndrome of the deep posterior compartment with insufficiency of fasciotomy. They assume a fifth compartment of the tibialis posterior muscle due to a separate osteofascial compartment [27]. Matsen et al. illustrated these sub-compartments [28]. No data is available on variations of human fascial thickness of the tibia. The individual anatomy of the osteofascial compartments of the lower leg seems to influence this behaviour. As far as we know, no previous publication describes the effect of raised pressures in the anterior or peroneal compartment on its adjacent limb compartments in a non-fractured, human lower limb model.

Recently Seel et al. inflated the deep posterior compartment by saline solution inducing an intracompartmental pressure of 100 mmHg. They proved a potential interaction with the adjacent compartments provoking the pathology of ACS. This data indicates a concrete interaction with a high risk of exceeding pressures in the adjacent compartment.

Our results do not concur with these findings. This difference may be due to the local diverging anatomy. While there is no osseous attachment between the deep and the superficial posterior compartment, the fascia between the fibula and the tibia is tightly spanned to form the syndesmosis. This fascial layer is usually stiffer and stronger than the one between both posterior compartments, because it adds to the stability of the distal tibial-fibular-joint. It is therefore likely to seal off any pressure changes exerted between the anterior and peroneal compartment. We feel, that the superficial pressure increase is caused by the communicating anatomy of the deep and superficial compartment. The large posterior myoseptal interface may lead to a direct impact of volume change of the deep compartment on its overlying neighbour. In case of an LCS of the anterior or anterolateral compartment the strong interosseous membrane protects the posterior compartments, if preserved. Thus, the biomechanical communication of the compartment interfaces maintain individual properties and should be categorized. All over limb fasciotomy is currently recommended in cases of an ACS of the lower limb [2,4,5,8]. The experimental data show, that an isolated ACS in the anterior tibial compartment, e.g. after a low velocity trauma or muscle tear may not affect the adjacent peroneal or the posterior compartments by pathological pressure changes in selected situations.

5. Conclusion

Our findings demonstrate that isolated pressure changes in the anterior or peroneal compartment do not transfer to the other compartment in case of an intact fascia, especially the deep and superficial posterior compartment. If not affected by trauma, those compartments may not have to be decompressed by fasciotomy in general. In case of an imminent compartment syndrome the monitoring of ICP tendency is recommended and raising pressure changes have to be detected early and confidently by the physician 3. This could be achieved either by improvements in pressure monitoring or investigation of muscle perfusion diagnostics.

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