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Is there a compartmental pressure transfer in lower limb fractures?

Research Article

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Abstract: Background. Limb fractures provoke elevated compartment pressures at the fracture site. This study investigates the pressure influence to adjacent compartments in cases of closed fractures. Methods. Paired lower limbs were used to simulate elevated pressures with and without fracture in a volume-controlled cadaveric model. Pressure increase was induced by saline infusion to a maximum pressure of 200 mmHg. The resultant pressure changes of all adjacent compartments (peroneal, deep and superficial posterior) were recorded simultaneously using handheld needle pressure measurement. Results. Six pairs of specimens were evaluated. The mean volume of saline infusion resulted in a significant volume increase in the fractured limbs compared with the anterior compartment of the control group ($p<0.05$). The pressure response in the affected anterior compartments was similar in the fractured and in the non-fractured limb. Adjacent compartment pressures did not increase noticeably even at high pressure levels. Conclusions. Elevated pressure simulation after closed fracture of the lower limb does not substantially affect the adjacent compartments compared with that of the non-fractured limb. The measurement of all compartments may be indicated to accurately assess indications of fasciotomy.

Keywords: *Acute compartment syndrome • Pressure measurement • Monitoring • Cadaveric tibia shaft fracture*

1. Introduction

Compartment syndrome requires an urgent and thorough exam and diagnostics to prevent irreversible muscle damage. Diagnosis of an acute compartment syndrome (ACS) relies mainly on clinical findings. In some cases, however, a reliable diagnosis cannot be made clinically, such as in unconscious or intubated patients. Thus, direct measurements of intracompartmental pressure (ICP) has been advocated [1-3]. While impaired soft tissue elasticity is a pivotal finding [4-8], manual palpation to detect decreased elasticity has low sensitivity [9] when used alone. To ensure a highly reliable diagnosis, pressure measurement of suspected compartments is required [10-12]. An ACS of the lower limb is mainly associated with a fracture. In the tibia, the anterior and deep posterior compartments are at greatest risk [10,13]. The intracompartmental pressure has been reported to vary within the compartment and is highest at the site of the fracture [13]. Thus, compartment pressure measurements should be performed close to the fracture site. However, the values of invasive pressure measurements may vary if the measurement device is not used correctly [13-15]. Although it is recognized that pressure changes within the compartment equilibrates [13,19], it is unclear whether this is also true for adjacent compartments in the case of a fractured limb.

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1.1. Hypothesis

Acute anterior compartment syndrome in a fractured lower limb may affect the adjacent compartments as a result of compartment injury. To test this hypothesis, we measured the pressure relationship between the anterior tibial compartment pressure increase and the adjacent compartments in a cadaveric closed fracture model of the tibia.

2. Material and methods

2.1. Specimens

This cadaver study utilized adult, fresh, never-frozen pairs of the lower extremities. The temperature of the extremities at the point of measurement was maintained between 60°F and 77°F. Only specimens without previous lower leg surgery, skin damage, fracture or other operations of the lower leg were included. One extremity was used randomly as a control. The other leg was fractured by a standardized blow to the middle of the tibia using a weight of 2 lb. This procedure creates a closed transverse fracture of the tibia and fibula. In the supine position, the heel and the knee were raised to a height of 5 cm and positioned on a board to ensure that the posterior compartments were unaffected.

2.2. Induction of increased compartment pressures

In this volume-controlled model, 300 cc saline was infused over 60 minutes. Infusion of 0.9% sodium chloride solution using a 2 mm cannula placed in the anterior compartment 2 cm proximal to the fracture. The pressure increase was performed stepwise, a infusing 5 ml bolus per 60 seconds.

2.3. Standardized pressure measurement

All limb compartments were assessed by intracompartmental handheld needle pressure measurement (Model 97010393, Stryker Instruments, Kalamazoo, Michigan, USA). Baseline pressure measurements of the peroneal, deep and superficial posterior compartments were quantified and recorded simultaneously. Four catheters were positioned 4 cm proximal of the fracture in each compartment. Calibration at atmospheric pressure was performed prior to measurements. In addition, the pressure changes in the anterior tibial compartment were detected 4 cm distally of the fracture to determine the intracompartmental pressure equilibration. In the non-fractured limb, the same setup was performed. All pressure changes were recorded every 60 second. Anterior pressure values of 200 mmHg were used as a cutoff point.

2.4. Statistics

Statistical analyses were performed using MedCalc, version 11.3 (MedCalc Software, Ostend, Belgium). The Pearson's correlation coefficients for each compartment were calculated to determine pressure-volume relationships. The volume of saline resulting in 30, 60, 100, and 160 mmHg anterior compartment pressures in the fractured and control limbs were determined (comparison of means). Further statistical analyses with the Mann-Whitney test were performed to calculate whether there was a clear difference of the ICP in all compartments compared to the anterior compartment before (≤30 mmHg) and after pressure increase (50 mmHg-150 mmHg). By this process we excluded the inconclusive pressure measurement between 30 and 50 mmHg.

3. Results

In this cadaver study we tested six pairs of fresh, unembalmed lower extremity specimens. The mean volume of saline infusion leading to anterior compartment pressures of 30, 60, 100 and 160 mmHg resulted in a significant volume increase in case of the fractured limbs compared with the anterior compartment of the control group (Table 1).

In all compartments, a volume-pressure correlation occurred. The mean coefficient showed strong correlations in the fractured ($r^2 = 0.961$) as well as in the non-fractured limb ($r^2 = 0.939$) (Figure 1-2).
Volume infusion in non-fractured specimens showed a rapid increase of the pressure in the anterior compartment. The adjacent limb compartments showed a statistically significant, but clinically irrelevant, pressure increase until the anterior ICP exceeded 160 mmHg (Table 2). Beyond these values a pressure increase occurred in the peroneal and in the deep posterior compartments.

The summarized pressure curves of the fractured specimens showed a similar behaviour (Figure 2). The mean pressure in the anterior compartment increases with rising volume injection, continuosly. A pressure change occurred in all adjacent compartments with anterior compartment pressures up to 150 mmHg. The peroneal compartment was mostly affected with maximum mean pressures of 35 mmHg. Afterwards

Table 1. Volume of saline infusion [ml] resulting in anterior compartment pressure levels of 30, 60, 100 and 160 mmHg in fractured and control limb.

Anterior ICP	fractured (n=6) mean		SD	control (n=6) mean		SD	p-value
30 mmHg	47,5	ml	±10,3	31,66	ml	±12,51	p=0.0377
60 mmHg	84,16	ml	±26,53	56,66	ml	±19,91	p=0.0697
100 mmHg	123,33	ml	±46,86	73,33	ml	±26.01	p=0.0450
160 mmHg	198,33	ml	±79,7	160,0	ml	±28,6	p=0.0106

Mann-Whitney test of adjacent ICP in normal (≤30mmHg) and increased (50-150mmHg) anterior compartment pressures in fractured and non-fractured specimens

Anterior	Peroneal		Deep posterior		Superficial posterior		Sum		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	p-values
Control group									
ICP ≤ 30 mmHg	4.17	±1.53	5.40	±0.3	3.36	±0.82	**4.28***	**±1.32**	
ICP 50-150 mmHg	9.80	±2.97	8.26	±1.83	5.27	±1.33	**7.78****	**±2.83**	**p<0.0001**
Fractured limbs									
ICP ≤ 30 mmHg	5.27	±0.34	5.02	±0.57	4.71	±0.51	**5.0***	**±0.53**	
ICP 50-150 mmHg	18.52	±8.84	13.31	±5.22	9.29	±2.57	**13.71****	**±7.14**	**p<0.0001**

* **p=0.079**
** **p<0.001**

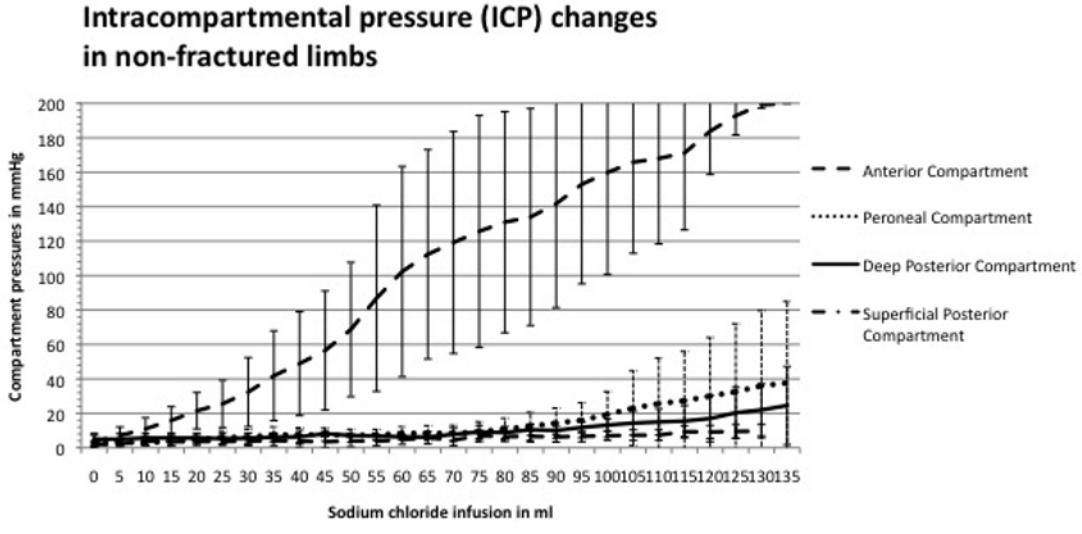

Figure 1. Resulting pressure changes in non-fractured limbs (n=6) (±SD included)

these neighbouring pressures increased gradually and exceeded marginal values.

The comparison of the pressure change in the anterior tibial compartment with and without fracture revealed a lower pressure increase in the fractured limb. The mean difference was not significant and indicated an excavation of the infused saline solution. No leakage of the setup was detected in any case., A statistically significant increase of pressure occurred in all adjacent

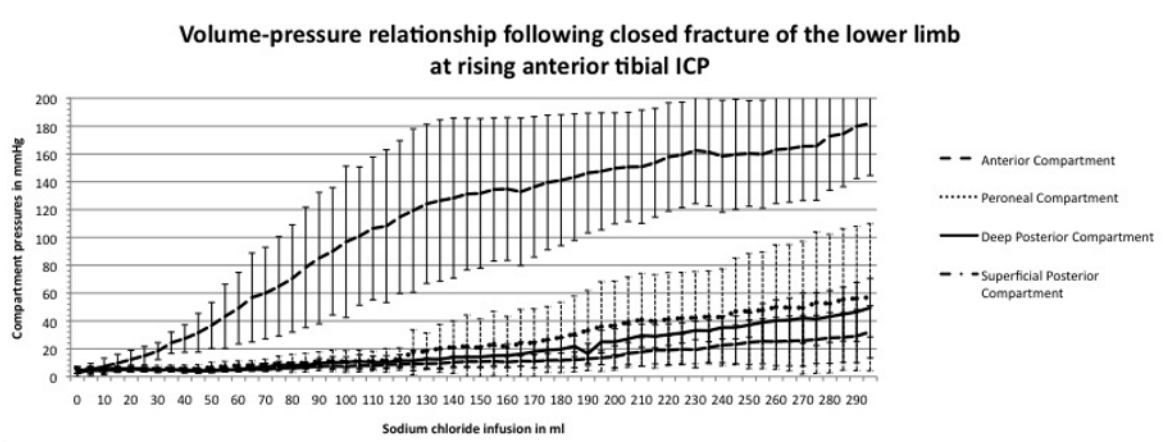

Figure 2. Resulting pressure changes in the fractured limbs (n=6) simulating anterior ACS (±SD included)

compartments in fractured compared with non-fractured specimens ($p<0.001$).

The simultaneously recorded pressure behaviour within the anterior compartment of the fractured limb showed deferred pressure dissemination. This pressure shift from the proximal to the distal part of the compartment implies a preserved entity of the fascial compartment.

4. Discussion

4.1. Intro

Subfascial compartment pressure measurement is used to determine the need for fasciotomy in uncertain cases [11,12]. In this study, numerous thresholds have been discussed and used historically to assist in estimating ACS [16,17]. In cases of local acute compartment syndrome (LCS) of the anterior compartment of the lower limb, there is no evidence that this affects the neighbouring compartment in a non-fractured limb. In contrast, a fracture of the lower limb may lead to significant dissemination of the compartment pressure in the case of ACS after laceration of the fascial structures. Therefore, we hypothesised the involvement of the adjacent compartments in cases of an isolated anterior LCS in a fractured limb. The main results of this study are as follows:

1. The volume required to achieve raised intracompartmental pressures is significantly higher in fractured lower limbs.
2. Increased pressures in the anterior compartment do not affect adjacent fascial layers both in fractured and unfractured lower limbs.
3. Pressure changes within each compartment do not demonstrate a significant delay between the area of infusion and the surrounding muscle.

This study has several limitations. The described method aims at the simulation of edema, not the fracture hematoma. The use of saline infusion may not imitate the intercellular fluid changes in an accurate manner. But the resulting pressure behaviour within the particular compartments seems to reproduce the nature of the pathology. The cadaveric model is not capable of simulating edema of the muscle tissue itself. However, this is an important aspect of the pathophysiology of the compartment syndrome. This was countered by the position of the infusing cannula, which was located two centimetres proximal of the fracture site. Thus edema, not a bleeding fracture, was simulated. In addition, the infused solution did not disseminate in a diffuse manner. Thus, an inaccuracy of the measurements has to be assumed. However, to observe the volume and pressure behaviour of the affected and threatened compartments, this model seems to achieve the highest analogy of the compartment syndrome mechanism. The number of cases is small, especially because the anatomic variation of compartment volumes varies with significant range between individual specimens. However, the resultant pressure behaviour was identical in every specimen.

It is suggested that invasive pressure measurement should be performed at the fracture site [11]. In this way, the source of raised pressure is restricted to the fracture hematoma. But the tissue involvement with edema resulting in an increased volume may be located elsewhere. This has to be considered, especially because the current efforts of improved diagnostics are aiming at the non-invasive determination of reduced blood perfusion [9,18]. Furthermore, the deep posterior and the peroneal compartments are at higher risk for

involvement in pressure increases than is the superficial posterior compartment [10]. This may be related to a higher impact of injury on the soft tissue envelope. In contrast, Prayson et al. demonstrated elevated compartment pressures of isolated lower extremity fractures up to 60 mmHg without any clinical appearance of an ACS [20].

We postulated that the discrimination of fracture hematoma and widely distributed edema of an ACS is important for a proper assessment of tissue involvement. However, there is no diagnostic device that can distinguish between hematoma and edema within the fascial layer. Both may lead to increased pressure changes within a single compartment, resulting in decreased tissue perfusion and saturation. Our results show that the fundamental compartment pressure behaviour does not change markedly with simple closed fractures of the lower limb. Understanding the pathophysiology of fluid changes and their compartmental interaction in cases of fractures of the lower limb may help to improve the estimation of soft tissue involvement. The pressure equalization within the affected compartment was not influenced to a high degree by the fracture and an assumed fascial tear. Thus, in cases of a simple and closed fracture of the lower limb, the compartmental pressure behaviour does not change. This is an important observation, in particular if the cause of the rising pressure can be differentiated between hematoma and edema.

5. Conclusion

Our findings demonstrate, contrary to our hypothesis, that isolated pressure changes in the anterior compartment do not transfer substantially to the adjacent compartments in the presence of a plain closed fracture of the lower limb. Despite the fracture and the expected fascial tear, the basic pressure behaviour of the single compartment does not change noticeably. This implies that acute compartment pressures do not equilibrate to adjacent compartments in cases of a limb fracture due to compartmental injury. Therefore, areas of local ACS should be considered as well as a fracture hematoma. In conclusion, the measurement of the compartment pressure may be more accurate at the area of highest firmness, regardless of the fracture site.

In cases of an imminent compartment syndrome, the monitoring of ICP is recommended. Raising pressure changes have to be detected early and confidently by the physician. We concur with other authors recommending multiple, repeated measurements in all adjacent compartments. Furthermore, we believe that a compartment pressure measurement should be completed on the fracture site and at the area of highest firmness. There is still a need for enhanced diagnostics. This could be achieved either by improvements in pressure monitoring or investigation of muscle perfusion diagnostics, considering the fracture hematoma and the soft tissue involvement.

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