INTRODUCTION:
The diagnosis of acute compartment syndrome (CS) of the extremity – increased pressure within the osseofascial compartments, leading to blocked circulation, lack of oxygen, and tissue necrosis – has been a grey area in clinical orthopaedics. The current warning criteria, which rests on the difference between the diastolic blood pressure and compartment pressure (∆P) in addition to primarily clinical observations, has been reported to be unreliable, resulting in a high percentage of unnecessary fasciotomy.

In investigating a more objective, accurate method of identifying CS, we measured the partial pressure of oxygen of the anterolateral (AL) muscle compartment in a canine model, as a novel approach to evaluating the direct marker of the underlying pathophysiology of the pressure-induced ischemia and tissue necrosis due to CS. Decreased tissue oxygenation (PmO2) due to reduced blood flow after CS is believed to be responsible for the tissue damage in the compartment. Continuous measurement of intramuscular PmO2 of the leg during controlled induction of CS in an established dog model1 has shown to be highly responsive and sensitive.

METHODS:
Animal model and PmO2 measurement: The study included 7 female beagles. After the induction of anesthesia, compartment syndrome was induced in the anterolateral compartment of right hind legs (CS limb) by colloid fluid (Hextend®) infusion through an intramuscular angiocath to maintain a compartment pressure 30mmHg above the diastolic blood pressure (∆P = -30mmHg) as measured by arterial line. In the contralateral positive control leg (TI limb), a tourniquet was applied over the upper leg and the pressure elevated to 300mmHg. Polarographic oxygen probes (Licox®, Integra LifeSciences) were placed percutaneously into the AL compartment of both legs to measure the PmO2.

After approximately 6 hours of compartment syndrome and tourniquet-induced ischemia, fasciotomy was performed and the tourniquet was deflated on respective legs. PmO2 was recorded every 30 seconds on the CS limb. Animals were euthanized at the conclusion of experiments. This protocol was approved by our institutional review board.

Tissue viability: Affected AL muscle tissue from the CS limb were biopsied at different time points: 3 hours after TI (rev TI), 6 hours after TI (end TI), 1 hour after tourniquet release (post TI), 4 hours after induction of CS (end CS), and 1 hour after fasciotomy (post CS). Tissue viability was assessed with MTT (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide) according to Crawford et al.3

The assay quantifies the reduction of a tetrazolium salt (MTT) to water-insoluble colored formazan crystals by mitochondrial enzymes of viable tissue. The absorbance of the formazan crystals at 570nm was normalized to the dry weight of the muscle sample. The tissue viability index was represented as the percentage of the normalized absorbance of affected tissue to that of negative control quadriceps tissue.

RESULTS:
The average duration of the controlled induction of compartment syndrome was 5.8 hours. Before colloid infusion, the averaged mean PmO2 of the CS limb was 30.71mmHg (range 10.56-50.18mmHg) and 3.65mmHg (range -0.50-3.65mmHg), and during induced compartment syndrome, the PmO2 decreased to an averaged mean of 1.22mmHg (range -0.06-0.41mmHg) and after fasciotomy, PmO2 promptly recovered in all animals to an averaged mean of 37.08mmHg (range 2.41-90.40mmHg, p<0.05). In the contralateral control limb, PmO2 decreased to 0mmHg after the application of tourniquet and promptly recovered to normal range after the release of tourniquet in all animals.

DISCUSSION:
We demonstrated that the controlled induction of compartment syndrome using an infusion method results in a substantial decrease in PmO2, as measured by polarographic oxygen probes, and in tissue viability index, as quantified by MTT, to values similar to tourniquet-induced ischemia. PmO2 was responsive and sensitive to changes in tissue oxygenation, as shown by the prompt recovery of PmO2 after fasciotomy and tourniquet deflation. Therefore, measurement of intramuscular tissue oxygenation appears to detect pressure-induced ischemia in an animal model with high translational potential.

SIGNIFICANCE:
This novel model for monitoring compartment syndrome with polarographic oxygen probes may represent a minimally invasive, physiologic, continuous, and reliable method for diagnosing compartment syndrome.

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REFERENCES:

Figure 1. Representative tissue oxygenation and temperature measurement in CS limb. Probe insertion at 9:33; start of infusion at 10:03; fasciotomy at 16:01.

Figure 2. Tissue viability index represented as percentage of the control quadriceps tissue.