Robert Ferrand, Inventor, Saddletech

In recent years, there are a group of veterinarians that are theorizing that Thermography can be a “proxy for pressure” measurement. The theory is put forward that the thermograph of the horses’ back after a short ride can reveal the fit of the saddle by determining the blood flow under the saddle. Some go even further to claim that a thermograph of the underside of the saddle can also reveal the fit……however, there is little to no scientific data to substantiate this theory. I am not aware of any direct comparisons of thermographs to interface pressure measurements

As you are well aware, I have studied the effect of pressure on blood flow on mammalian tissue extensively, initially in the development of a state of the art hospital bed for the prevention and treatment of bed sores on humans, and during the past decade applying this research to saddle fit on horses, using my patented saddle interface pressure measurement technology. (I probably have the largest collection of clinical research on this particular subject).

I put forth the proposition that it is clinically impossible for a thermograph to reveal saddle fit, because the reactive hyperemic response caused by the pressure of the saddle itself on the horses’ back will compromise the data set, regardless of fit. Since the hyperemic response causes the oxygen deprived tissues to be flooded (profusion) until balance is achieved, unfortunately, the proportionality of this effect is over time, not the amount of blood flow proportional to pressure and therefore not relevant to being a proxy for pressure. There is even evidence that there is not a direct relationship between skin temperature and blood flow, for a

number of reasons. For example, as the reactive hyperemic response decays the infrared heat from the core body temperature of the horse will further compromise the temperature data.

This data is further skewed by the calibration for the emissivity of the hair, thickness, and moisture content, relative to ambient temperature. Further claims that the saddle panel temperature is relevant is skewed by the thermal retention properties of the saddle panel materials themselves and cannot be related to blood flow or pressure, because the infrared heat from the horse will penetrate the tissues, regardless of blood flow, or the lack thereof.

The Bottom line, if you want to determine pressure measure pressure.

The marketing departments of some saddle companies have found a “story” to run with……So…last year, in an effort to better understand Thermography relating to saddle fit, I personally spoke with Dr. Tracy Turner about this issue of using Thermography for determining saddle fit. At the time, he told me that he thought, at best, it was a NO GO Gauge, to reveal a problem, and just told the client to go have someone solve it. I asked him, at the time, if he had ever tested Thermography against Interface Pressure and he told me that he had not, I offered my equipment and we even discussed that Dr. Mathew McKay-Smith was interested in such a clinical study for EQUUS magazine…..well he did not take me up on the offer.

……Then I discovered that Turner has been hired by the Bates Saddle Company to prove that the CAIR air panel “fits better than a custom saddle” by using Thermography.

Interface pressure measurement has been validated in a clinical setting in 1999, by Leo Jeffcott DVM, At Cambridge University Veterinary Hospital and published in a paper called, *"Validity of Saddle Pressure Measurements Using Force-Sensing Array Technology"* – Preliminary Studies. Veterinary Journal 1999 158 pgs 113-119

"The longer the period of occlusion, the greater the metabolic stimulus for vasodilation leading to increases in peak reactive hyperemia and duration of hyperemia.

All saddles cut off blood flow. The best fitting saddles that I have measured are about 1 PSI. Capillary Closure Pressure is less than .5 PSI
and 60% of blood flow occurs below .25 PSI.

Etiology

Several theories exist on the etiology of pressure sores, mostly based on ischemia and hypoxia resulting in decreased oxygen delivery to the tissues. In 1879, Charcot suggested that injury to CNS trophic centers decreases tissue tolerance to local pressure and leads to skin necrosis.[6] However, Brown Sequard demonstrated that pressure ulcers can heal equally well in paralyzed and nonparalyzed animals.[7]

The pressure ischemia theory maintains that pressure sores result from constant pressure sufficient to impair local blood flow to soft tissue for an extended period. This external pressure must be greater than arterial capillary pressure of 32 mm Hg to impair inflow and greater than venous capillary closing pressure of 8-12 mm Hg to impede the return of flow for an extended time. Constant external pressure for 2 hours or more produces irreversible changes in tissues in animal model studies. One study demonstrated no histologic changes with pressure release at 5-minute intervals.

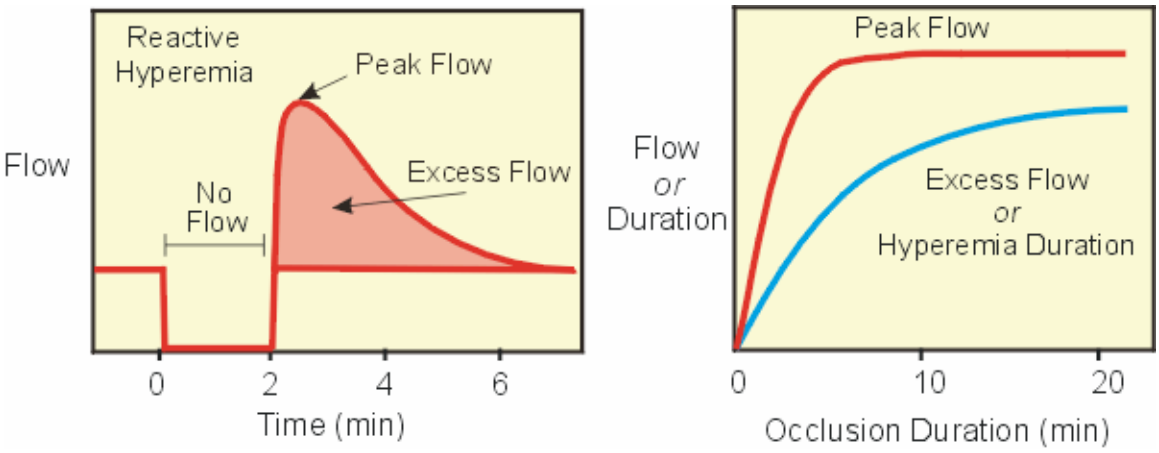
Lindan et al documented ranges of pressure applied to various anatomic

points in certain positions.[8] The points of greatest pressure with the patient supine included the sacrum, heel, and occiput, at 40-60 mm Hg. With the body in prone position, the chest and knees absorbed the greatest pressure, at 50 mm Hg. When the patient is sitting, the ischial tuberosities are under the most pressure, at 100 mm Hg. Obviously, these pressures are greater than end capillary pressure, indicating why these are the most common areas to develop pressure sores.

Furthermore, studies have demonstrated the pathologic changes caused by pressure to be more severe in muscle than in skin and subcutaneous layers.[9] These histologic studies revealed that early signs of damage occur in the upper dermis, with dilation of capillaries and venules and swelling and separation of endothelial cells.[10] Then, perivascular infiltrates, platelet aggregates, and perivascular hemorrhage develop in the dermis. Additionally, subcutaneous fat demonstrates signs of necrosis along with early vascular changes. Interestingly, the epidermis shows no signs of necrosis until late because epidermal cells are able to withstand a prolonged absence of oxygen both in vivo and in vitro.

Others have postulated that pressure ulcers result from metabolic deficits. Muscle has the highest nutritional demands, which helps explain this deeper tissue involvement preferential to skin.

http://emedicine.medscape.com/article/1293724-overview



http://www.cvphysiology.com/Blood%20Flow/BF006.htm