Direct Heel Pressure Causes a Greater Blood Flow Hyperemia Than Pure Occlusive Ischemia

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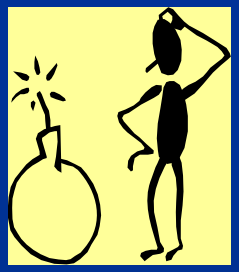
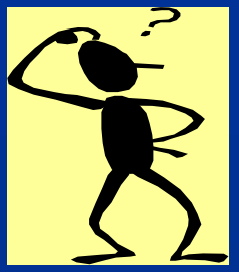
Problems & Questions



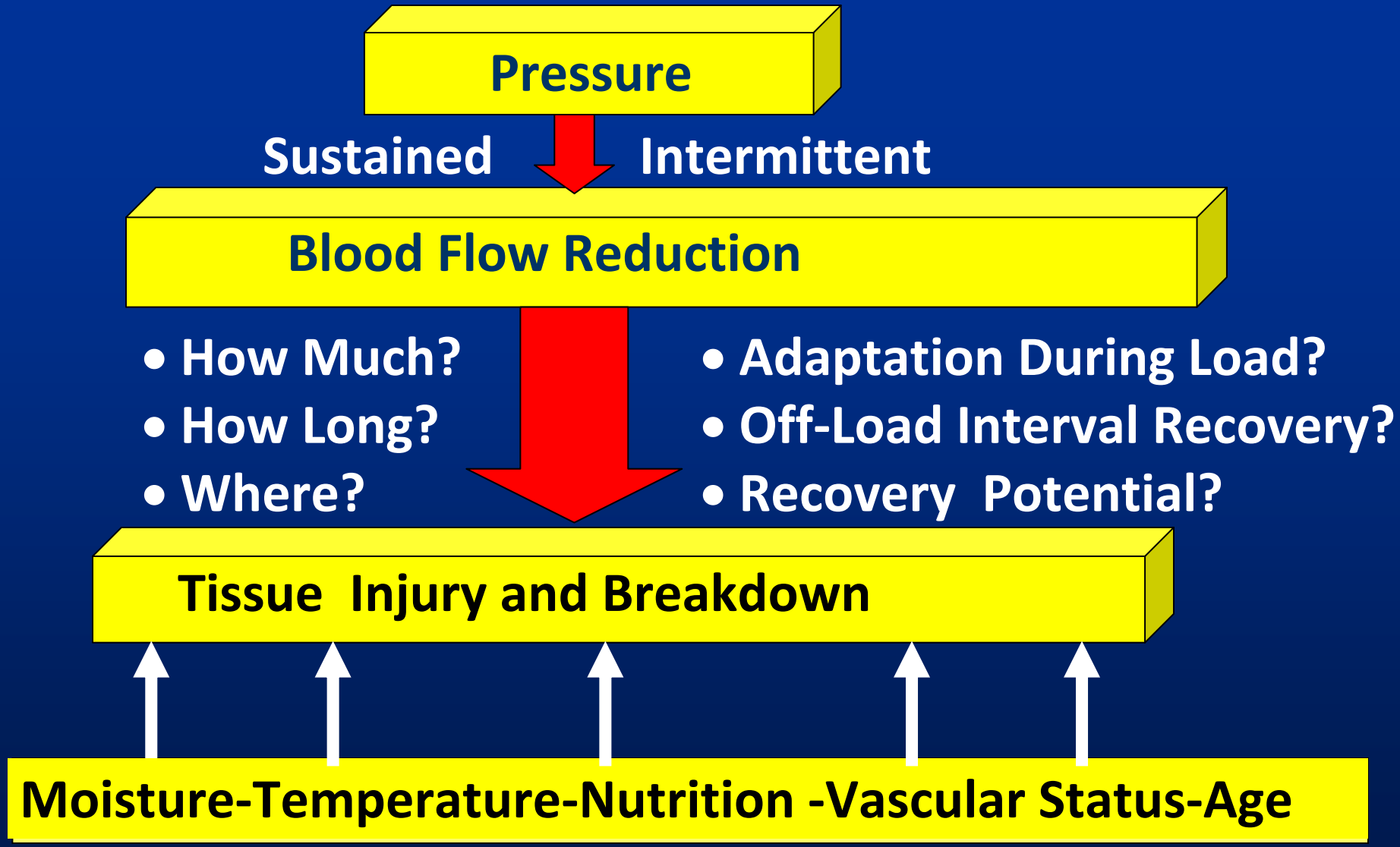


Pressure

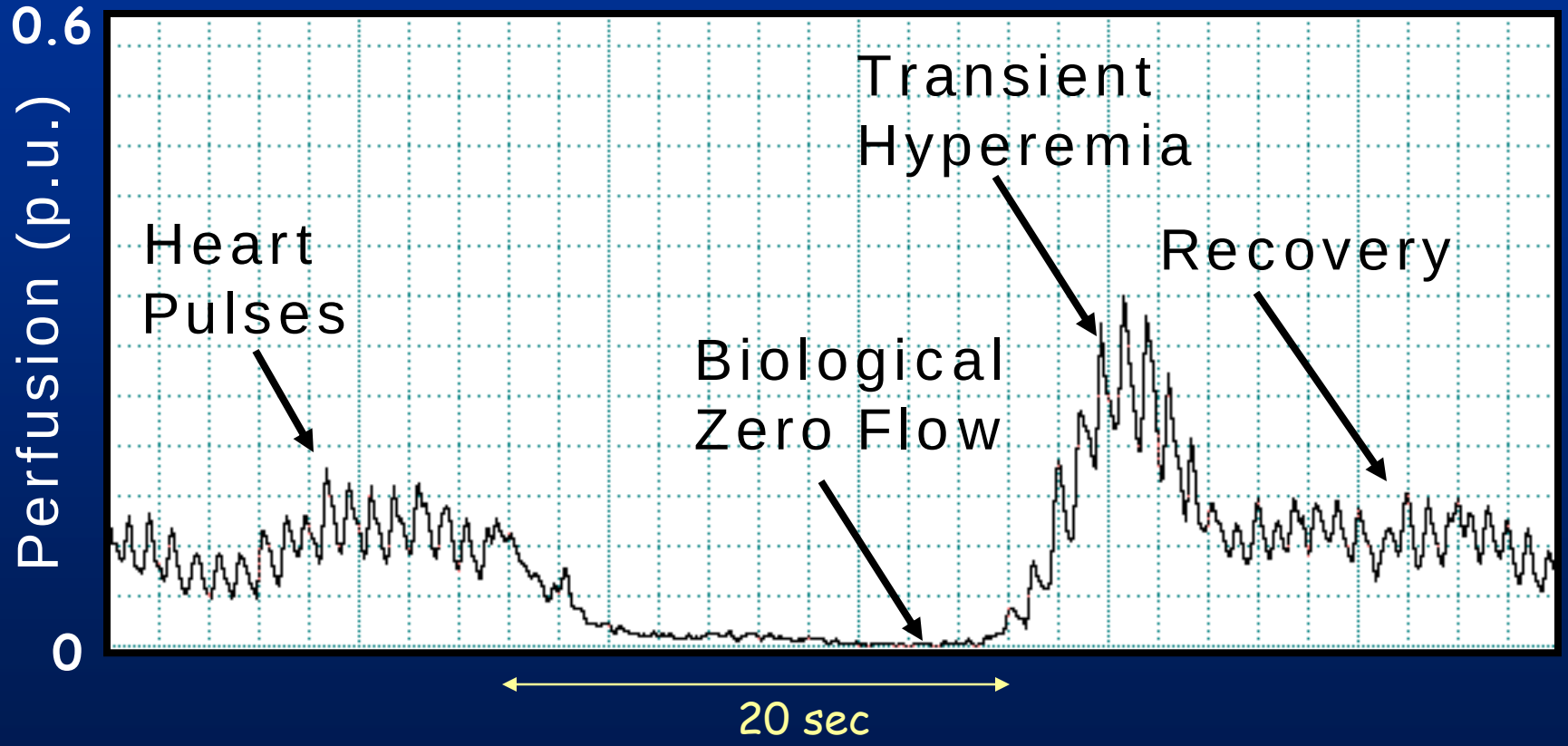
Blood Flow Reduction

Tissue Injury & Breakdown

Moisture-Temperature-Nutrition-Vascular Status-Age

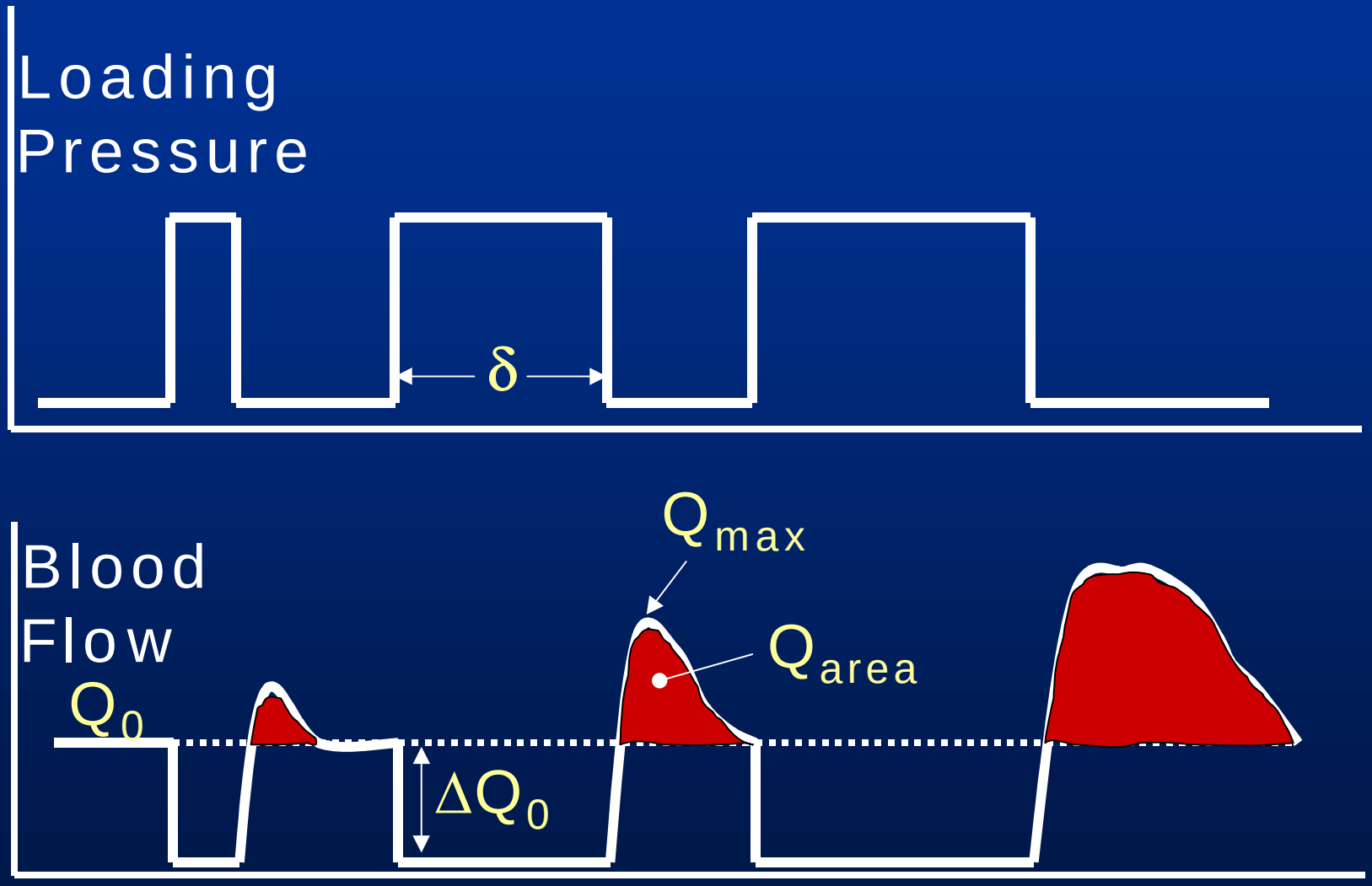

Pressure
Sustained
Intermittent
Blood Flow Reduction
• How Much?
• How Long?
• Where?
• Adaptation During Load?
• Off-Load Interval Recovery?
• Recovery Potential?
Tissue Injury and Breakdown
Moisture-Temperature-Nutrition -Vascular Status-Age

Blood Flow Arrest

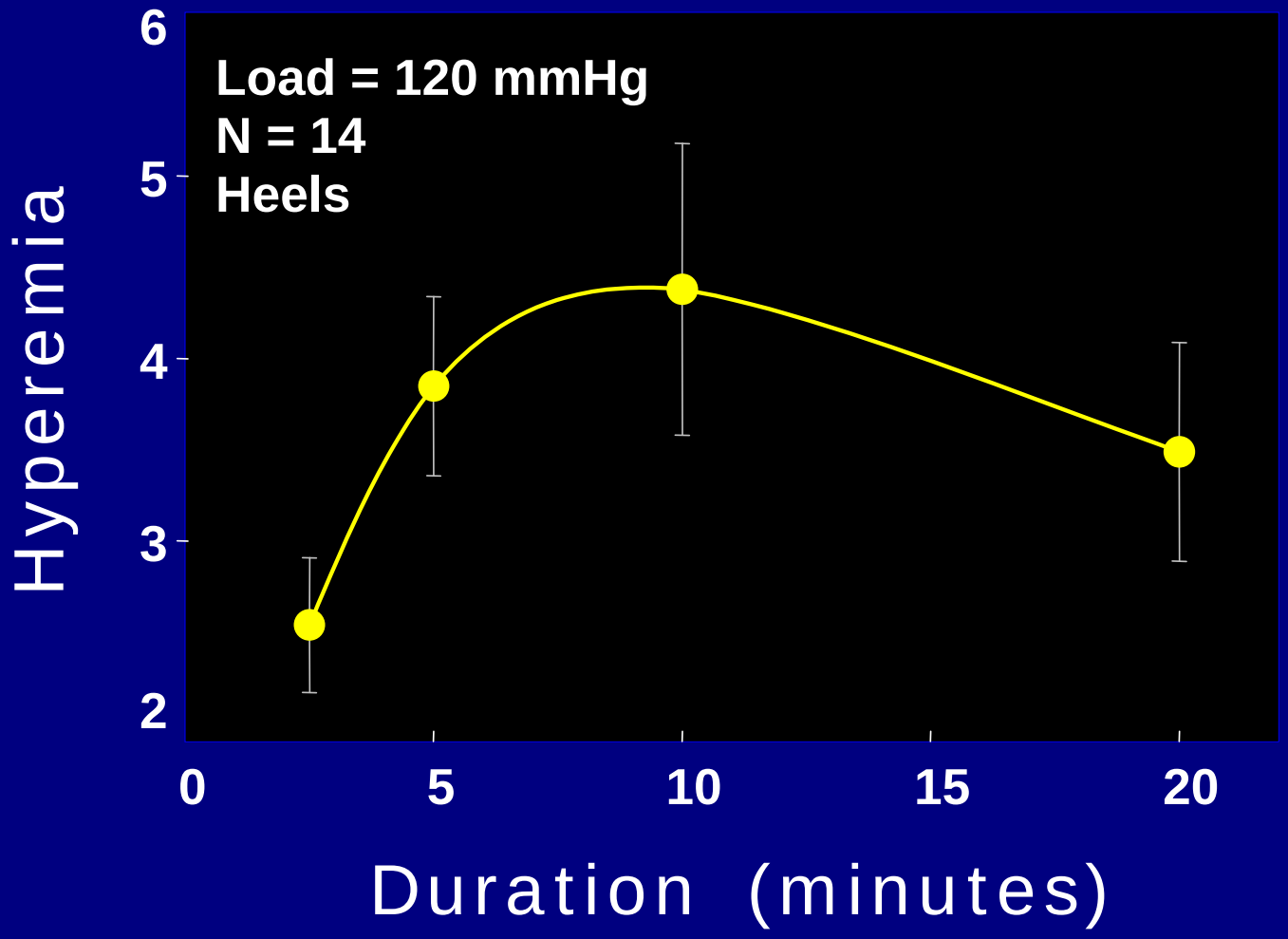


Post-Compression Hyperemia

Response to Load-Induced Ischemia

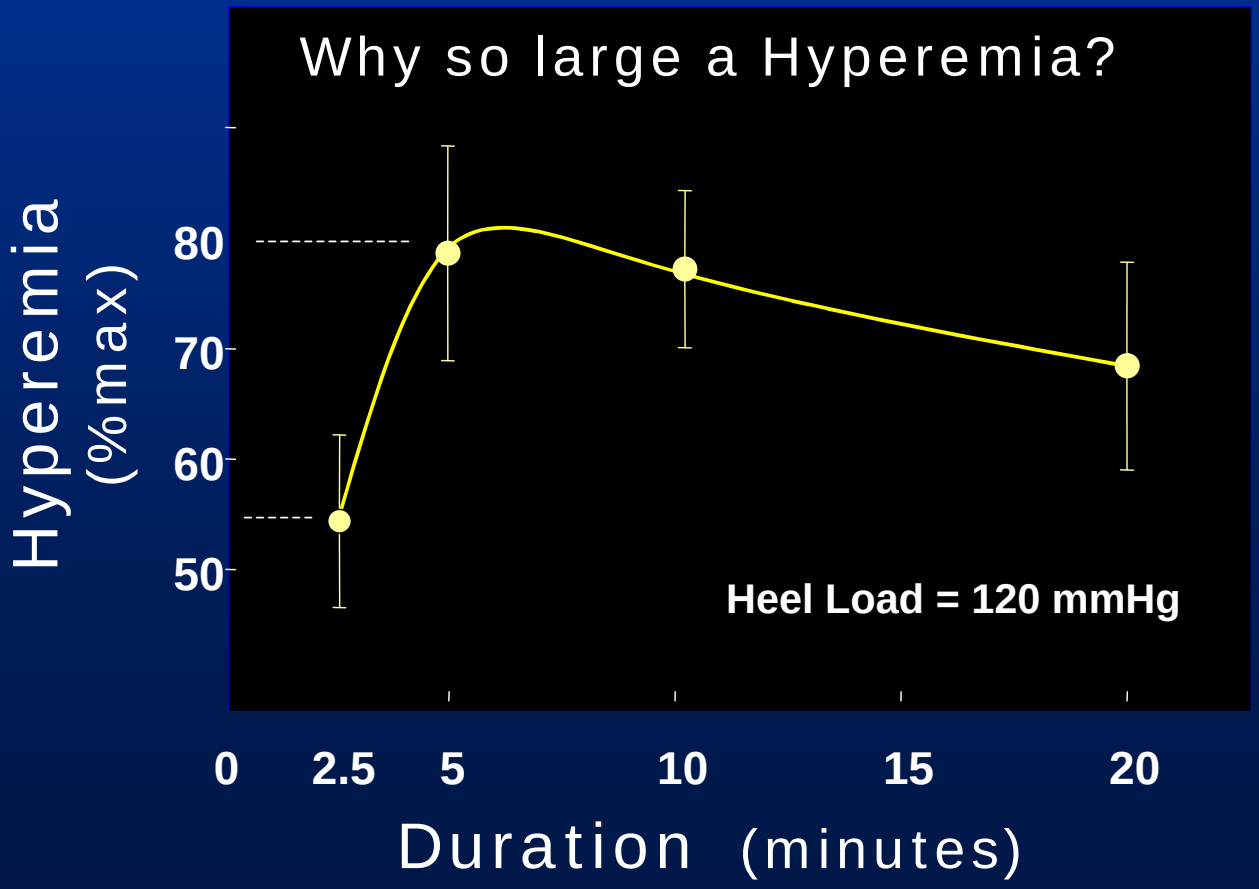


Load Duration Dependence



Basic Question

Is Post Compression Hyperemia responding only to prior intervals of tissue ischemia?



Basic Question

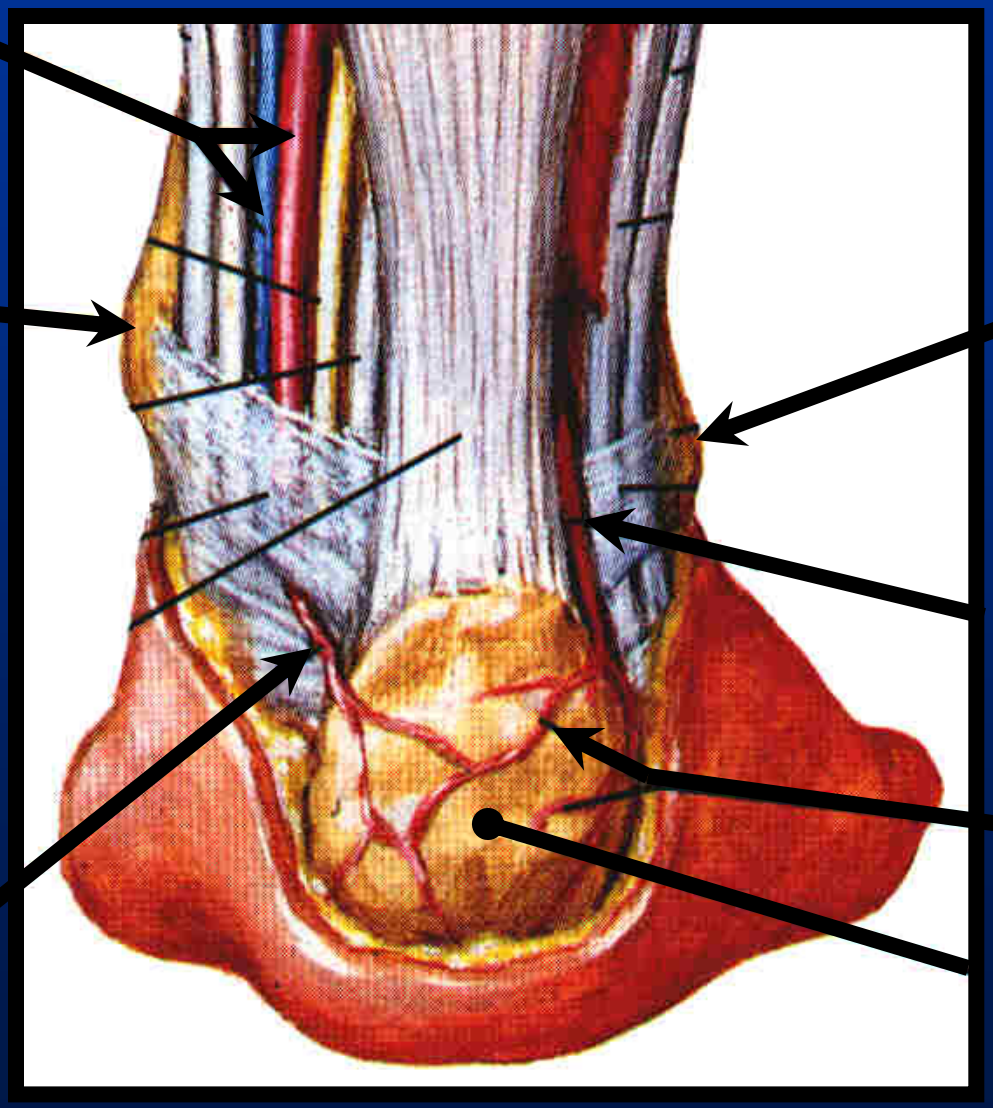
Is Post Compression Hyperemia responding only to prior intervals of tissue ischemia?

Studied heel hyperemic responses to 5-minute intervals of

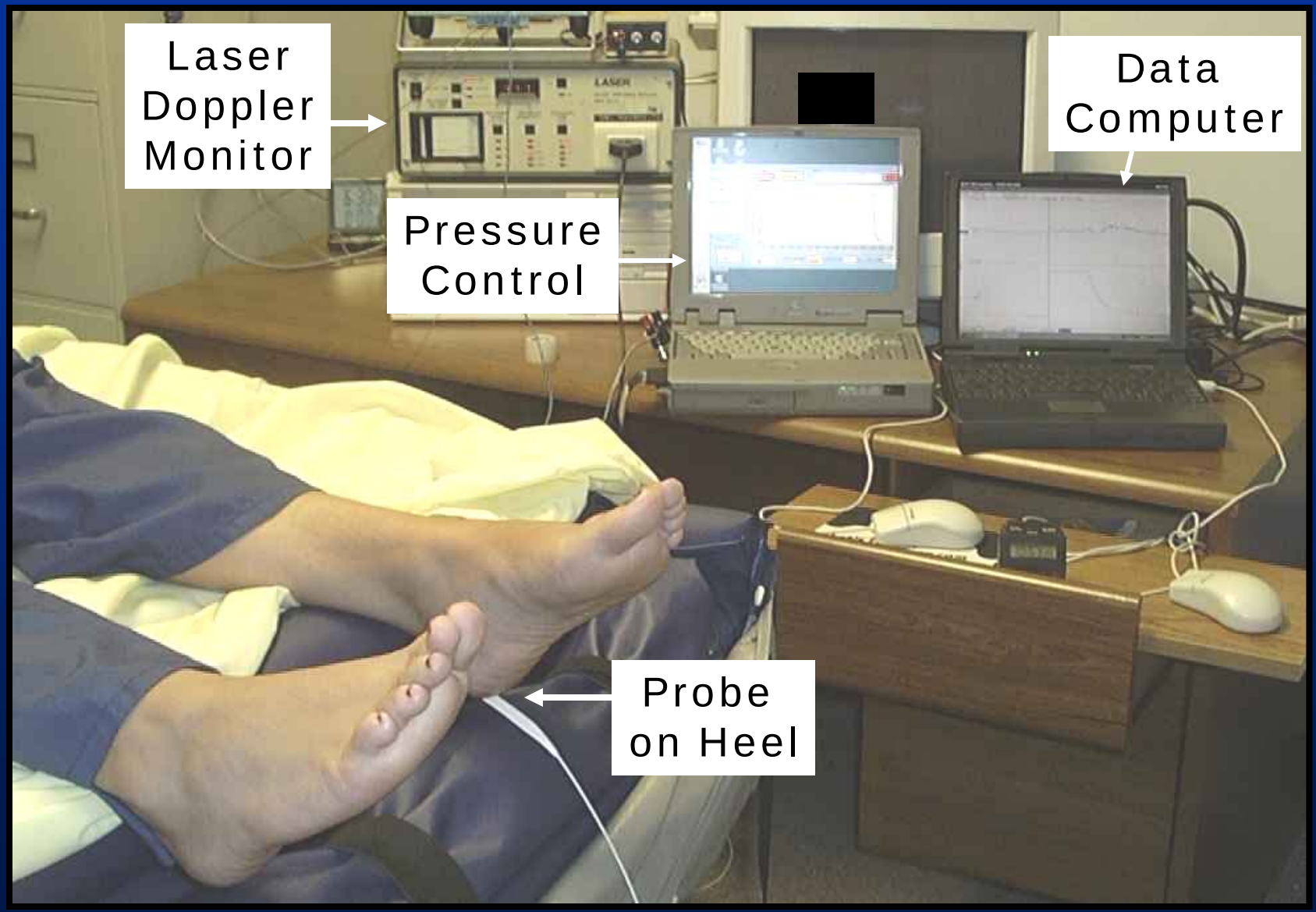
- Direct Compression and
- Occlusion ischemia

in 28 healthy subjects

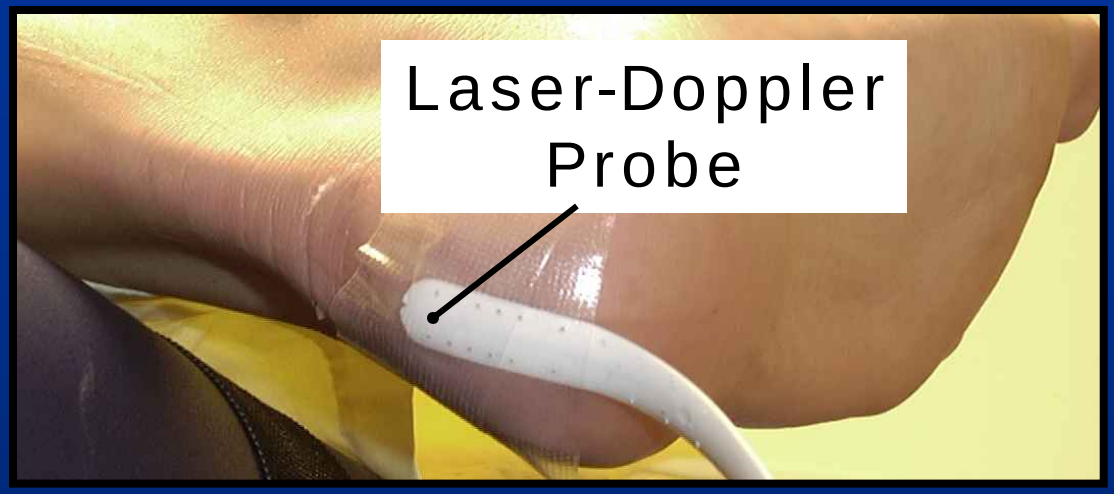
Heel Target

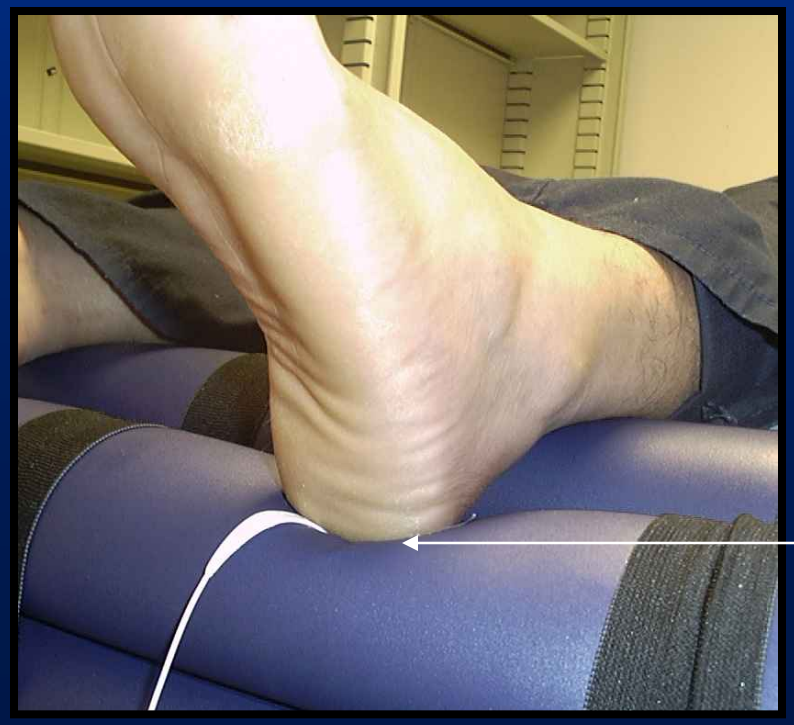

Posterior Tibial A & V
Medial Malleolus
Calcaneal Branch of PT
Lateral Malleolus
Peroneal artery
Calcaneal Branches
Calcaneal Tuberosity

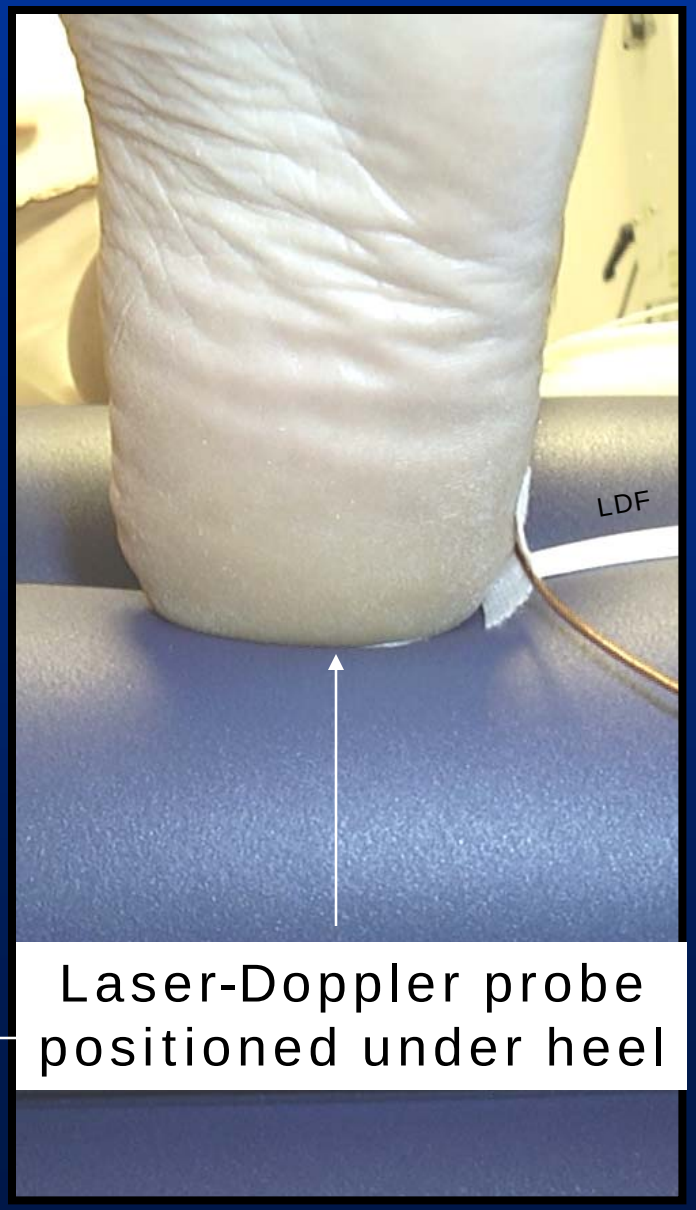
Data Acquisition



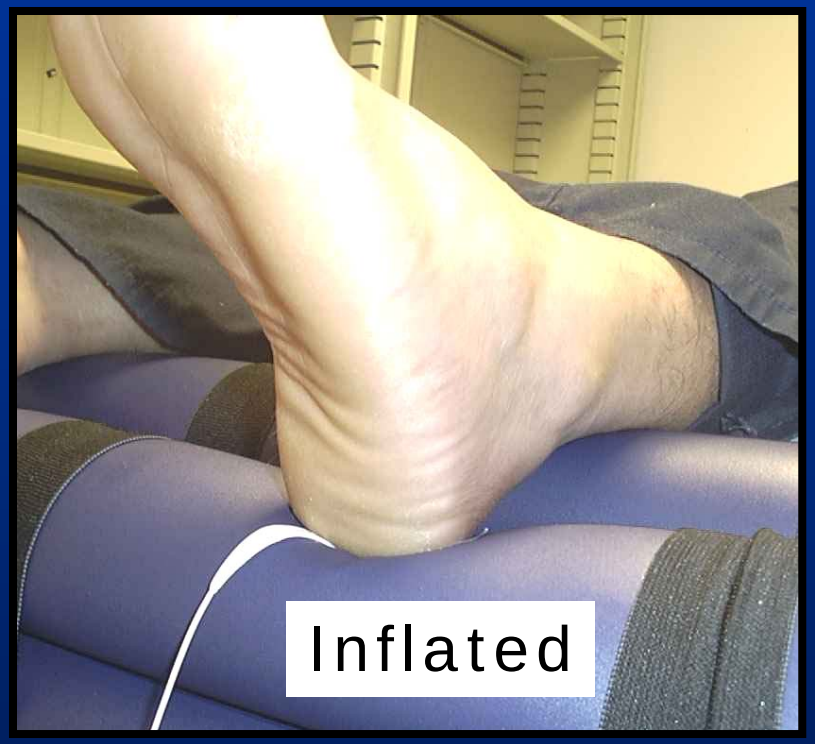
Heel Compression





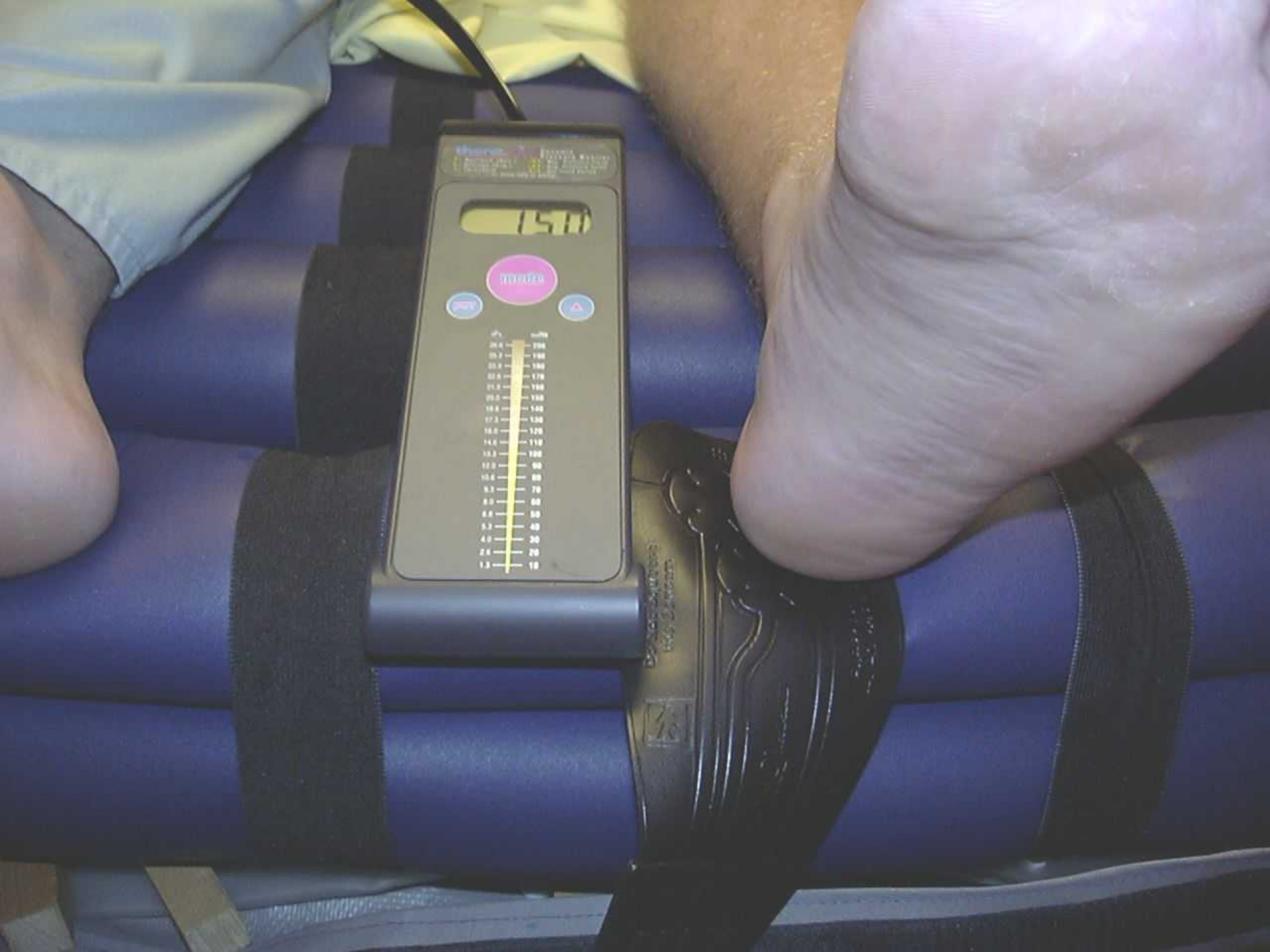


Heel Loading and Unloading

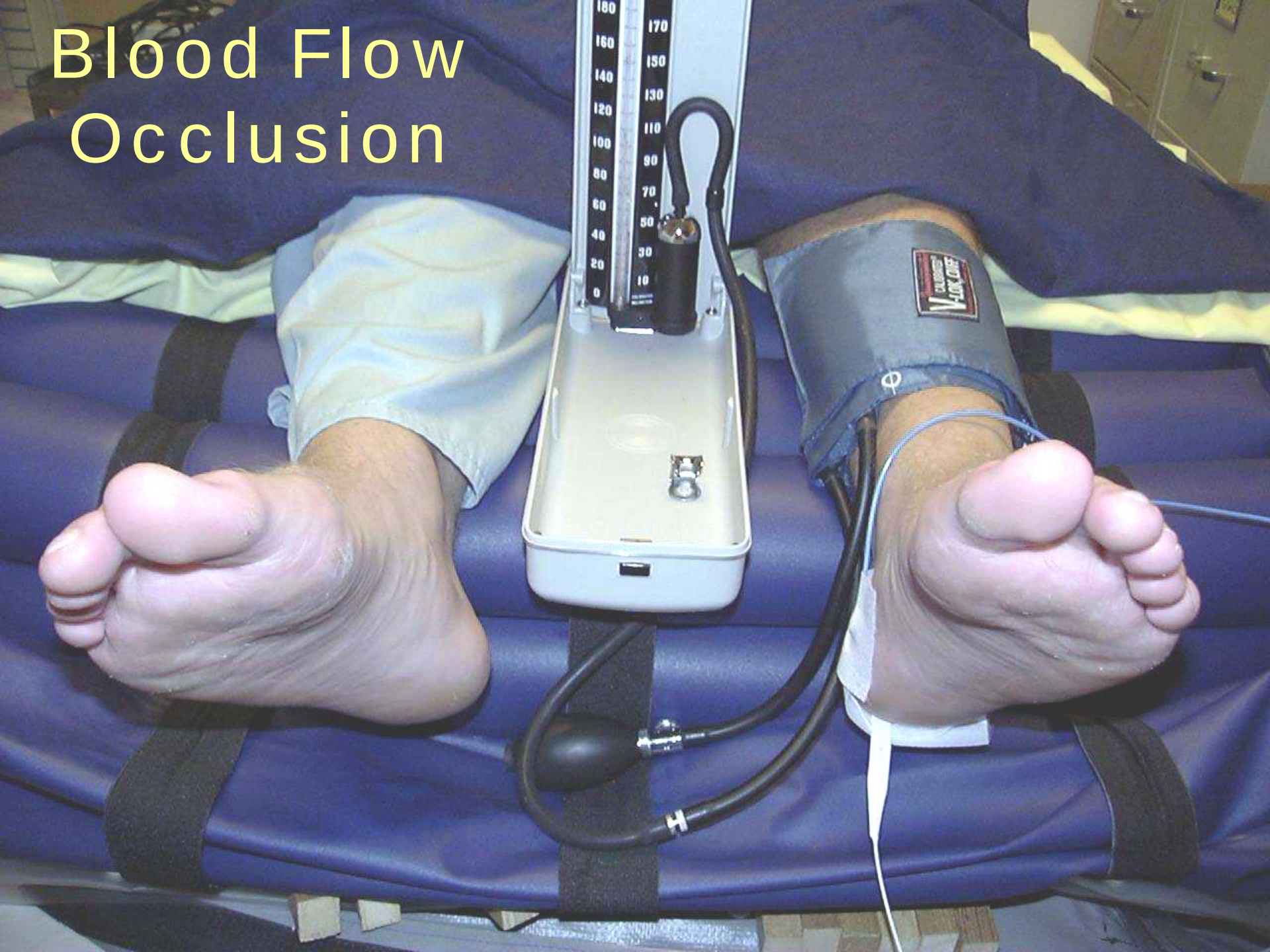


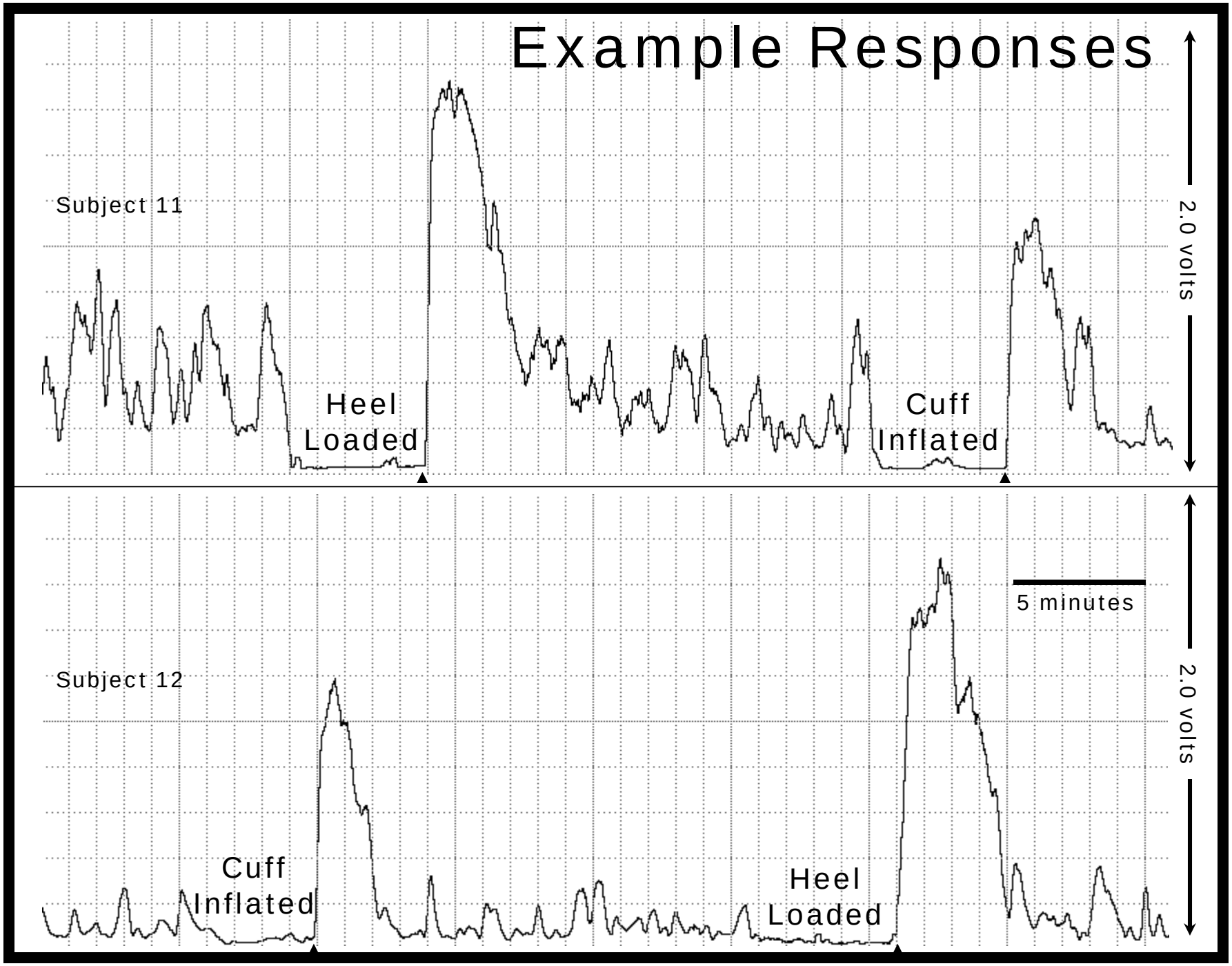
Heel Loaded

Heel Fully Unloaded

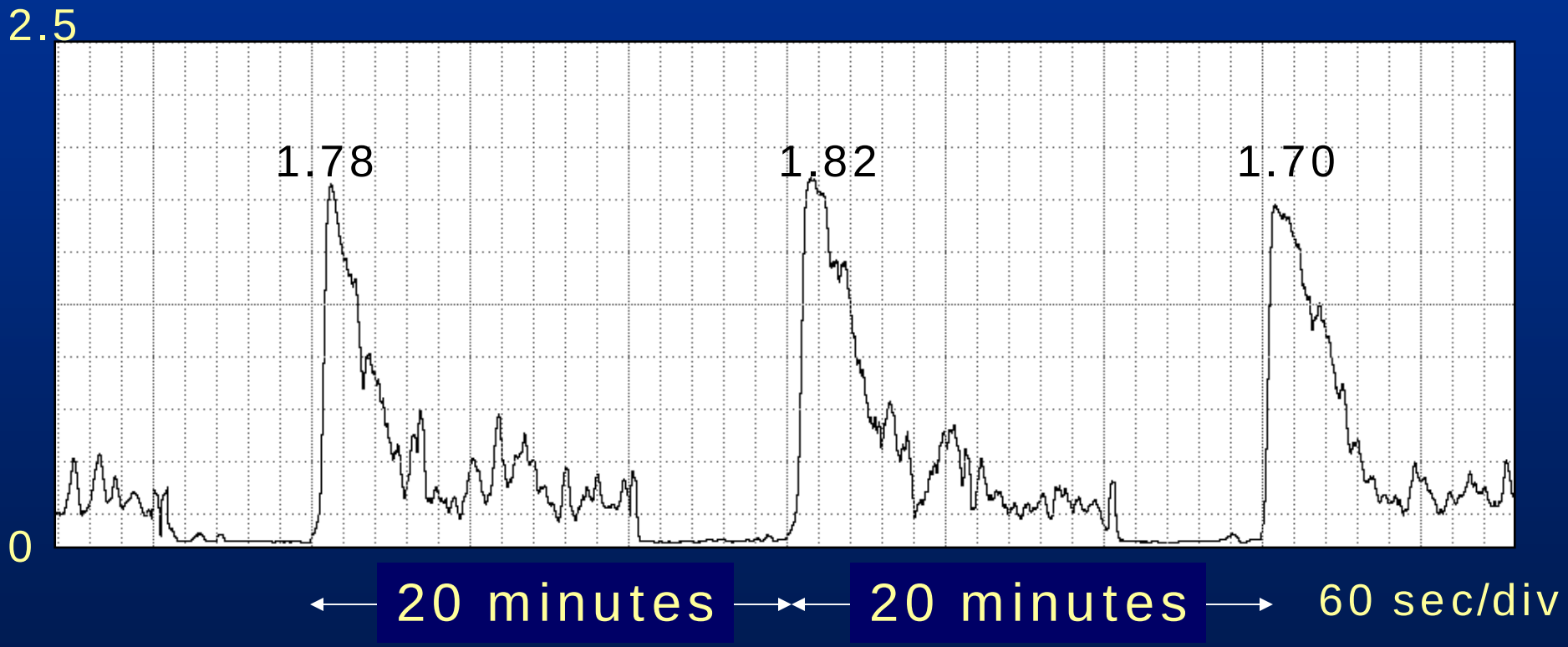


Blood Flow Occlusion

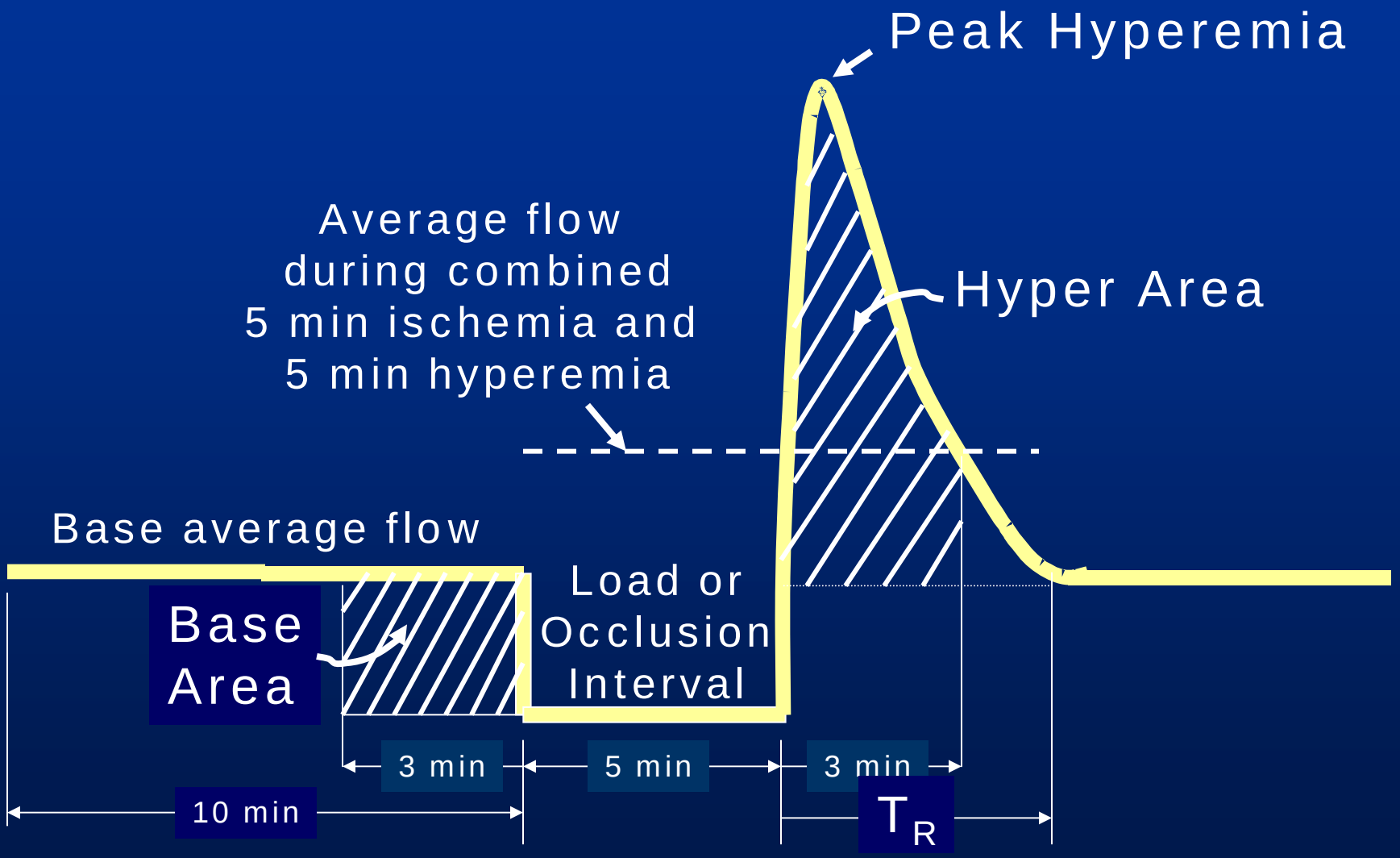



Example Responses
Subject 11
Heel
Loaded
Cuff
Inflated
2.0 volts
Subject 12
Cuff
Inflated
Heel
Loaded
5 minutes
2.0 volts

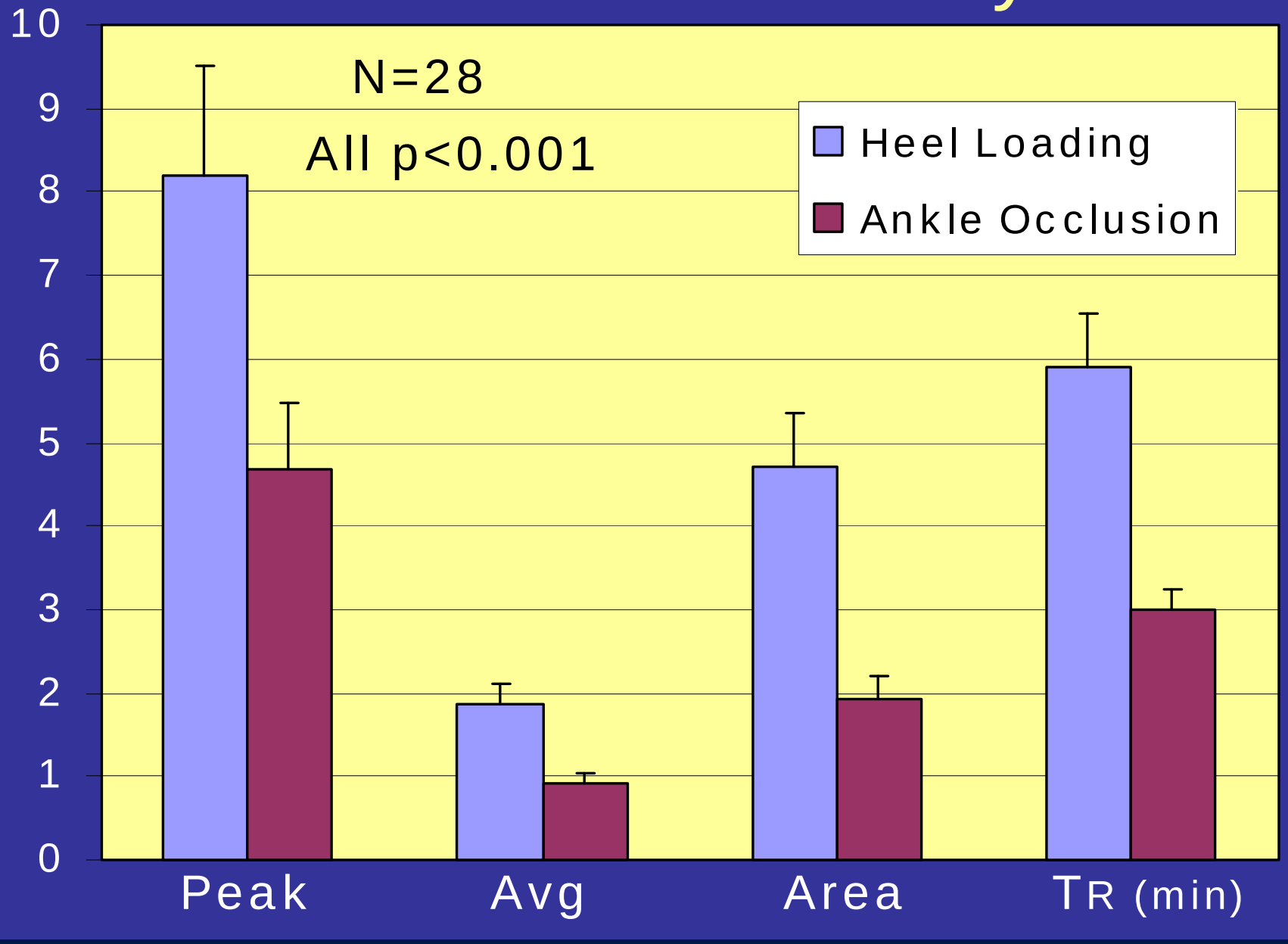
Compression Reproducibility



Response Parameters



Result Summary



Main Experimental Finding

Hyperemia after direct compression significantly exceeds that due to an equal duration of occlussive ischemia

Parameter	Heel Loading	Ankle Occlusion	p-value
Q_P/Q_B	8.20 ± 1.32	4.68 ± 0.80	<0.001
Q_{AVG}/Q_B	1.87 ± 0.23	0.92 ± 0.11	<0.001
QH_{area}/ QB_{area}	4.70 ± 0.65	1.95 ± 0.29	<0.001
T_R (seconds)	352 ± 39	181 ± 14	<0.001

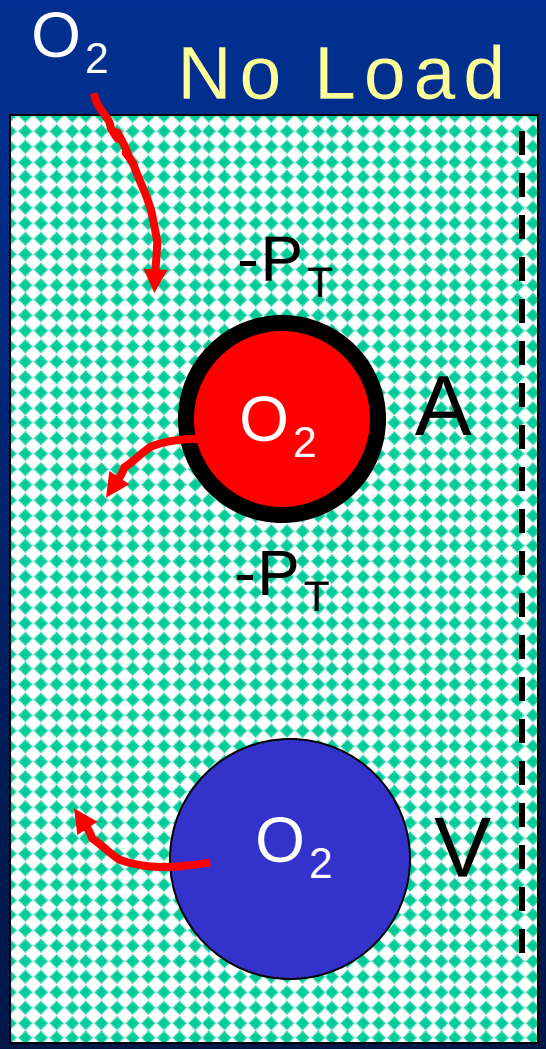
What Explains the Difference?

Factors during compression Not present with occlusion

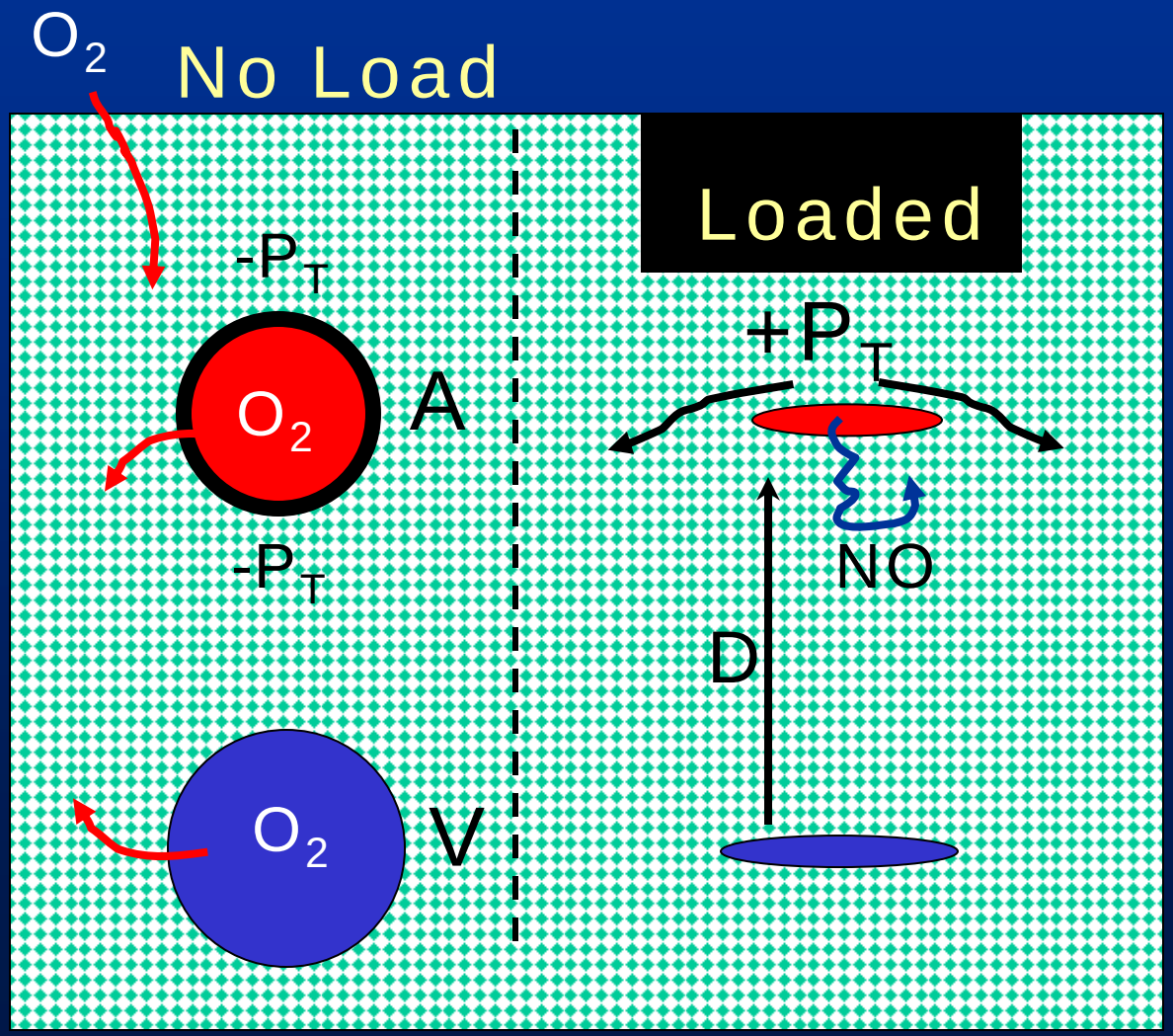
- Interstitial Fluid Squeezed Out
- Blood is expelled from Vessels
- Skin is covered and “sealed”

Combination Tends to Cause A Greater Hyperemia

Compress: Greater Hyperemia



Compress: Greater Hyperemia

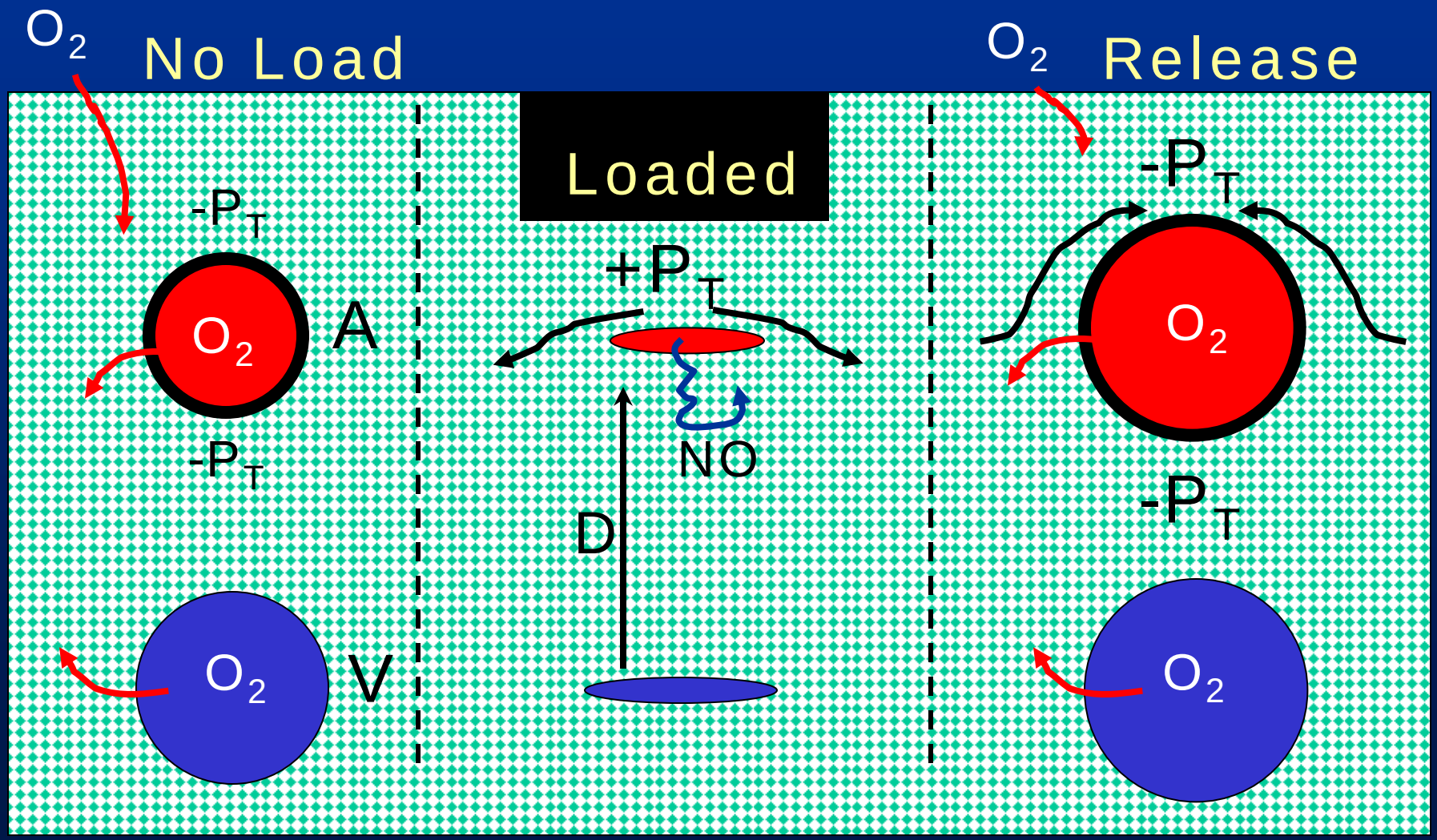


Blood Expulsion
More Distortion
More NO
Less Internal O_2
Venous D Signal

Skin “seal”
Less External O_2

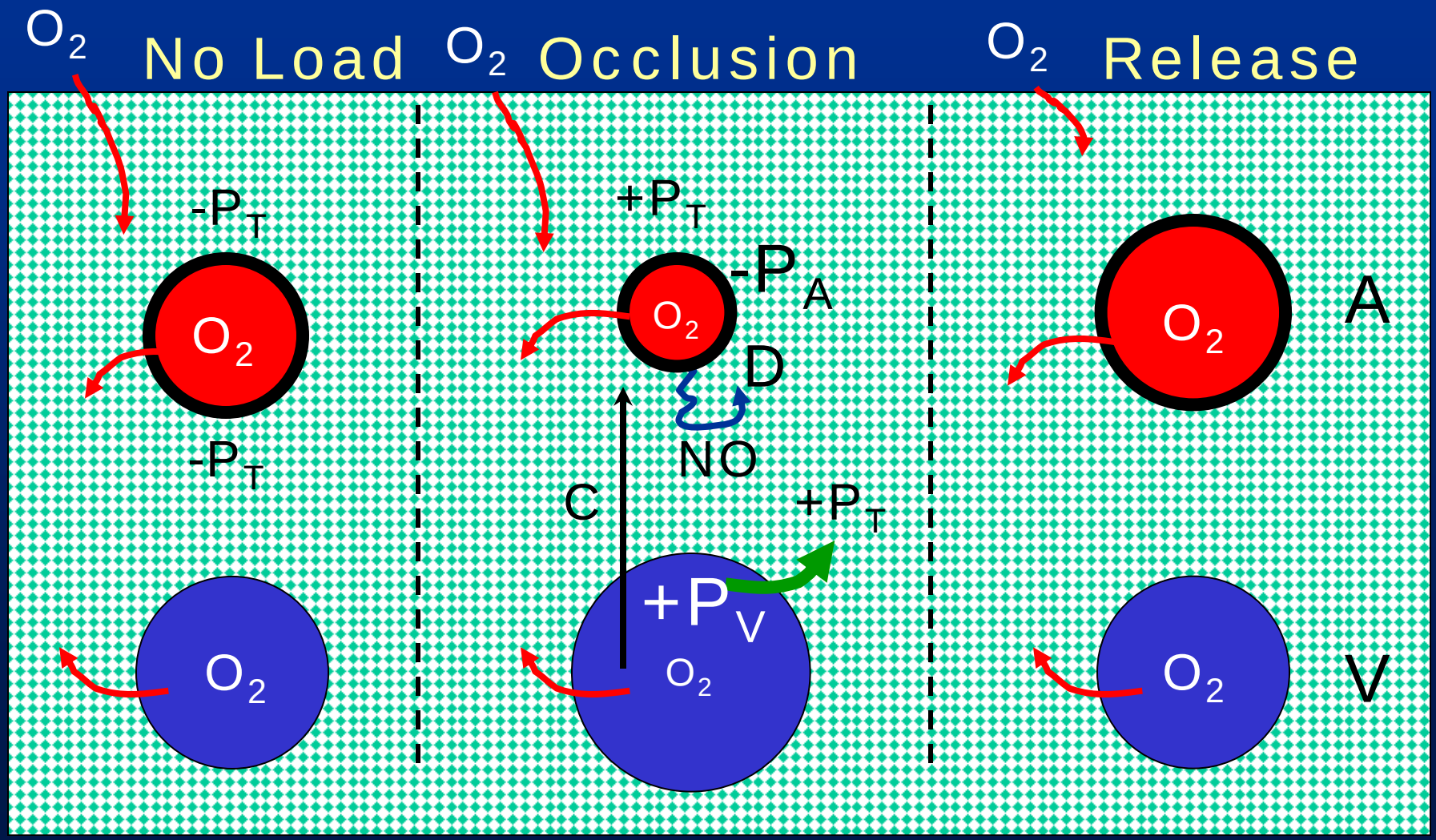
IF Squeeze
Sets-up $-P_T$

Compress: Greater Hyperemia



Occlusion Effects

Blood trapping	$+P_V$ $-P_A$	Competitive D/C



Is PCH still a good indicator of compression-related effects?

YES!

Probably better than we previously realized because it intrinsically *includes factors beyond ischemia* that likely negatively impact on pressure ulcer risk and development

BUT: Interpret With Care!

Extent of Hyperemia

- Ischemia Components
- Compression Components

Depend on Both

- Patient condition (general & local tissue)
- Compression-relief mode and parameters

Factors during compression Not present with occlusion

Interstitial fluid squeezed out

- Solid contact with vessels - greater distortion
- Sets up condition of more neg IP when released resulting in a larger transmural pressure

Blood is expelled from vessels

- Vessel flattening easier and more extensive
- Greater Arteriolar EC distortion ~ +NO
- Venous emptying mediates arteriolar dilation
- Internal residual O_2 supply diminshed

Skin is covered and “sealed”

- External O_2 supply via skin is diminished

All tend to cause greater hyperemia

Future Questions

- What are the physiological implications?
- What does it mean to surface evaluations?
- What does it mean to clinical practice?

Concept Extensions

Support surfaces that cause the least Post Compression Hyperemia (PCH) have the least negative impact on Blood Circulation

1. Less Tissue Load Magnitude

2. Less Average Loading Duration (Adequate off-loading time)

Possible Interpretations

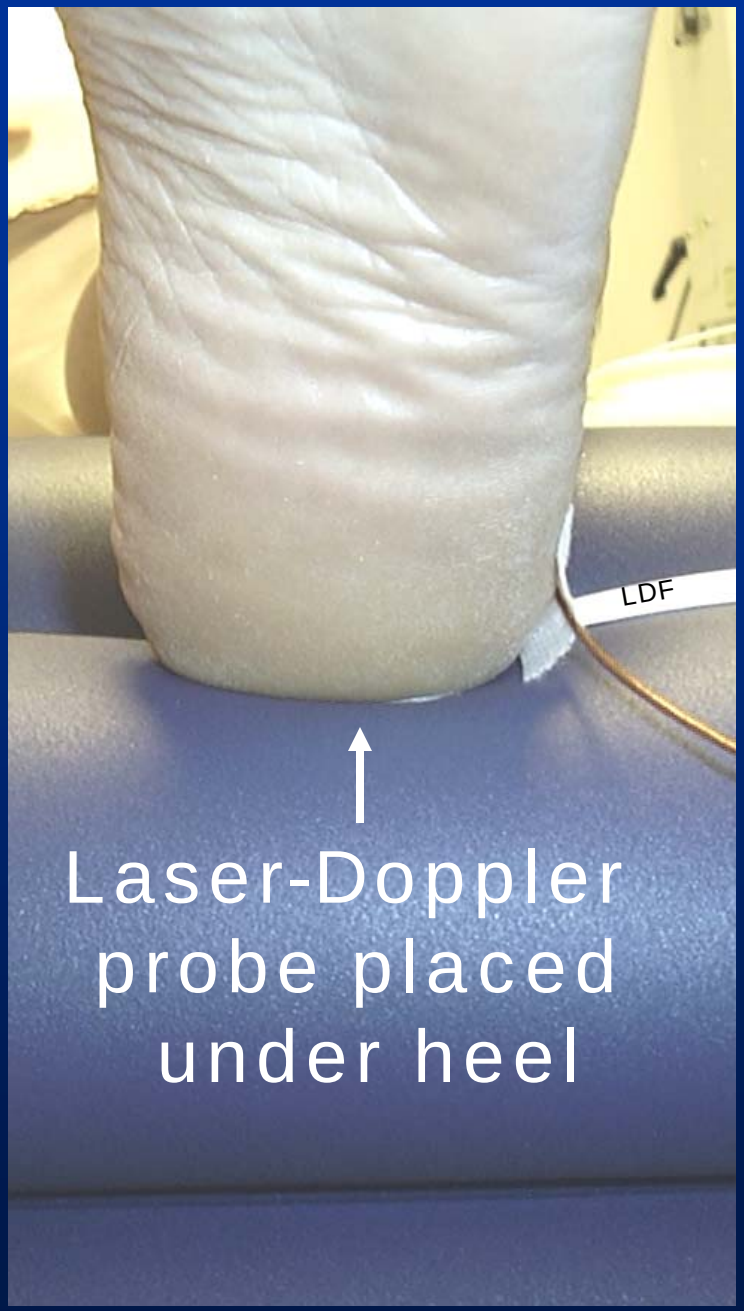
Support surfaces that cause the least Post Compression Hyperemia (PCH) have the least negative impact on Blood Circulation

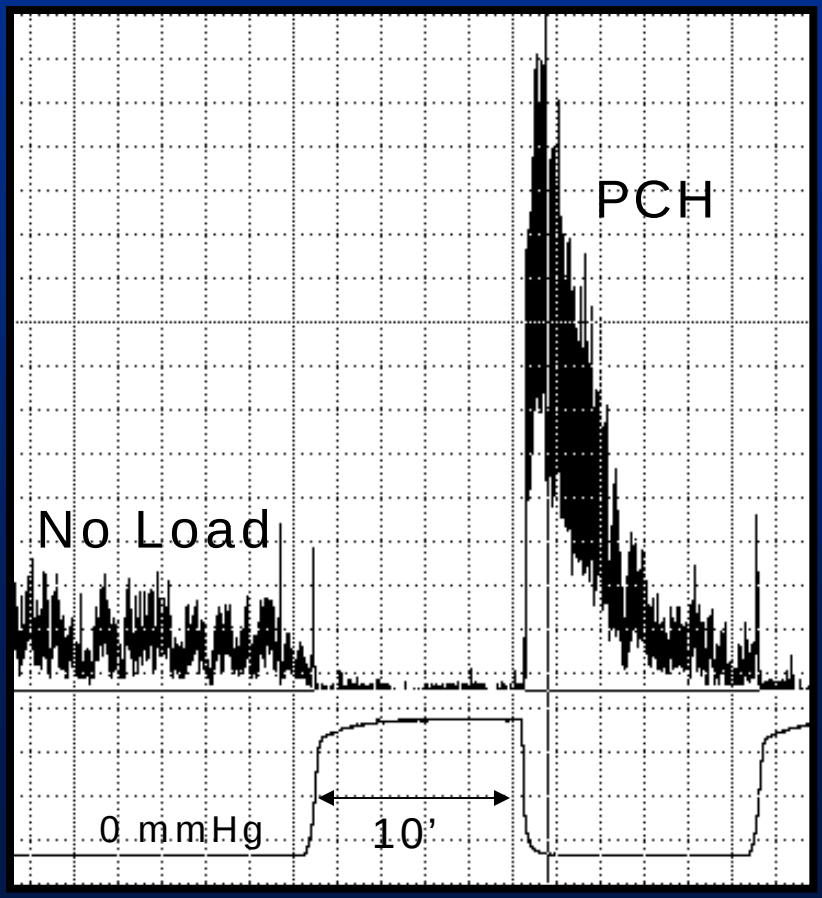
Support Surfaces that allow or induce Post Compression Hyperemia (PCH) offer a protective benefit

General Aspects Of Heel Loading Unloading and Blood Flow Measurement

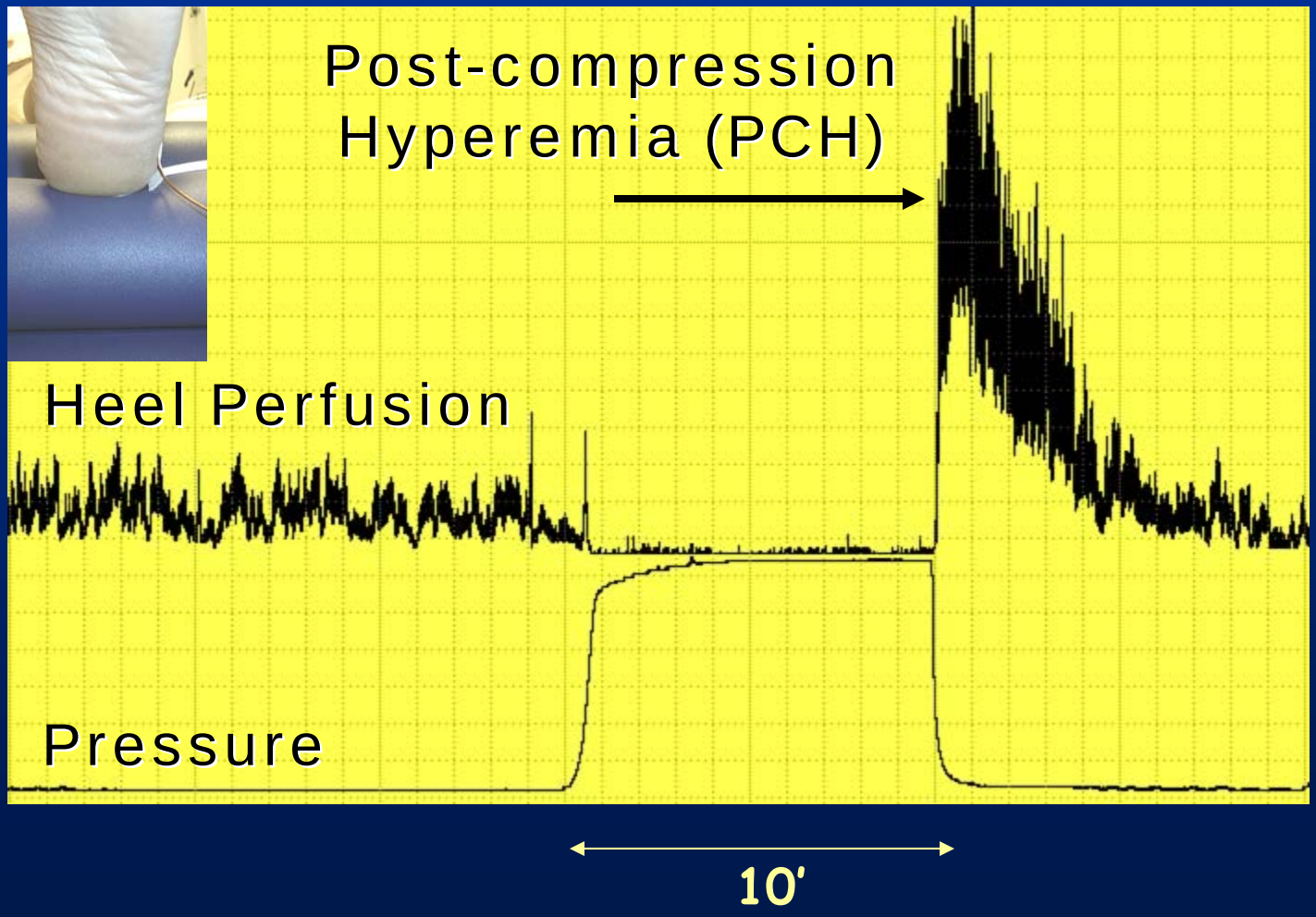
Loading and Scanning Methods

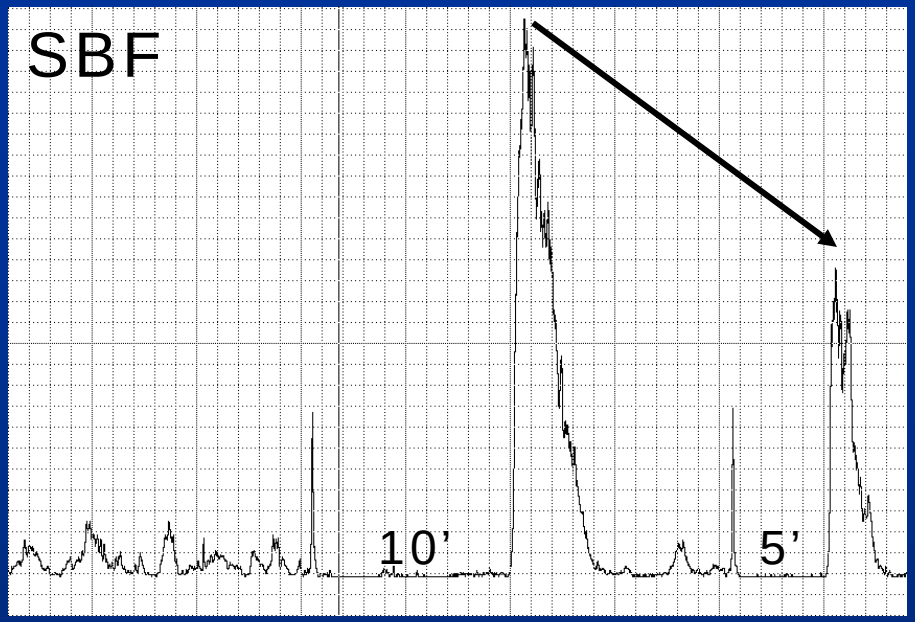
Post Compression Hyperemia -LDF





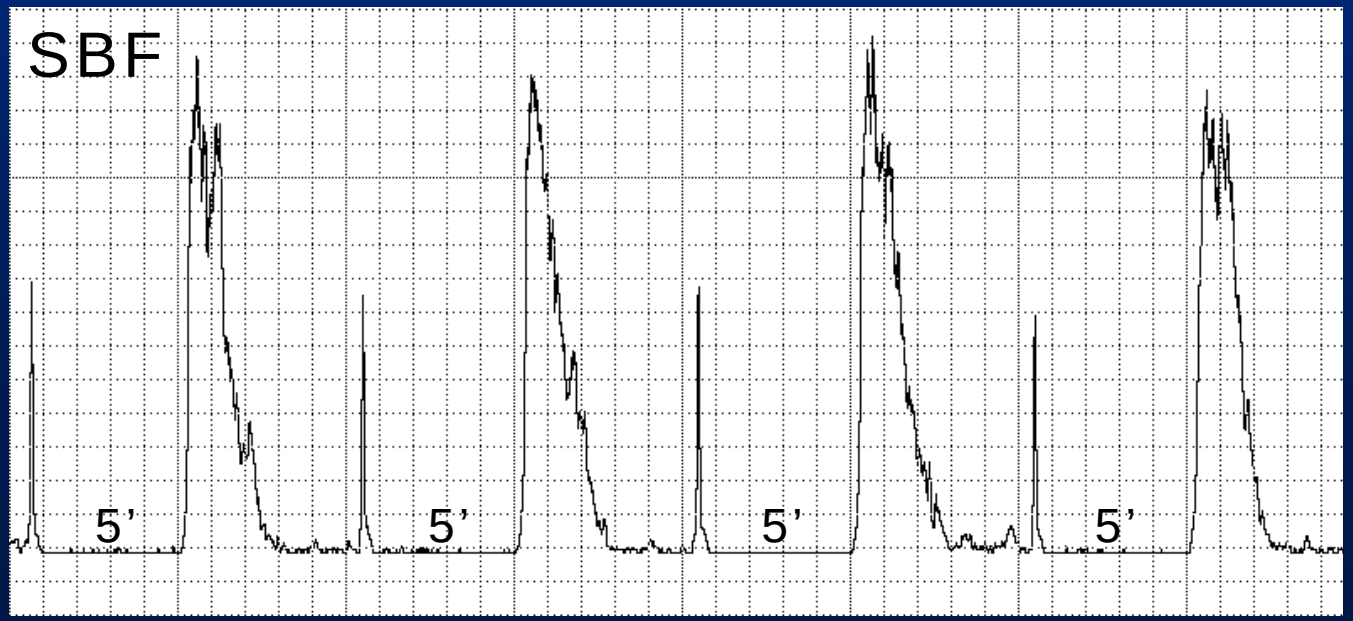
Surface Heel Loading



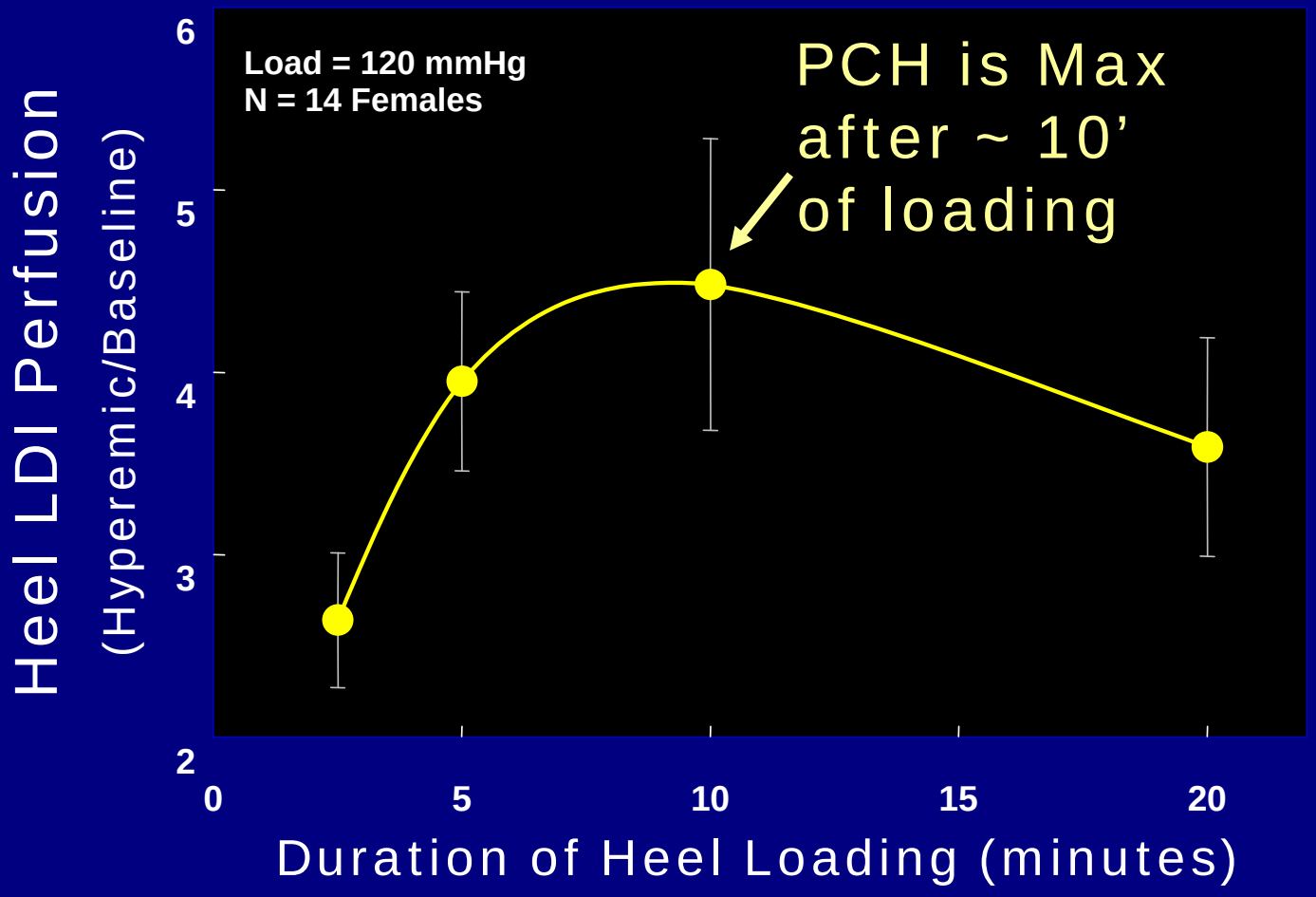


Effect of Compression Duration δ on Post Compression Hyperemia (PCH)

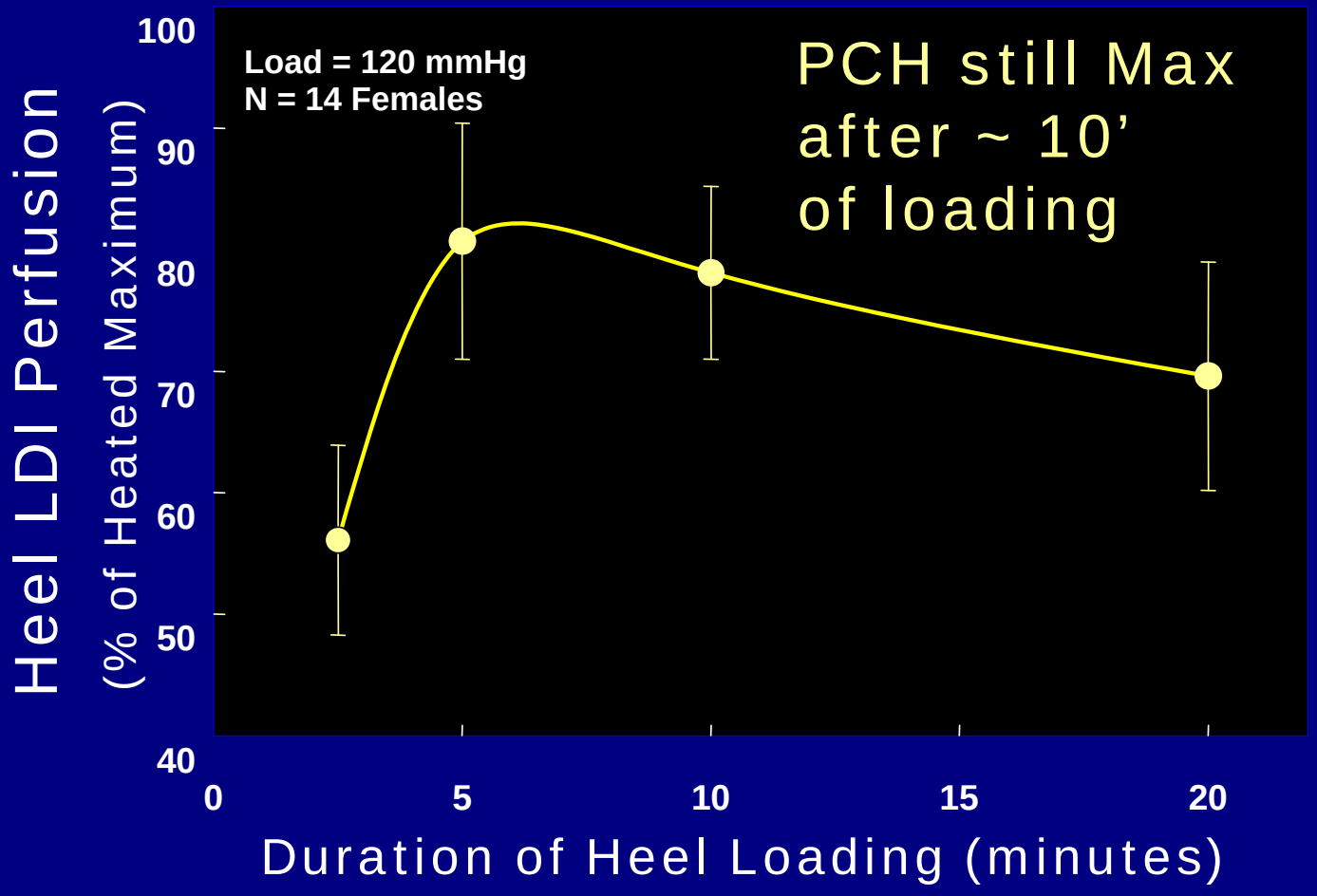
PCH Reproducibility



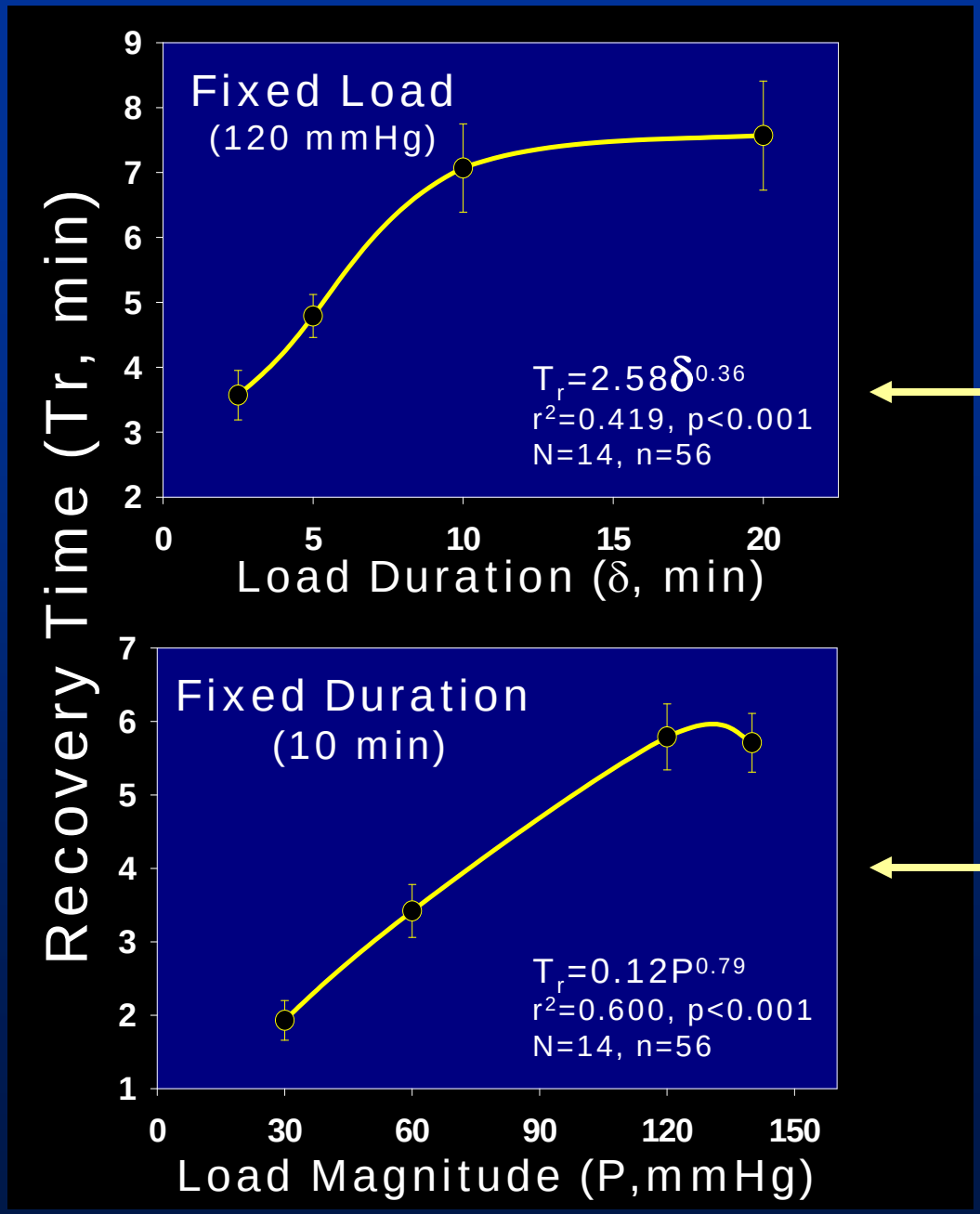
PCH Relative to Baseline



PCH Relative to Heat Max



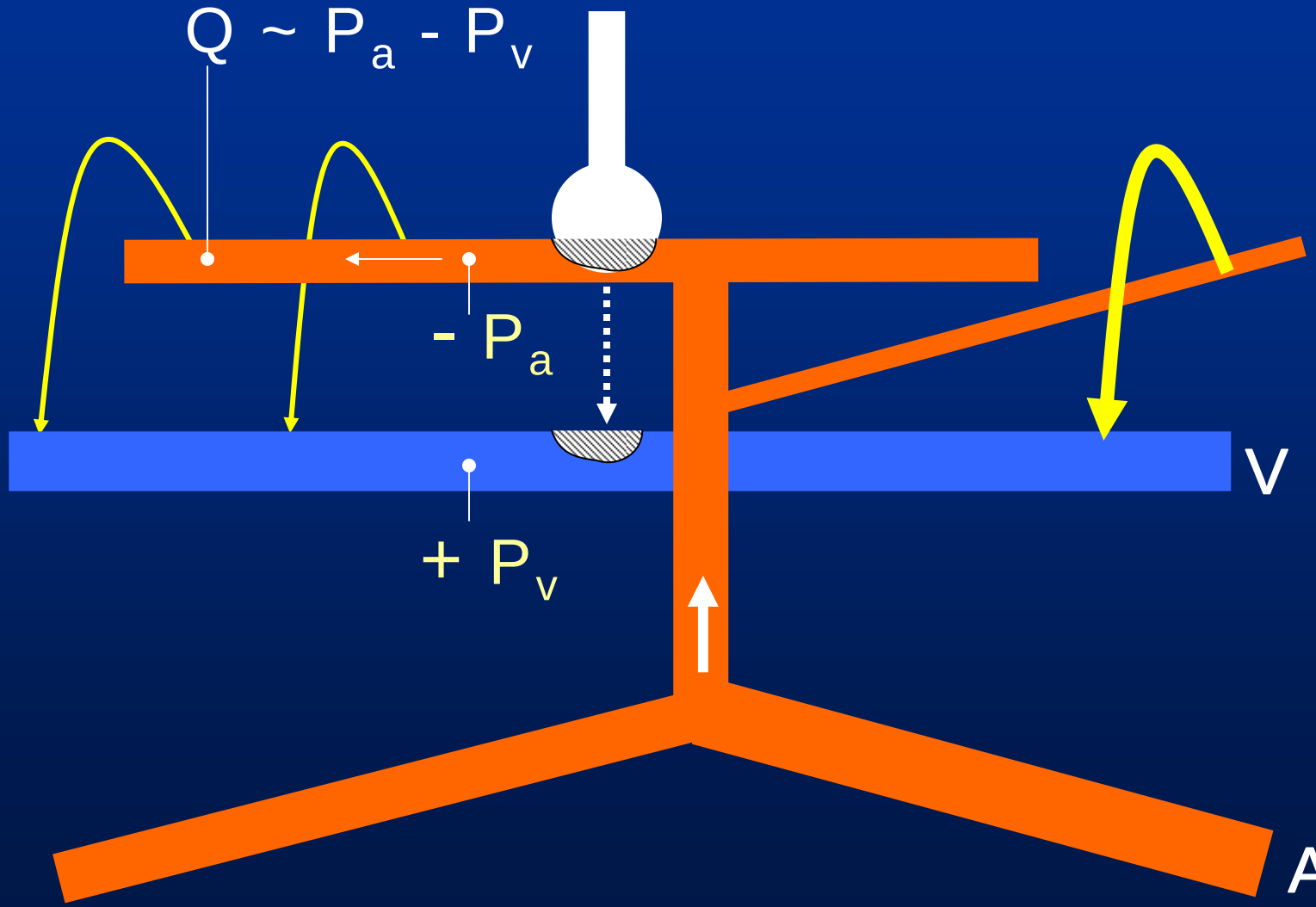
Recovery Features



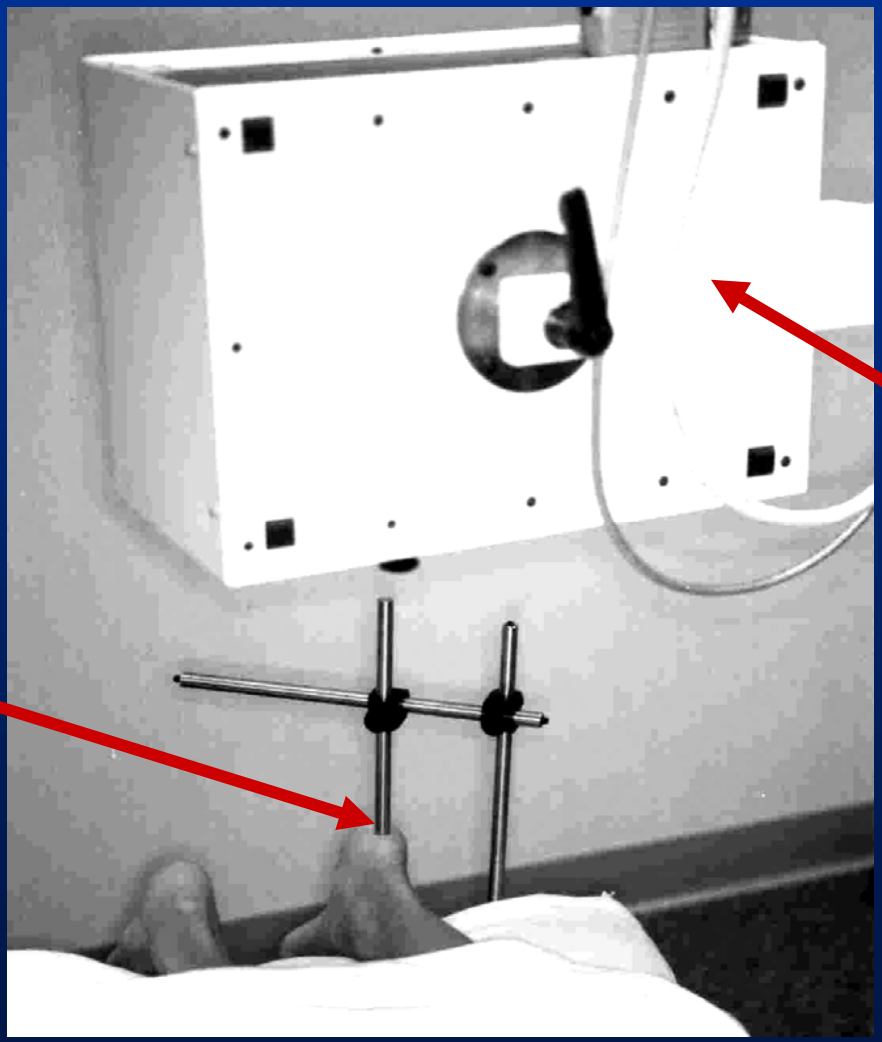
T_r increases to a load duration of about 10 minutes

T_r increases to a load magnitude of about 120 mmHg

Compression Effects on Perfusion Pressure



Local Loading with Post Load Scanning



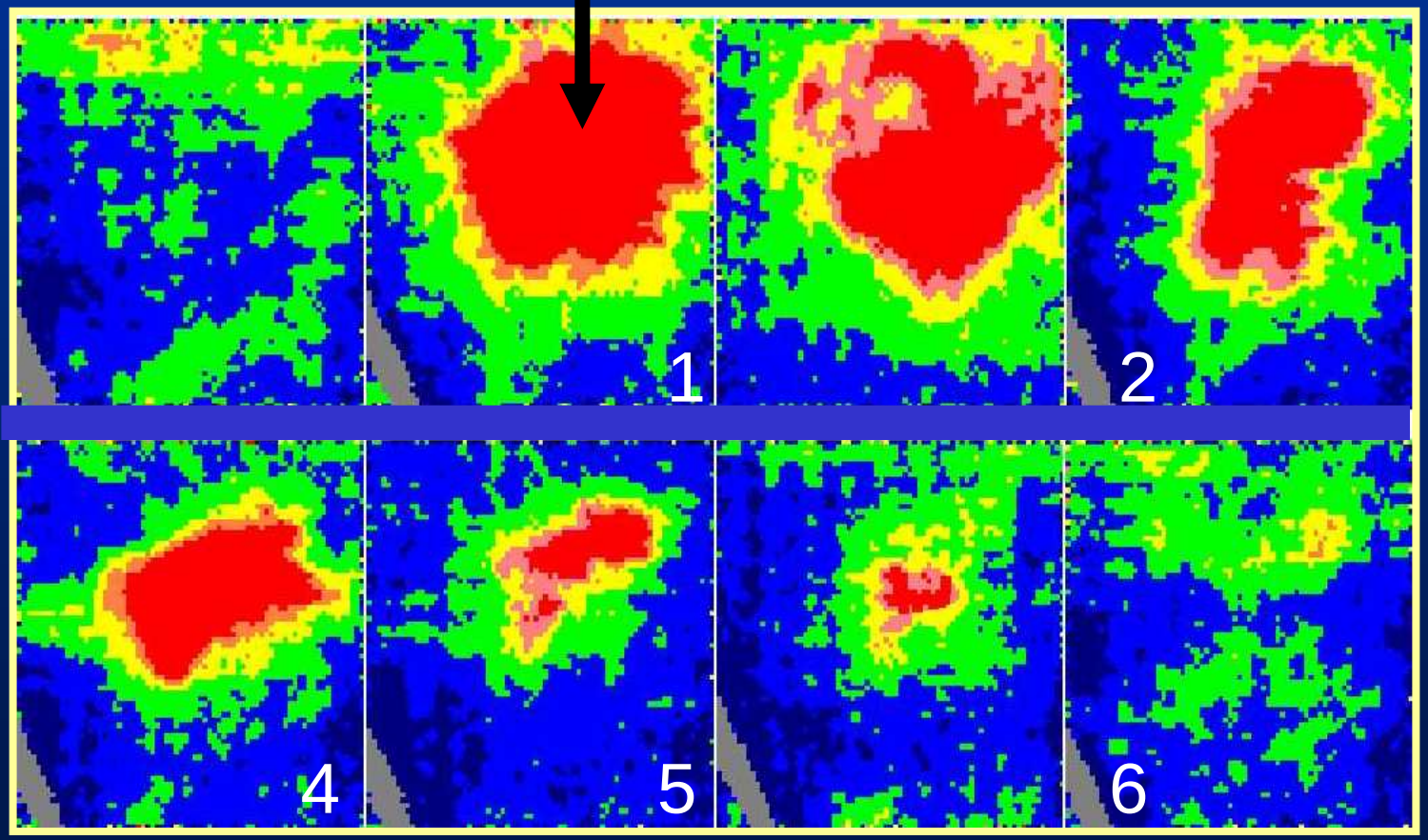
Load on Heel

Laser Doppler Imager

LDI Scan Responses

Load Center

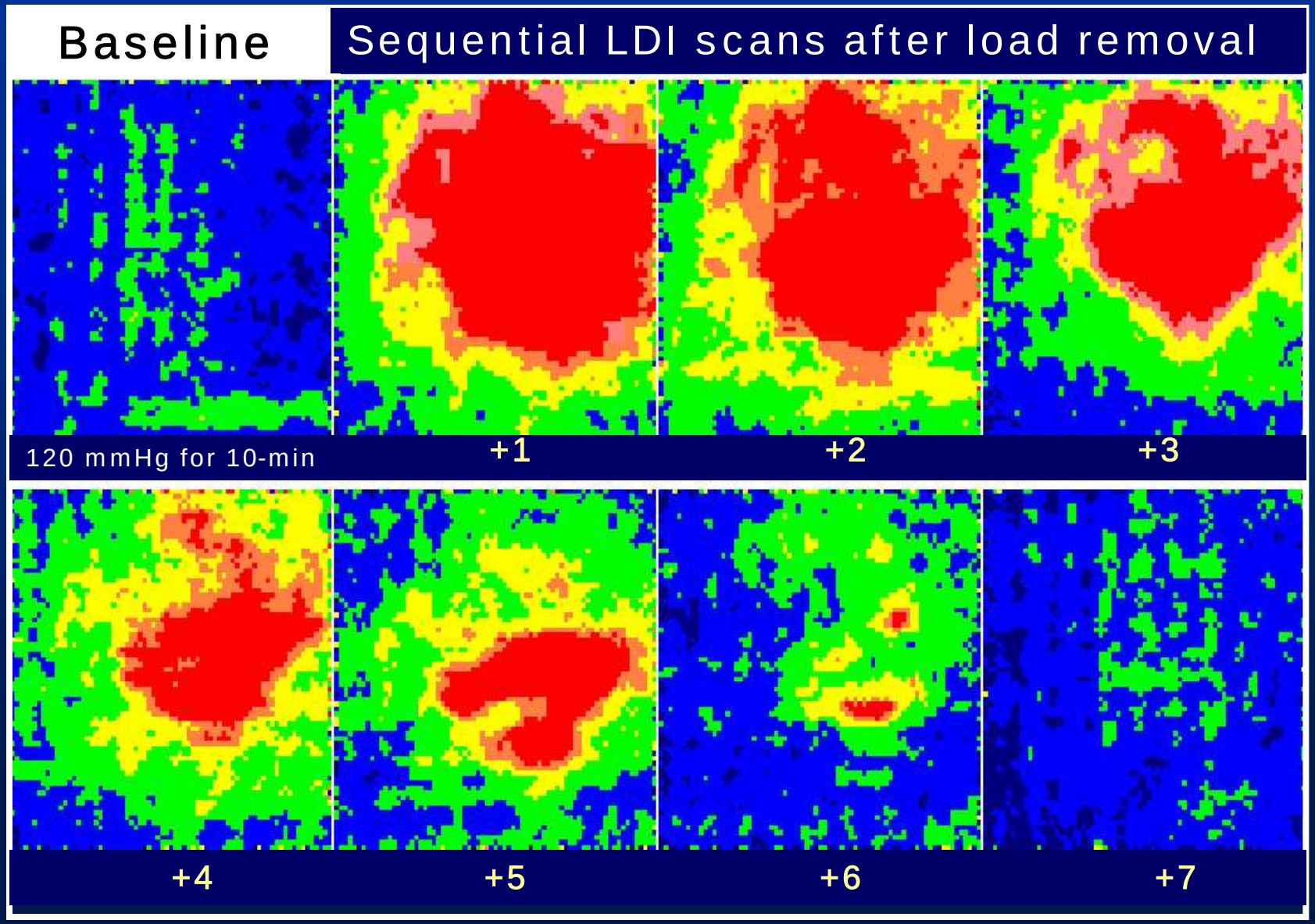
Preload



High flow (red) area contracts & flow in center gets smaller as off-load time increases

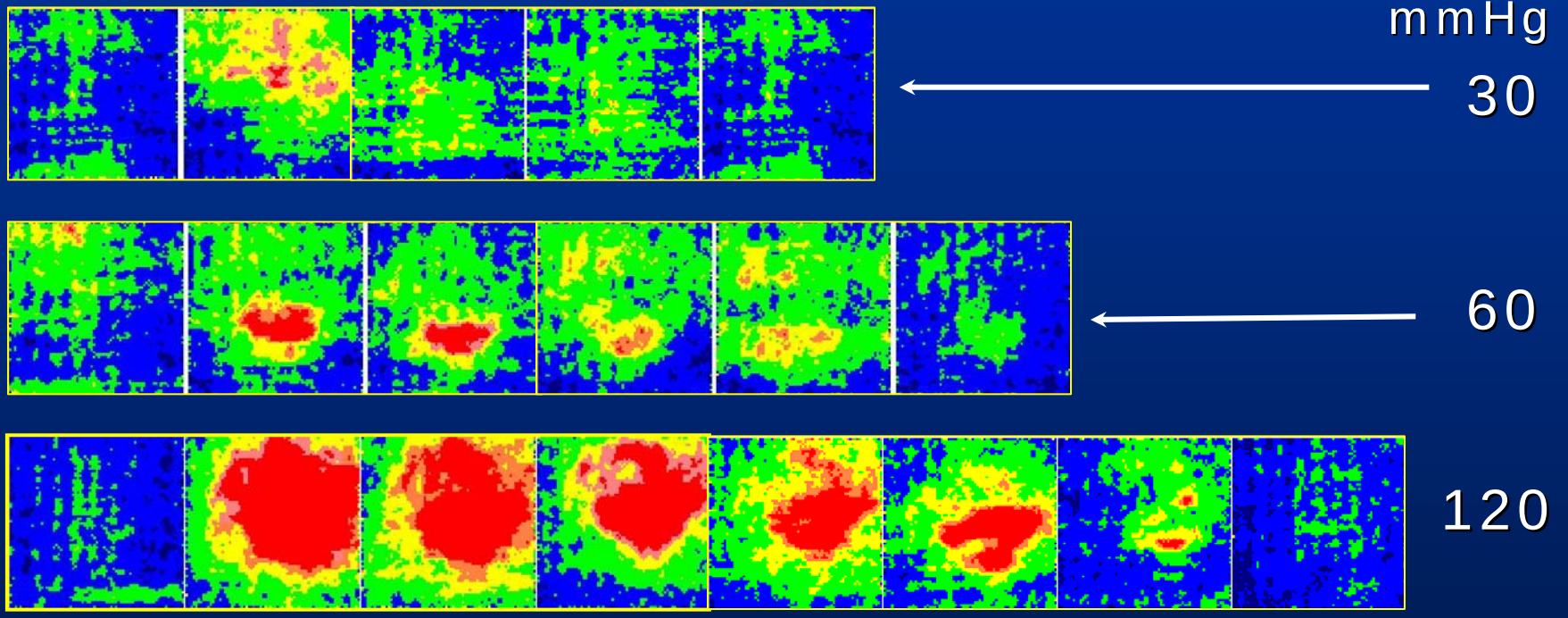
Scans at one minute intervals

LDI Scan Responses



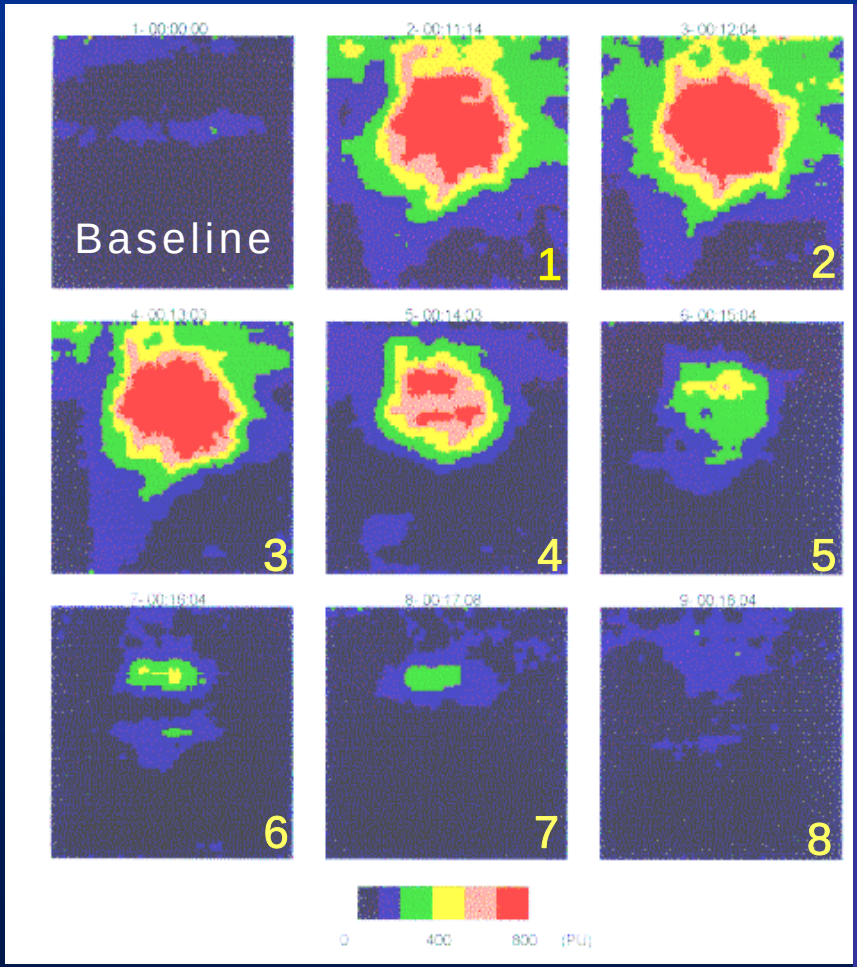
LDI Scan Responses

All 10-min Loading

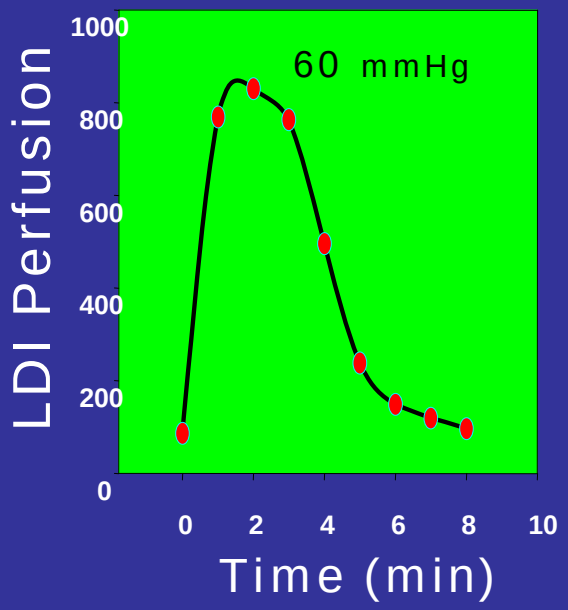


Minutes after load removal

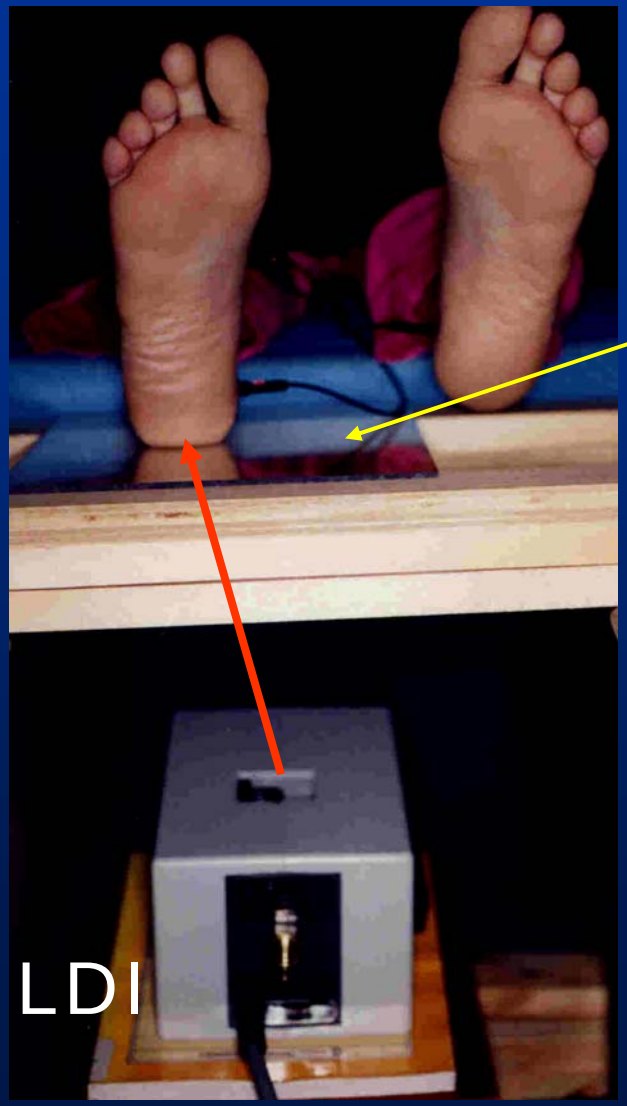
Heel Hyperemia After 10’ Local Loading with 60 mmHg



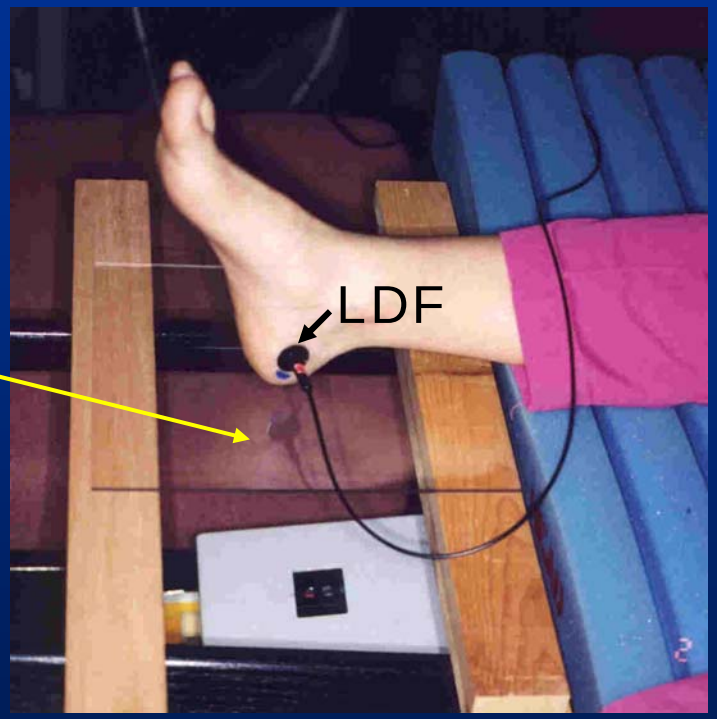
Temporal Response



Longer Duration Loading and Scans During Loading

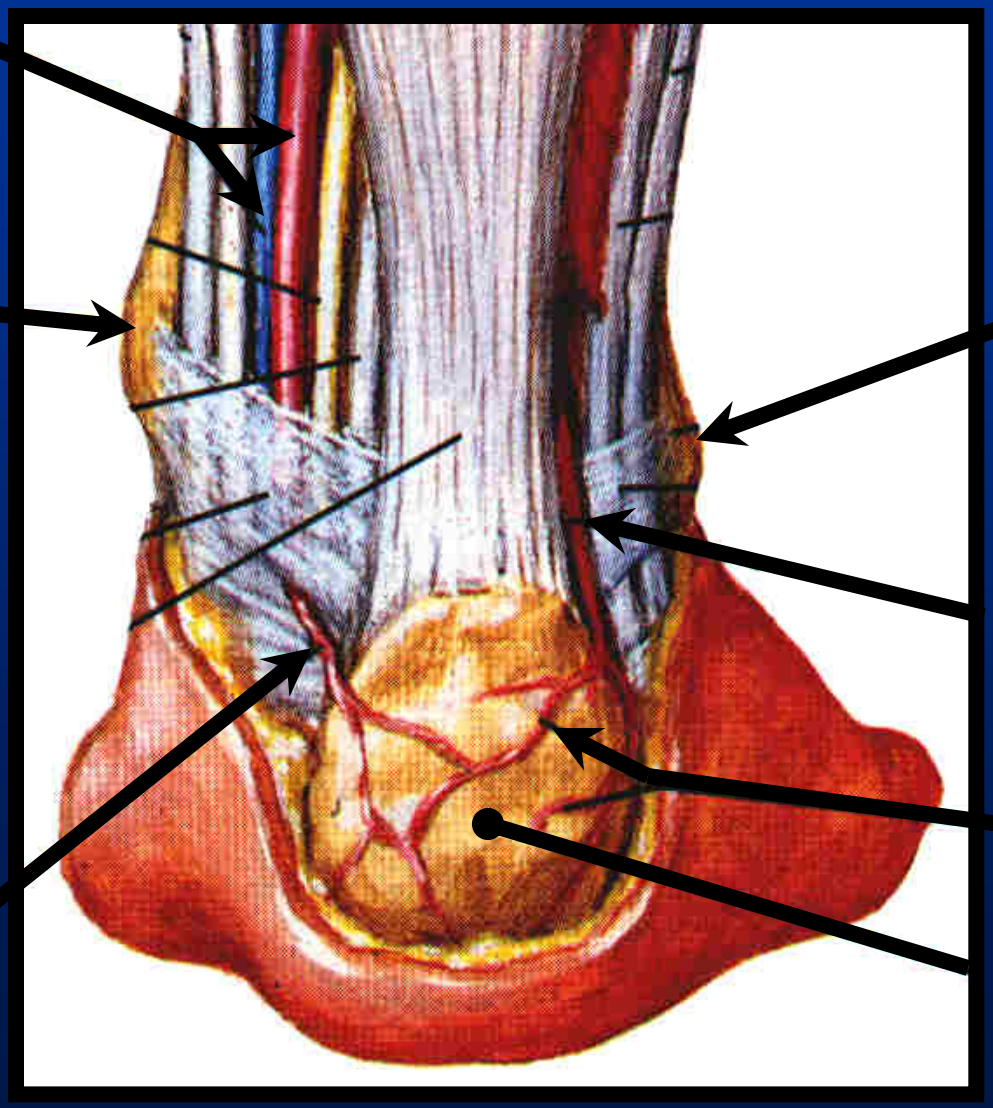


Clear Hard Surface

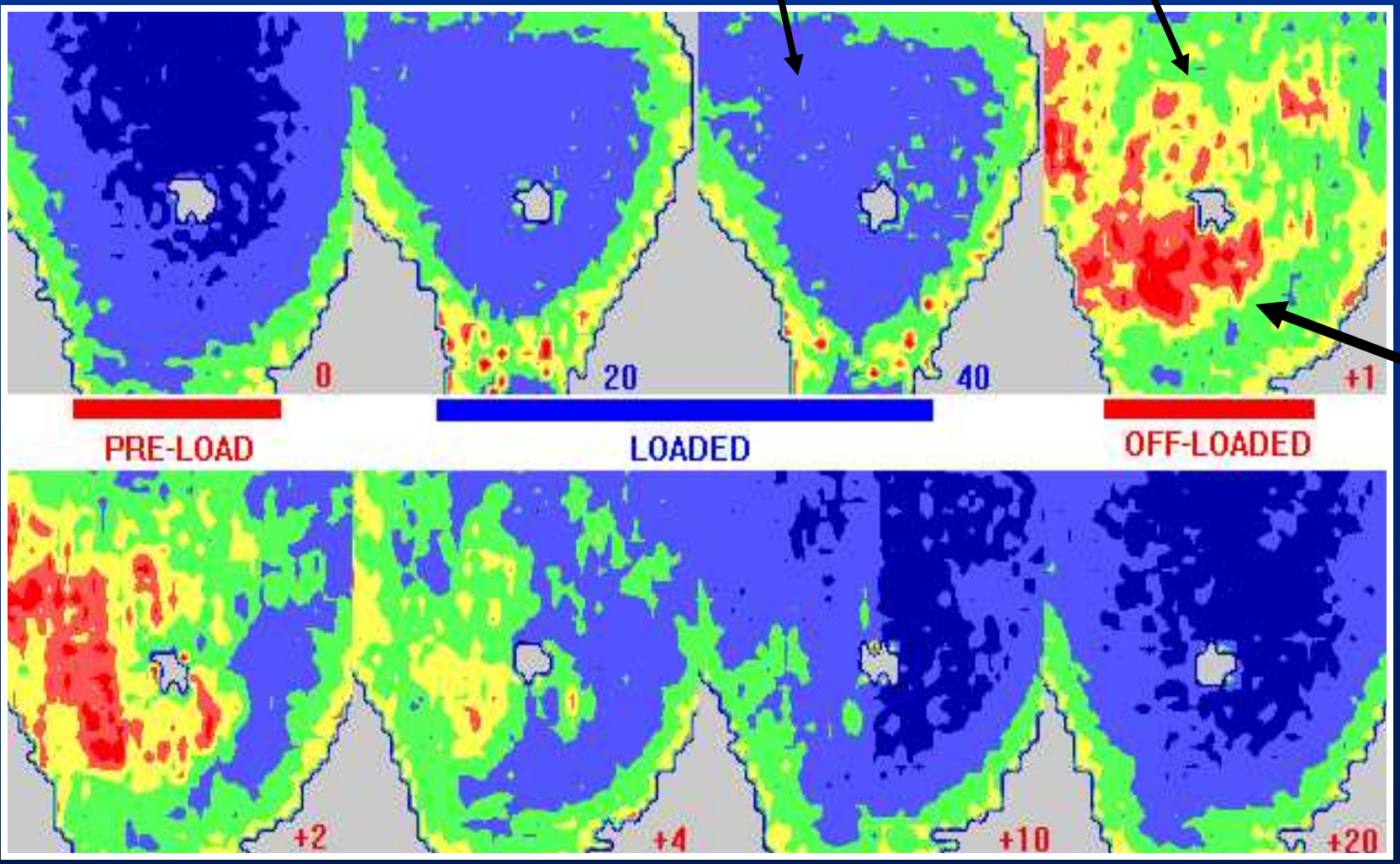


- Pre-Load: Remove Plate
- Load: 40 Minutes
- Off-Load: Hyperemia (PCH)

Heel Target


Posterior Tibial A & V
Medial Malleolus
Calcaneal Branch of PT
Lateral Malleolus
Peroneal artery
Calcaneal Branches
Calcaneal Tuberosity

Heel LDI Images Before, During and After Loading



Hyperemi

40” Supine Lying

Sequential SBF Changes