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

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Post-Compression Hyperemia
Response to Load-Induced Ischemia

Loading Pressure

Blood Flow

\[ Q_{0} \]

\[ \Delta Q_{0} \]

\[ Q_{\text{max}} \]

\[ Q_{\text{area}} \]
Basic Question

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

Why so large a Hyperemia?

Heel Load = 120 mmHg
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 Compression

Laser-Doppler Probe

Laser-Doppler probe positioned under heel
Heel Loading and Unloading

Heel Loaded

Heel Fully Unloaded

Inflated

Deflated
Blood Flow Occlusion
Example Responses

Subject 11
- Heel Loaded
- Cuff Inflated

Subject 12
- Cuff Inflated
- Heel Loaded

2.0 volts

5 minutes
Response Parameters

- **Peak Hyperemia**: 3 min
- **Hyper Area**: 3 min and 5 min
- **Base Area**: 10 min
- **Base average flow**: Average flow during combined 5 min ischemia and 5 min hyperemia
- **Load or Occlusion Interval**: 3 min, 5 min, 3 min, TR
Result Summary

N=28
All p<0.001

Heel Loading
Ankle Occlusion

Peak
Avg
Area
TR (min)
Main Experimental Finding

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

No Load

\( O_2 \)

\( -P_T \)

\( O_2 \)

A

\( O_2 \)

V
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 $-P_T$

O$_2$ No Load

Loaded

O$_2$

A

D

V

+$P_T$

NO

-$P_T$
Compress: Greater Hyperemia

No Load

Loaded

Release

O₂

-D

V

A

O₂

O₂

O₂

O₂

NO

-PT

+PT

-PT

-PT

-PT

-PT
Occlusion Effects

Blood trapping  \(+P_V -P_A\)  Competitive D/C

No Load  \(O_2\)  Occlusion  \(O_2\)  Release  \(O_2\)

\(-P_T\)  \(+P_T\)  \(-P_A\)  \(+P_T\)  \(+P_V\)

\(O_2\)  \(O_2\)  \(O_2\)  \(O_2\)  \(O_2\)
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
Future Questions

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