Neurovascular transduction in human obesity and following bariatric surgery

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BACKGROUND

- The sympathetic nervous system plays an important role in modulating vascular tone.
- Activity of the sympathetic nervous system is often reported in “bursts” of activity, which signifies activation of a given number of sympathetic axons (1).
- The transduction from a given “burst” of sympathetic activity dictates the level of constriction occurring in the vasculature (2).
- Sympathetic nervous system activity increases with obesity (3) and is reduced following bariatric surgery (4).
- In contrast, there are data to suggest plasma catecholamines remain constant (5) and systemic vascular resistance increases (6) following bariatric surgery.
- Taken together, these data suggest a disconnect between sympathetic nervous system activity and vascular tone following bariatric surgery.

AIM

To quantify sympathetic transduction in lean, obese, and bariatric surgery participants at rest by evaluating relationships between muscle sympathetic nerve activity (MSNA) and its effect on diastolic blood pressure (DBP).

HYPOTHESES

- Obese individuals will exhibit greater sympathetic-neurovascular transduction when compared to lean individuals.
- Neurovascular transduction will be lowest in individuals that have undergone bariatric surgery compared to lean and obese individuals.

METHODS

- Participants: Cross-sectional study in three groups of young, otherwise healthy female research participants (Lean n=8; Obese n=8; Post-bariatric surgery n=8).
- Instrumentation: Heart rate (electrocardiography), arterial blood pressure (finger photoplethysmography), and MSNA (peroneal nerve, microneurography).

RESULTS

Figure 2: Neurovascular transduction. We assessed the summed MSNA burst area in a two cardiac cycle lag preceding a change in DBP. Transduction was quantified as the slope of the relationship between MSNA burst area and a change in DBP at a given lag.

Figure 3: Determining cardiac lag. The lag that produced the greatest transduction (stepest slope) for each group was used to quantify transduction. For each group this was 4-6 cardiac cycles following a burst.

Figure 4: Resting muscle sympathetic nerve activity (MSNA) in lean, obese, and individuals following bariatric surgery. Data are reported as Mean±SEM from n=8 per group. MSNA burst frequency (A: Main effect of group, P<0.09) and burst incidence (B: Main effect of group, P=0.08) tended to be greater in obese women when compared to lean women and women following bariatric surgery.

Figure 5: Neurovascular transduction in lean, obese, and individuals following bariatric surgery. Data are reported as Mean±SEM from n=8 per group: (A) Group averages, (B) Individual data. Transduction of MSNA into DBP was not different between groups (Lean 0.124±0.020, Obese 0.127±0.034, Bariatric 0.147±0.024 Δ mmHg%; s: P=0.82).

CONCLUSIONS

These data support an increase in sympathetic activity in obese women that is attenuated following bariatric surgery. However, any obesity-related changes in sympathetic activation within the skeletal muscle of young women occur independent of changes in neurovascular transduction. Taken together, neurovascular transduction is not altered with obesity and is unlikely to contribute to increases in systemic vascular resistance following bariatric surgery.

REFERENCES


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