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Change in Stroke Volume by Iced Temperature Injectate for Thermodilution Cardiac Output Determination

To the Editor:—Cold temperature injectate has been recommended for thermodilution cardiac output (TDCO) determination to enhance the signal-to-noise ratio, but several clinical studies have reported transient reductions in heart rate and blood pressure with the use of iced temperature injectate. ¹⁻³ Stroke volume also may be altered but has not been studied to date. We examined the relative changes in heart rate and stroke volume during room temperature and iced temperature TDCO measurements.

After institutional approval and informed consent, six adult patients in the intensive care unit who required pulmonary artery catheterization were studied. TDCO measurements were obtained by manual injection of 10 ml D₅W through a closed delivery system. Heart rate (electrocardiogram) and noninvasive beat-to-beat stroke volume (SORBA CIC-1000 impedance cardiograph, Milwaukee, WI) were monitored during the injection of both iced (≤6° C) and room temperature (23° C) injectate. The order of injectate temperature was alternated with each patient. At each injectate temperature, two injections, made at 3-min intervals during the onset of an end-expiratory breathhold, were used to generate the cardiac output measurements; however, if the two measurements were not within 10% of each other, a third injection was performed. Hemodynamic values measured at the onset of injectate were used as baseline. Individual maximal responses in heart rate and stroke volume were selected during each TDCO and compared to baseline using Student's paired t tests. Two-way analysis of variance for repeated measures was used to determine the significance of hemodynamic changes. Values are expressed as mean \pm SEM. Results were considered significant at P ≤ 0.05 .

TDCO using iced temperature injectate (8.6 \pm 0.5 l/min) did not differ from that measured using room temperature injectate (8.6 \pm 0.8 l/min). Table 1 summarizes maximal hemodynamic responses. Iced temperature TDCO was followed by a 5 \pm 2% maximal reduction in heart rate ($P \ge 0.05$) and a 30 \pm 8% maximal increase in stroke volume ($P \le 0.05$). These responses were not observed during the use of room temperature injectate.

Our data showed a significant increase in stroke volume when iced injectate was used for cardiac output determination. However, unlike several other studies, ¹⁻³ a reduction in heart rate was not seen. Heart rate slowing during iced temperature TDCO measurements has been attributed to local cooling of the sinoatrial node rather than a reflexmediated autonomic mechanism. ¹ Surgically induced autonomic

Table 1. Peak Hemodynamic Responses during Room Temperature (23° C) and Iced Temperature (≤6° C) Thermodilution Cardiac Output Measurement

| | Baseline | Peak Response | Time (s) to Peak Response |
|--------------------|-------------|------------------|------------------------------|
| Heart rate (b/min) | | | - |
| Room temperature | 88 ± 8 | 86 ± 8 | 2.5 ± 0.4 |
| Iced temperature | 88 ± 8 | 83 ± 9 | 3.8 ± 0.7 |
| Stroke volume (ml/ | | | |
| min) | | | |
| Room temperature | 83 ± 16 | 94 ± 11 | 3.8 ± 0.9 |
| Iced temperature | 85 ± 15 | 111 ± 20* | 4.3 ± 0.8 |
| | | | |

Values are mean ± SEM.

blockade does not prevent iced temperature bradycardia, and room temperature injectate delivered in a manner similar to iced temperature injectate does not elicit a comparable reduction in heart rate. Our observed augmentation in stroke volume during iced temperature TDCO has not been previously documented in a clinical setting. The increase in stroke volume may be due to a prolonged diastole, resulting in an increase in cardiac filling (Frank Starling mechanism). We conclude from our limited study that iced temperature injectate has a minor effect on the heart, and if a slowing of heart rate does occur, cardiac output may be unaffected secondary to an augmentation in stroke volume. Further studies are needed to investigate these preliminary findings.

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^{*} P

0.05 compared with baseline value.

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The Attending Anesthesiologist Meets the Patient

Did you ever have an allergic reaction to medication? Can you open your mouth wide for me? And tilt back your head? Any capped teeth? Have you had anything to eat or drink today? Are you warm enough out here? Do you have any questions about your anesthesia?

Did you ever have a reaction to a poem?
Can you look at my eyes and see me?
And my children?
Any grant deadlines this week?
Have you had anything to drink or inject today?

Are you warm enough—in there?

Do you have any questions about my anesthesia?

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More on Inspiratory Stridor

To the Editor:—Recently Sukhani et al.\(^1\) described a patient with inspiratory stridor in the recovery room presumably associated with paradoxical vocal cord motion. I have also observed inspiratory stridor (presumably the same paradoxical vocal cord motion) to occur in a nonclinical setting and in the absence of depressant drugs or alcohol.

Case Report

A 67-yr-old man suffered from a chronic cough for about 18 months coinciding with the period when his wife was dying of cancer. He was otherwise asymptomatic, and because the cough did not disturb his sleep and a medical workup was negative, the cough was believed to be psychogenic in origin.

The event of inspiratory stridor was precipitated by a joke, the

punch line of which coincided with the beginning of the swallowing reflex as this individual was trying to take a vitamin tablet. The man involuntarily spit out the tablet in a fit of laughter. As the laughter of the others present subsided, I became aware that he was leaning forward, unable to speak, and able to inspire only a small amount of air with each breath, judging from the effort, chest motion, and quality of the stridor. The respiratory distress resolved slowly over about 15 min without intervention other than reassurance. The victim of this event stated that it had been quite horrifying, similar to drowning or being strangled.

I had seen this type of inspiratory stridor in the recovery room on several occasions and had presumed it to have a functional rather than anatomic basis, because it invariably resolved without sequelae. The above-mentioned incident shows that it also can occur in the absence of depressant drugs and muscle relaxants. A sufficiently re-