Tactile stimulation of the oropharynx elicits sympathoexcitation in conscious humans

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Muller MD, Mast JL, Cui J, Heffernan MJ, McQuillan PM, Sinoway LI. Tactile stimulation of the oropharynx elicits sympathoexcitation in conscious humans. J Appl Physiol 115: 71–77, 2013. First published April 18, 2013; doi:10.1152/japplphysiol.00197.2013.—Tactile stimulation of the oropharynx (TSO) elicits the gag reflex and increases heart rate (HR) and mean arterial pressure (MAP) in anesthetized patients. However, the interaction between upper-airway defense reflexes and the sympathetic nervous system has not been investigated in conscious humans. In Experiment 1, beat-by-beat measurements of HR, MAP, muscle sympathetic nerve activity (MSNA), and renal vascular resistance (RVR) were measured during TSO and tactile stimulation of the hard palate (Sham) in the supine posture. In Experiment 2, TSO was performed before (pre) and after (post) inhalation of 4% lidocaine via nebulizer. Rate pressure product (RPP) was determined. Compared with Sham, TSO elicited the gag reflex and increased RPP [absolute change (Δ)36 ± 6 vs. 17 ± 5%, MSNA (Δ122 ± 39 vs. 19 ± 19%), and RVR (Δ55 ± 11 vs. 4 ± 4%). This effect occurred within one to two cardiac cycles of TSO. The ΔMAP (12 ± 3 vs. 6 ± 1 mmHg) and the ΔHR (10 ± 3 vs. 3 ± 3 beats/min) were also greater following TSO compared with Sham. Lidocaine blocked the gag reflex and attenuated increases in MAP (Δpre: 16 ± 2; Δpost: 5 ± 2 mmHg) and HR (Δpre: 12 ± 3; Δpost: 2 ± 2 beats/min) in response to TSO. When mechanically stimulated, afferents in the oropharynx not only serve to protect the airway but also cause reflex increases in MSNA, RVR, MAP, and HR. An augmented sympathoexcitatory response during intubation and laryngoscopy may contribute to perioperative cardiovascular morbidity and mortality.

sympathetic nervous system; afferent; pharynx; local anesthesia; blood flow

MANY AIRWAY DEFENSE REFLEXES (e.g., cough reflex, gag reflex, swallow reflex) originate in the mouth, pharynx, and/or larynx (3, 7, 25). These upper-airway reflexes are operable under physiological conditions and effectively route food and air to the proper anatomical locations. During an acute perturbation, such as choking or coughing, blood pressure (BP) homeostasis is likely to be affected. Tactile stimulation of the oropharynx (TSO) is also a potent stimulus that increases heart rate (HR), BP, and rate pressure product (RPP) in humans. Specifically, endotracheal intubation, laryngoscopy, and bronchoscopy are clinical procedures that increase myocardial oxygen demand (24, 45, 47, 48). Considering that many patients are at risk for cardiac ischemia and arrhythmia during these procedures (29, 30, 35, 41), attenuating HR, BP, and RPP in response to TSO has received much attention (1, 8, 16, 47, 53, 55). However, the basic physiology is unclear, because patients in these cited experiments were premedicated, sedated, and/or under general anesthesia when TSO was applied.

A number of physiological stressors (i.e., orthostasis, exercise, hypoxemia) elicit sympathoexcitation, which serves to redistribute blood flow and maintain perfusion to critical organs. Studies in anesthetized patients have shown that muscle sympathetic nerve activity (MSNA) and BP increase rapidly and robustly during intubation and laryngoscopy (9, 11, 43), providing evidence that TSO elicits vasoconstriction within skeletal muscle. Another vascular bed—the kidney—receives ~20% of cardiac output at rest, and alpha-adrenergic renal vasoconstriction occurs in responses to physiological stress (5, 27, 28, 40). In dogs, renal sympathetic nerve activity and BP both increase during intubation (42), but the renal vascular responses to TSO in humans are unknown. This may be clinically relevant, considering that perioperative renal failure (seemingly due to reductions in renal blood flow) is not uncommon.

The purpose of this study was to characterize the integrated neurovascular responses to TSO in conscious, unmedicated humans (Experiments 1) and to block this response using local anesthesia of the upper airway (Experiments 2 and 3). In these experiments, we tested two separate but related hypotheses. First, TSO will cause greater changes in BP, HR, MSNA, and renal vascular resistance (RVR) compared with tactile stimulation of the hard palate (Sham). Second, inhalation of 4% topical lidocaine prior to (pre) TSO will block the gag reflex and attenuate increases in HR and BP. Herein, we demonstrate that stimulation of mechanically sensitive afferents in the oropharynx elicits an increase in MSNA, along with acute hypertension, tachycardia, and renal vasoconstriction; these effects can be abolished with local anesthesia of the upper airway.

METHODS

All study protocols were approved in advance by the Institutional Review Board of the Penn State Milton S. Hershey Medical Center and conformed to the Declaration of Helsinki. A total of 23 healthy, unmedicated, young (range 22–33 years) subjects volunteered to participate and provided written, informed consent. Due to the unpleasant nature of gag-reflex testing, we attempted to recruit different groups of subjects for each study. In total, two subjects participated in both Experiments 1 and 2, and two different subjects participated in both Experiments 2 and 3. The sample size for Experiment 1 was determined based on power analyses of MSNA and RVR in response to TSO compared with Sham (i.e., power > 80% for each measure).

Experiment 1: Effect of TSO on MSNA and RVR

Thirteen individuals (eight men, five women; 25 ± 1 years, 1.76 ± 0.03 m, 74.3 ± 4.0 kg, 24.0 ± 0.7 kg/m²) participated in Experiment 1. All experiments were conducted in the supine posture. Subjects
were first instrumented with a finger BP cuff (Finometer; Finapres Medical Systems, Amsterdam, The Netherlands) to monitor beat-by-beat BP, a three-lead ECG (Cardiopac5; GE Healthcare, Waukesha, WI) to measure HR, and a custom-designed pneumotrace to detect respiratory movement. Baseline recordings of MSNA (n = 6) or RVR (n = 8) were obtained for 5 min. For testing, the subject was instructed to “close the eyes, open the mouth, and stick out the tongue.” A registered nurse (same investigator for all trials) then applied stimulation to either the oropharynx (TSO; eliciting the gag reflex) or the hard palate of the mouth (Sham; no gag reflex) in a counterbalanced fashion. TSO was conducted using a wooden tongue blade or cotton swab, per standard clinical practice (7, 22), and was of very short duration (i.e., <2 s). Clinical intubation and laryngoscopy are typically 10–30 s in duration (21, 30, 47) and elicit the same airway defense reflex as TSO. The subject was not aware which stimulus was about to occur. A recovery period followed for 3–5 min, and once parameters returned to baseline, the opposite stimulus occurred. The goal was to administer one trial of TSO and one trial of Sham per subject, but additional trials were sometimes conducted if coughing (n = 2 trials) or limb movement (n = 3) occurred, since both of these cause measurement error. Although not the primary purpose of this study, during some MSNA trials, TSO was applied twice to explore whether hemodynamic and neural responses were attenuated in the second trial (Fig. 1). At the end of the experiment, subjects performed a maximal voluntary end-expiratory apnea to ensure that the quality of the MSNA recording was consistent from beginning to end.

Experiment 2: Effect of Lidocaine Inhalation on HR and BP during TSO

Ten individuals (six men, four women; 26 ± 1 years, 1.78 ± 0.03 m, 79.0 ± 5.2 kg, 24.8 ± 1.0 kg/m²) participated in Experiment 2. After baseline measurements of MSNA and BP, TSO was conducted in the supine posture. Next, the participants assumed the seated posture, and the TSO procedures were repeated within 2–3 min. Subjects were not allowed to leave the laboratory until the gag reflex returned (typically 20–40 min). TSO procedures were standardized, and beat-by-beat hemodynamics were always measured such that comparisons could be made within a given subject on the same day (i.e., test-retest reliability of hemodynamic responses to gag-reflex testing).

Experiment 3: Effect of Lidocaine Inhalation on HR and BP during Cold Pressor Test

On a separate day, four subjects underwent additional studies, which sought to determine whether the aforementioned lidocaine-inhalation protocol had a systemic effect (10, 26). After instrumentation and baseline measurements in the supine posture, subjects underwent the cold pressor test (CPT; hand into 1°C water) for 90 s. This procedure activates the sympathetic nervous system (23) and increase RPP, an index of myocardial oxygen demand (33). Immediately after the CPT, subjects were asked to rate their hand thermal sensation (where 0 = neutral/no sensation of cold, and 11 = unbearable cold) (15) and hand pain (where 0 = no pain, and 10 = unbearable pain) (18). Lidocaine inhalation was then conducted identically to the procedures listed above, and the CPT was repeated.

Measurements. During Experiment 1, multifiber recordings of MSNA were obtained, with a tungsten microelectrode (FHC, Bowdoin, ME) inserted in the peroneal nerve of a leg. A reference electrode was placed subcutaneously, 2–3 cm from the recording electrode, which was adjusted until a site was found in which muscle sympathetic bursts were clearly identified using previously established criteria (52). Briefly, MSNA was distinguished from other nerve signals when there was increased burst activity in response to maximal voluntary end-expiratory apnea (to activate arterial chemoreflex) (17) and/or passive muscle stretch but not with skin stroking of the innervated area, rapid inspiration, or arousal stimuli (52). The nerve signal was amplified, band-pass filtered with a bandwidth of 500–5,000 Hz, and integrated with a time constant of 0.1 s (Model 662C-3; The University of Iowa Bioengineering, Iowa City, IA). The

![Fig. 1](http://jap.physiology.org/). Representative beat-by-beat recordings of muscle sympathetic nerve activity (MSNA), blood pressure (BP), heart rate (HR), and respiratory movement (Resp) in the same subject. The 1st arrow denotes opening the mouth, and the 2nd arrow denotes tactile stimulation of the oropharynx (TSO; eliciting the gag reflex). Within 1–2 cardiac cycles after TSO, there was a rapid increase in MSNA, BP, and HR. Trials 1 and 2 were separated by 5 min.
nerve signal was also routed to a loudspeaker and a computer for monitoring throughout the study.

During Experiment 1, transabdominal Doppler ultrasound (HDI 5000; ATL Ultrasound, Bothell, WA) was used to measure renal blood flow velocity (RBV), as described previously (28, 40). The artery was scanned with a curved array C5-2 transducer.

Prior to all experiments, resting measures of systolic BP (SBP) and diastolic BP (DBP) were obtained via automated sphygmomanometry of the brachial artery (SureSigns VS3; Philips Healthcare, Andover, MA) in triplicate. Beat-by-beat BP, HR, MSNA, and respiratory movement were sampled at 200 Hz by a data acquisition system (PowerLab; ADInstruments, Colorado Springs, CO).

Data collection and statistical analysis. Beat-by-beat physiological parameters were analyzed offline using LabChart 7 (ADInstruments). The variables of interest included HR, SBP, DBP, mean arterial pressure (MAP), MSNA burst rate, and MSNA total activity. RPP was calculated as HR × SBP and was considered to be the primary outcome measure for two reasons. First, it was collected in all experiments, allowing consistent comparison (i.e., MSNA and RVR were only collected in Experiment 1). Second, it is a primary determinant of myocardial oxygen consumption that is relevant during upper-airway clinical procedures (29, 41). RBV was measured offline using ProSolv 3.0, and RVR was calculated as MAP/RBV; an increase in RVR is considered to be renal vasoconstriction (5, 27, 28, 40). Changes in MSNA total activity (area under the curve with baseline set to zero) and RVR were expressed as a percent change (%Δ) from baseline. All other data were expressed as an absolute change (Δ) from baseline.

Previous research has demonstrated that peak HR and BP responses occur within 5 s after the oropharynx is stimulated (21, 50). Therefore, we chose the peak cardiac cycle within this time frame as the peak hemodynamic response in the current study. Opening the mouth and sticking out the tongue (duration 3–5 s) were common to all experiments, so we also analyzed the five cardiac cycles preceding TSO (mouth open) and report an average. In a similar way, we analyzed the five cardiac cycles after the peak. When the gag reflex was not initiated (during Sham stimulation of Experiment 1 and after lidocaine in Experiment 2), the same latency after stimulation was reported as the peak response. For MSNA recordings, a bin containing 10 cardiac cycles (five before and five after TSO or Sham) was used as the peak. This approach was chosen, because a MSNA burst can only occur once/cardiac cycle.

Statistics were conducted using IBM SPSS 19.0, and graphics were produced with Microsoft Excel and Adobe CS5. For Experiment 1, two-way ANOVAs (time and stimulus) were conducted, followed by uncorrected paired t-tests on the peak ΔRPP, ΔMAP, ΔHR, %ΔMSNA total activity, %ΔRVR, and ΔRBV. For Experiment 2, a two (before lidocaine, after lidocaine)-by-five (time) repeated-measures ANOVA was used, followed by uncorrected paired t-tests when an interaction effect was found. With the use of data from Experiment 2, exploratory bivariate correlations were performed between ΔRPP, with TSO at the beginning of the study, and ΔRPP, with TSO at the end of the study (i.e., to clarify test-retest reliability of ΔRPP when undergoing gag-reflex testing within the same day). For Experiment 3, paired t-tests were used to compare SBP, DBP, MAP, HR, RPP, hand thermal sensation, and hand pain responses with CPT before and after lidocaine inhalation. Data are presented as means ± SE, and P < 0.05 was considered statistically significant.

RESULTS

Experiment 1: Effect of TSO on MSNA and RVR

Resting baseline values for MAP (77 ± 1 mmHg), HR (59 ± 3 beats/min), MSNA burst rate (11 ± 2 burst/min), and RBV (69 ± 8 cm/s) were within normal levels for young, healthy subjects. In all subjects, TSO elicited the gag reflex, which was audible and confirmed by the subject and the investigators. Qualitative data (Fig. 1) indicate that TSO increased MSNA total activity as well as HR and MAP. Indeed, the ΔMAP (12 ± 3 vs. 6 ± 1 mmHg), ΔHR (10 ± 3 vs. 3 ± 3 beats/min), and ΔRBV (−15 ± 4 vs. 1 ± 1 cm/s) were greater following TSO compared with Sham (i.e., without eliciting the gag reflex). As shown in Fig. 2, the ΔRPP (P = 0.042), ΔMSNA (P = 0.040), and ΔRVR (P = 0.010) were significantly greater with TSO compared with Sham. Relative to baseline, Sham stimulation caused significant increases in RPP (P = 0.048) and MAP (P = 0.005) but not HR (P = 0.366), RBV (P = 0.691), or RVR (P = 0.304). Additionally, beat-by-beat analysis of MSNA revealed that in the first cardiac cycle following TSO, six of six subjects had a MSNA burst compared with zero of six subjects following Sham. In the second cardiac cycle following TSO, five of six subjects had a MSNA burst compared with two of six subjects following Sham.

Fig. 2. Changes in rate-pressure product (ΔRPP; top, n = 13), ΔMSNA (middle, n = 6), and renal vascular resistance index (ΔRVR; bottom, n = 8) in response to TSO (black bars) and tactile stimulation of the hard palate (Sham; white bars) during Experiment 1. Data are means ± SE. *P < 0.05 between TSO and Sham; †P < 0.05 vs. respective baseline.
Experiment 2: Effect of Lidocaine Inhalation on HR and BP during TSO

Inhalation of lidocaine did not affect supine resting MAP (pre: 77 ± 2; after post: 78 ± 2 mmHg) or HR (pre: 58 ± 3; post: 56 ± 3 beats/min). During and after lidocaine inhalation, subjects reported a lack of sensation of the tongue and throat, and some individuals complained of hoarseness. Lidocaine blocked the gag reflex in all subjects (Fig. 3). The RPP response to TSO was reduced drastically following lidocaine (Fig. 4). Opening the mouth and sticking out the tongue elicited a modest increase in RPP, but this was not different between treatments ($P = 0.122$). Lidocaine reduced the peak RPP response to TSO ($P = 0.004$), as well as the RPP response within the first five cardiac cycles of recovery ($P = 0.014$). As expected, the peak SBP (pre: 25 ± 4 vs. post: 7 ± 2 mmHg), peak HR (pre: 12 ± 3 vs. post: 2 ± 2 beats/min), and peak MAP (pre: 16 ± 2 vs. post: 5 ± 2 mmHg) response to TSO were also attenuated following lidocaine inhalation (all $P < 0.01$). As shown in Fig. 5, there was a strong, positive relationship between TSO-induced RPP responses within the same day (Cronbach’s $\alpha = 0.743$, $P = 0.036$).

Experiment 3: Effect of Lidocaine Inhalation on HR and BP during CPT

To determine whether inhaled lidocaine would attenuate hemodynamic and perceptual responses to the CPT, paired $t$-tests were conducted. The $\Delta$RPP was not different before (2.347 ± 873 mmHg × beats/min) and after (2.559 ± 629 mmHg × beats/min) lidocaine ($P = 0.591$). Similarly, the $\Delta$MAP (pre: 30 ± 6; post: 26 ± 8 mmHg) and the $\Delta$HR (pre: 4 ± 6; post: 6 ± 4 beats/min) were also not statistically different. Furthermore, hand thermal sensation (pre: 9 ± 1; post: 8 ± 1) and hand pain (pre: 7 ± 1; post: 7 ± 1) were comparable before and after lidocaine, indicating that lidocaine inhalation did not cause systemic sympathoinhibition.
DISCUSSION

Previous research has demonstrated that laryngoscopy and upper-airway intubation cause rapid increases in HR, BP, MSNA, and plasma catecholamines (1, 8, 16, 21, 38, 53). These changes are likely to be reflex mediated, because they can be blunted or abolished by using different pharmacotherapies (i.e., ganglionic blockade, beta blockade) (46, 51). However, these cited studies were performed in anesthetized patients, and it is known that general anesthesia can affect reflex pathways (44). For this reason, we sought to document how conscious, unmedicated humans would respond to TSO. The primary, novel findings are: 1) TSO elicited reflex increases in MSNA, MAP, HR, and RVR compared with Sham; 2) inhalation of 4% topical lidocaine prior to TSO blocked the gag reflex and attenuated increases in HR and BP; and 3) inhalation of 4% topical lidocaine did not affect the hemodynamic or perceptual responses to the CPT. To our knowledge, this is the first report of TSO eliciting sympathoexcitation in conscious, unmedicated humans.

The afferent arm of the gag reflex is comprised of the glossopharyngeal nerve (cranial nerve IX) and the laryngeal branch of the vagus nerve (cranial nerve X). These nerves relay sensory information from the pharynx, tonsils, epiglottis, and base of the tongue to the medulla (31, 57). The efferent arm of the gag reflex includes the vagus nerve (cranial nerve X) and results in contraction of the posterior oral and pharyngeal musculature, thus preventing foreign bodies from entering the trachea (25). On the other hand, the hard palate is innervated by the maxillary branch of the trigeminal nerve, also called the nasopalatine nerve. The application of pressure to this nerve does not typically elicit the gag reflex. Therefore, Sham was considered to be the control in Experiment 1.

As noted in Fig. 1, TSO elicited rapid increases in HR and MAP, as detected by the Finometer device. Previous experiments using an arterial catheter have shown that upper-airway stimulation increases SBP by 30–60 mmHg and DBP by 10–30 mmHg, while causing a modest tachycardia (HR 15–30 beats/min) (1, 8, 16, 21, 53). Our data are comparable, considering that TSO was of shorter duration than laryngoscopy and intubation (20, 37, 53). Clinical observations have also demonstrated that lidocaine blocks sodium channels and prevents afferent nerves from generating an action potential. Because of this, lidocaine has also been used in research studies to understand how peripheral afferents (e.g., within muscle or blood vessel) affect BP homeostasis (6, 10, 36). With the use of lidocaine inhalation to block sensory afferents in the oropharynx (presumably cranial nerves IX and X), we have demonstrated that TSO elicits a sympathoexcitatory reflex (in addition to the well-characterized gag reflex) in conscious, healthy humans. It is important to note that our lidocaine-inhalation protocol did not affect the physiological or perceptual responses to the CPT (Experiment 3). Regional administration of lidocaine into a limb is known to block the pressor and pain response to the CPT (14). Taken together, these data provide strong experimental evidence that inhalation of lidocaine is exerting a focal, not systemic, anesthetic effect. Lidocaine inhalation is simple, well tolerated, and does not affect resting hemodynamics; these factors make it ideal to use in future research studies.

Experiment 2 required that each subject experience the gag reflex twice within the same day (i.e., before lidocaine was given and after it had worn off). Although not the intended purpose of our study, this experimental design allowed for test-retest reliability to be determined. As displayed in Fig. 5, the ΔRPP in response to TSO was similar within the same day, such that higher RPP responses to the first TSO were related to higher responses to the second TSO. This supports the previously established concept that individual differences in cardiovascular reactivity to upper-airway stimulation exist (19, 35).

On a physiological level, the current data indicate that short-duration tactile stimulation of the upper airway elevates sympathetic outflow to the kidney and skeletal muscle. These data in conscious human subjects extend upon prior publications, suggesting that the oropharynx is a sensory organ capable of initiating sympathetic reflexes (51, 56). On a clinical level, thousands of intubations are performed each day throughout the world, and many of these patients have underlying cardiovascular disease (29, 35). The sympathetic nervous system likely contributes to cardiac and renal complications observed in these patients. Specifically, longer intubation durations are linked with increased risk of myocardial infarction (2), and electrocardiographic abnormalities are most common during intubation (20, 37, 53). Clinical observations have also shown that postoperative renal failure (seemingly due to reductions in blood flow) is linked to increased mortality (49). Furthermore, ventricular tachycardia and cerebrovascular accident have been documented in one kidney-transplant patient following endotracheal intubation (12). During upper-airway procedures, it is desirable to prevent a large increase in RPP (i.e., with afferent blockade) rather than to give vasoactive medication or additional inhalation anesthetics (i.e., with ef-
ferent blockade, which would not normally be used) to combat an increased RPP. Whether oropharyngeal afferents also contribute to adverse outcomes resulting from other airway stimuli (e.g., cold-air inhalation, cigarette smoking, prolonged ventilator use) is yet to be determined.

The current experiments used a physiological approach to understand how TSO impacts cardiovascular homeostasis in young, healthy humans. As such, extrapolation to healthy, older adults or patient populations must be done with caution. It should be noted that TSO was of short duration in the current study, leading to similar yet smaller responses compared with previous studies (1, 8, 16, 21, 53). For ethical reasons, we chose not to intubate our conscious, healthy subjects and instead, focused on the physiological mechanisms underlying TSO. It is also possible that stimulation of the larynx or lower respiratory tract may elicit a different response, and this response may be modulated by the type of stimulus (e.g., pressure, temperature, irritation) (32, 33, 39). Additional studies are warranted to unravel how TSO impacts human physiology in both healthy and diseased states.

Clinical Implications

In the current study, TSO elicited acute increases in MSNA, MAP, HR, and RVR in conscious humans, and this effect could be blocked with local anesthesia of the upper airway. These data provide evidence that airway defense mechanisms (e.g., gag reflex) engage the sympathetic nervous system and elevate HR and BP. We speculate that a sensitized upper airway, due to allergies, cigarette smoking, or gingivitis, places a patient at an elevated cardiovascular risk during intubation and laryngoscopy via a sympathetic neural mechanism. To our knowledge, this concept has not been established previously, but the current physiological data relating upper-airway afferents to systemic vasomotor control support this idea.

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