

Effects of Suboccipital Release Osteopathic Manipulation on Autonomic Nervous System: Insights from Short-Term QT Interval Measurements in Healthy Young Adults

Ryan Witczak¹, Jennifer Kachelmeyer¹, Kristina Cummings¹, Maxim Crasta^{1*}

Abstract

Introduction: Osteopathic physicians employ the Sub Occipital Release (SOR) manipulation technique to promote wellness and modulate the Autonomic Nervous System (ANS). It has been found that this technique promotes relaxation and balance throughout the body. The principle behind SOR involves targeting mechanoreceptors found within the deep fascia, connective tissue, and muscles of the sub occipital area. By effectively balancing the ANS, SOR techniques are believed to alleviate stress and improve blood flow in the suboccipital region. This could potentially relieve any potential compression on the vagus nerve, enhancing vagal activity in cardiac myocytes. The primary objective of this research is to examine how SOR impacts cardiac control, specifically using QT variability as a surrogate measure.

Methods: A crossover design was incorporated with three interventions: a control group with no physical contact, sham treatment, and a SOR group. Data was collected using 12-lead EKG recordings, with intervals for QRS, QT, QTcB, JT, QTa, and QTend. The index of cardiac electrophysiological balance (iCEB) was determined by measuring QTcB and QRS duration.

Results: The multiple comparisons showed that there was no significant difference in the QTa measurements between the V2 and aVF leads, as these two leads record the maximum and minimum QTa intervals [F (1.705, 34.11) = 1.294, P = 0.06]. The mean values (Mean±SEM) for the control, sham, and SOR groups (96.29 ± 4.37msec, 97.14 ± 5.85 msec, and 89.48 ± 4.62msec respectively). This was significantly lower for the SOR group [F (1.705, 34.11) = 1.294, P = 0.05]. The variability in the QTcB was also found to be statistically significant [F (1.410, 28.19) = 0.4429, P=0.051]. To get a more accurate measurement of relative variation compared to just using QTend alone, we looked at the ratio of QTend to either QT or QTc interval. The mean ratio values (Mean±SEM) for the control, sham, and SOR groups were 0.26±0.01, 0.25±0.01, and 0.19±0.01 respectively. A significant decrease was observed in the SOR group when compared to the other two groups.

Conclusions: The effects of SOR on QT metrics were diverse, resulting in a moderate increase in both QT and QTcB length. Additionally, there was a decoupling of the QTend and JT intervals, leading to shortened QTend intervals potentially due to increased vagal activity. This could be a result of alterations in repolarization or a temporary decrease in heart rate due to vagal stimulation. This, along with a moderate increase in QTcB, suggests that SOR may improve cardiac function by prolonging the effective refractory period enhancing ventricular relaxation.

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Introduction

Osteopathic manipulation techniques (OMT) applied to the upper cervical spine or suboccipital region have been found to exert a beneficial effect on the functioning of the parasympathetic division of the autonomic nervous system (ANS) [1,2]. OMT is a manual therapy technique used by Osteopathic Physicians to address various musculoskeletal conditions. The SOR manipulation has gained attention due to its potential to influence parasympathetic nervous system (PSNS), which plays a crucial role in regulating various bodily functions, including heart rate, digestion, and stress response [3,4]. This procedure entails the application of pressure to a targeted area in the suboccipital region, aimed at alleviating tension and inducing relaxation.

This manipulation targets specific mechanoreceptors located beneath the connective tissue system through myofascial movements [5]. These movements stimulate the receptors, initiating a parasympathetic response. SOR works on the principle that by manipulating these receptors, it can have a positive effect on the ANS, promoting a state of balance and well-being [6]. This gentle yet effective technique is often used to treat conditions such as headaches or tension in the neck and shoulders. By increasing the parasympathetic response, SOR aims not only to alleviate physical symptoms but also induce a sense of calm and relaxation in the body [7].

The primary objective of this research was to investigate the effects of SOR on cardiac control by analyzing the quantification of QT variability. Giles and colleagues proposed a hypothesis that suggests that suboccipital decompression has the potential to improve vagal output to the heart, as evidenced by changes in heart rate variability [1]. Our proposal suggests that implementing SOR techniques to relieve stress and improve blood flow in the suboccipital areas may help alleviate any potential compression on the vagus nerve. This could ultimately enhance vagal activity in cardiac myocytes, potentially resulting in observable changes in EKG variables. This could lead to improved function and a reduction in associated symptoms. The scope of this investigation centered on QT variability as an instrumental factor in evaluating cardiac regulation. Our ultimate objective was to unveil the potential advantages and efficacy of utilizing SOR as a viable form of treatment for enhancing cardiac autonomic function.

The QT interval is a crucial measure in electrocardiography that reflects the duration of ventricular depolarization and repolarization. Variations in the QT interval have been linked to changes in autonomic function and fluctuations in parasympathetic tone [8]. When parasympathetic tone

increases, there is a decrease in heart rate and prolongation of the QT interval. Therefore, monitoring changes in the QT interval can provide valuable insights into autonomic function [9].

The anatomical relationship between the ANS and the restoration of a normal homeostatic milieu and physiological balance has been an area of interest for researchers [10,11]. Studies have shown that stimulation of the vagus nerve can have positive effects on conditions such as chronic pain, anxiety, and even autoimmune diseases [12,13,14] and sympathovagal balance in sleep apnea patients [15]. More research is needed to fully understand this theory.

Touch alone has been found to have significant effects on physiological outcomes, as supported by various reports [16,17]. We conducted a sham control intervention as well as a no-touch control to validate these findings. The study group, designated as the SOR group, underwent an intervention that involved manipulating anatomical structures to elicit specific physiological responses. During such manipulations, the vagus nerve may be modulated as it passes through the skull. Additionally, it has been reported that SOR techniques might potentiate the function of the vagus nerve, particularly in individuals with cardiac complications, as observed in recent clinical trials [4,18].

Methods:

Subject Recruitment

This study was approved by the Institutional Review Board of the Lake Erie College of Osteopathic Medicine (LECOM) in Elmira, NY, USA (approval no. 30-097). A total of 24 healthy adults, aged 22 to 30, were recruited at the research lab. The participants consisted of 12 male and 12 female students from the first year and second-year medical programs. Informed consent was obtained from all participants after meeting the inclusion and exclusion criteria outlined in the research protocol.

Study Groups

In this study, a crossover design was employed, wherein each subject received each intervention in a sequential manner. To test the hypothesis, three different experimental interventions were implemented: No physical contact group (control), sham treatment (sham), and suboccipital release (SOR). To ensure proper recovery and evaluation, a 15-minute rest period was allotted between each intervention. The SOR manipulation was carried out according to the procedures outlined by earlier researchers [19]. The height, weight, age, gender, and heart rate of all participants were recorded.

SOR and Sham Procedure:

To execute a suboccipital release, the subject was positioned lying flat on their back to ensure maximum

comfort. The investigator administering the test sat at the head of the bed and placed both hands underneath the subject's head in the occipital region. By palpating the scalp for the occipital ridge, they were able to guide their hands slightly downward until they felt tension in the suboccipital muscles. Then, using upwardly flexed fingers against these muscles, they maintained this pressure for five minutes or until a significant release in muscle tension was achieved to alleviate discomfort and improve mobility. Throughout this process, proper form and pressure were carefully sustained to yield optimal results.

During a sham treatment protocol, subjects were given what seemed to be a real treatment, but it had no therapeutic effect. The investigator placed their fingers near the occipital condyles to create the illusion of treatment, while holding the subject's head in their hands for about five minutes. No tension or force was applied in any direction during this process.

No touch controls- The subject was asked to lie in a supine position for fifteen minutes in a quiet and relaxed manner. The purpose of this was to allow for a period of rest and relaxation before subsequent treatments were carried out. This was to minimize any potential therapeutic effects that could result from touch by the investigator.

Reproducibility

For this study, the Nasiff CardioCard PC-Based EKG device (San Pedro CA, USA) with inbuilt computer calipers was used. The screen calipers were employed using keyboard controls. These calipers can be adjusted precisely by holding down specific keys on the computer and dragging the mouse cursor allowing for accurate measurement of tangent lines. In addition to the automated measurements provided by the device, a manual check was conducted using random sampling of the EKGs stored in the computer. Both investigators involved in the study independently measured the intervals, and their values were subsequently cross-checked by the Principal Investigator. The intra-personal measurement difference between the manual method and Caliper method was found to be insignificant, demonstrating reproducibility.

Recording of EKGs and QT parameters

Recordings of a 12-lead EKG were made at the speed of 25 mm/s and at the voltage of 10 mm/mV, and they were subsequently stored on a computer for analysis. The readings were obtained after each subject had rested in a supine position for 20 minutes prior to each intervention. Prior to the initial intervention, a baseline period of data was recorded for the control group. Following the baseline period, two additional interventions were conducted: sham manipulation and a SOR. Each intervention lasted for five minutes, with a 15-minute break separating each intervention.

The duration of the QT intervals was determined through precise measurements taken in lead II. To ensure accuracy, at least two uninterrupted sinus rhythm cycles were recorded and analyzed. Only EKG waves displaying a distinct T wave without a U wave were used for this analysis. To determine the QT interval, a tangent line was drawn to intersect with the steepest downward slope of the dominant repolarization wave (as seen in Figure 7) and the isoelectric line. This intersection point served as the termination point for measuring the QT interval.

To account for variations in heart rate, Bazett's formula was employed to calculate QTcb. This method involves dividing the QT interval by the square root of the R-R interval in milliseconds ($QT_{cb} = QT / \sqrt{RR}$). The QT_e (or QT_{end}) interval was determined as the interval from the peak to the end of the T waves.

The QT_a interval was measured from the start of the QRS complex to the highest point of the T wave, specifically defined as the peak of the T wave. In cases where there were biphasic T waves, the highest point of the largest component of the T wave was identified. The JT interval was measured from the end of the QRS complex to the end of the T wave. This interval indicates the initial phase of repolarization, while the QT_{end} interval represents the final portion of ventricular repolarization. An average value was taken over three consecutive heartbeats for each measurement.

The index of cardiac electrophysiological balance (iCEB) is calculated by dividing the QT interval by the QRS duration and iCEB_c obtained by dividing QT_{cb} by QRS duration as done by earlier workers [20]. The differences between the longest (QT max) and shortest (QT min) in the EKG were used to calculate QT dispersion (QTd) in milliseconds. QTd was determined using the following formula: $QTd = (QT_{max} - QT_{min})$ and a normal value for QTd was in the range of 30 to 60 milliseconds [21].

Statistical analysis

The mean±SEM (standard error of the mean) was used to present the numerical data. The study utilized a crossover design and employed repeated measure analysis of variance (ANOVA) with each participant acting as their own control. To evaluate fundamental attributes within the groups, including age, body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate, a one-way ANOVA was performed. The Shapiro-Wilk test was employed to determine whether the data sets were normally distributed. Post-hoc comparisons of the three groups, control, sham, and SOR, were adjusted using Bonferroni's method for multiple comparisons. The statistical analyses were performed using SPSS (version 29, IBM SPSS Statistics for Windows, Armonk, NY: IBM Corp) and Prism (version 10.0; GraphPad Software Inc., La Jolla, CA). A

significance level of $P < 0.05$ was used to determine statistical significance. To assess the agreement between the QTend values in control and sham-treated participants, Bland-Altman limits of agreement were calculated and presented graphically as shown in Figure 6.

Results

The findings of this study were derived from data collected through a sample of 21 participants, consisting of 11 females and 10 males. The recruitment process involved 24 young adults, of whom 3 participants were excluded from the final analysis due to abnormal EKG results during the initial recording.

The remaining 21 participants had an average age of 24.10 ± 1.832 (mean \pm SEM) and a Body Mass Index (BMI) of 24.70 ± 4.68 (mean \pm SEM). Their systolic blood pressure was measured at 117.9 ± 4.02 (mean \pm SEM), and diastolic blood

pressure at 67.90 ± 3.01 (mean \pm SEM), both falling within normal levels. The heart rate of the participants was 69.29 ± 3.14 (mean \pm SEM), before introducing any interventions. No adverse effects were reported by any of the participants during the study.

Table 1 Presents the mean \pm SEM data for the intervals described in the method section. As indicated, participants underwent testing at three levels: control, sham, and SOR. A normality check was conducted, and the results of a repeated ANOVA revealed no significant differences among the mean changes observed at the different time points for the QT intervals [$F(1.607, 32.14) = 1.828, P = 0.092$]. Similarly, there were no significant QTa values recorded [$F(1.499, 29.99) = 1.828, P = 0.073$]. The multiple comparisons revealed no significant difference when comparing the QTa measurements obtained from the specifically V2 and aVF leads, since these two leads record the maximal and minimal QTa intervals [$F(1.705, 34.11) = 1.294, P = 0.06$].

Table 1: QT variables derived from electrocardiographic data obtained from three groups.

QT variables derived from electrocardiographic data obtained from three groups.						
Interval	Group	Mean \pm SEM	N	95% Confidence Interval		p-value ^a
				Lower Bound	Upper Bound	
QT	Control	403.14 \pm 7.26	21	388.62	417.67	P > 0.05
	Sham	406.67 \pm 7.14	21	392.14	421.19	P > 0.05
	SOR	410.81 \pm 8.91	21	396.29	425.33	P > 0.05
QTcb	Control	420.71 \pm 7.32	21	409.81	431.61	P > 0.05
	Sham	422.18 \pm 6.81	21	411.28	433.08	P > 0.05
	SOR	425.05 \pm 3.32	21	420.15	430.95	P > 0.05
QTa	Control	306.86 \pm 7.23	21	291.3	322.13	P > 0.05
	Sham	309.52 \pm 6.94	21	292.71	326.33	P > 0.05
	SOR	321.33 \pm 8.81	21	304.52	338.14	P > 0.05
QTend	Control	96.29 \pm 6.44	21	86.3	106.27	P > 0.05
	Sham	97.14 \pm 7.12	21	87.16	107.13	P > 0.05
	SOR	89.48 \pm 8.04	21	79.49	99.46	p < 0.05
JT	Control	306.71 \pm 8.39	21	291.3	322.13	P > 0.05
	Sham	311.62 \pm 7.19	21	296.2	327.04	P > 0.05
	SOR	312.86 \pm 8.96	21	297.43	328.27	P > 0.05

^a computed using alpha = .05. The data represents the mean value with associated standard error (Mean \pm SEM). QTcb refers to QT calculated using the Bezett's formula. QTa is the interval from the onset of Q wave to the peak of T wave, while QTend measures the interval from peak of T wave to its end. JT is measured from J point to the end of T wave. N is the total number of subjects participated. p < 0.05 is considered a significant level.

The average QTend values for the control, Sham, and SOR groups were 96.29 ± 4.37 msec, 97.14 ± 5.85 msec, and 89.48 ± 4.62 msec (mean \pm SEM), respectively. There were no differences between control and Sham groups observed [$F(1.870, 37.40) = 0.8662, P = 0.08$]. Nevertheless, the comparison between the two groups (control vs SOR) indicated a noteworthy decrease in QTend values within the SOR group, indicating a statistically significant difference. The calculated difference in the QTend between the three groups was statistically significant [$F(1.705, 34.11) = 1.294, P = 0.05$]. The JT interval, which represents a significant portion of the repolarization phase in ventricular action potentials, displayed no substantial variation amongst the three groups [$F(1.692, 33.84) = 1.053, P = 0.3496$].

The findings of this study suggest that there was a minimal difference in the QT and JT intervals when measured from the V2 and aVF leads. The data showed only a small variation between minimum and maximum values, indicating a consistent pattern of electrical activity. The observed difference between the QT and JT intervals was found to be 4 ± 0.1 msec ($P > 0.05$), which suggests that any variance was negligible.

This indicates that there is a consistent depolarization in the myocardial muscle across different regions, without any significant differences in the QT and JT intervals among the groups that underwent SOR intervention. This steady depolarization can be attributed to an overall balanced electrical activity throughout the heart, with no dispersion.

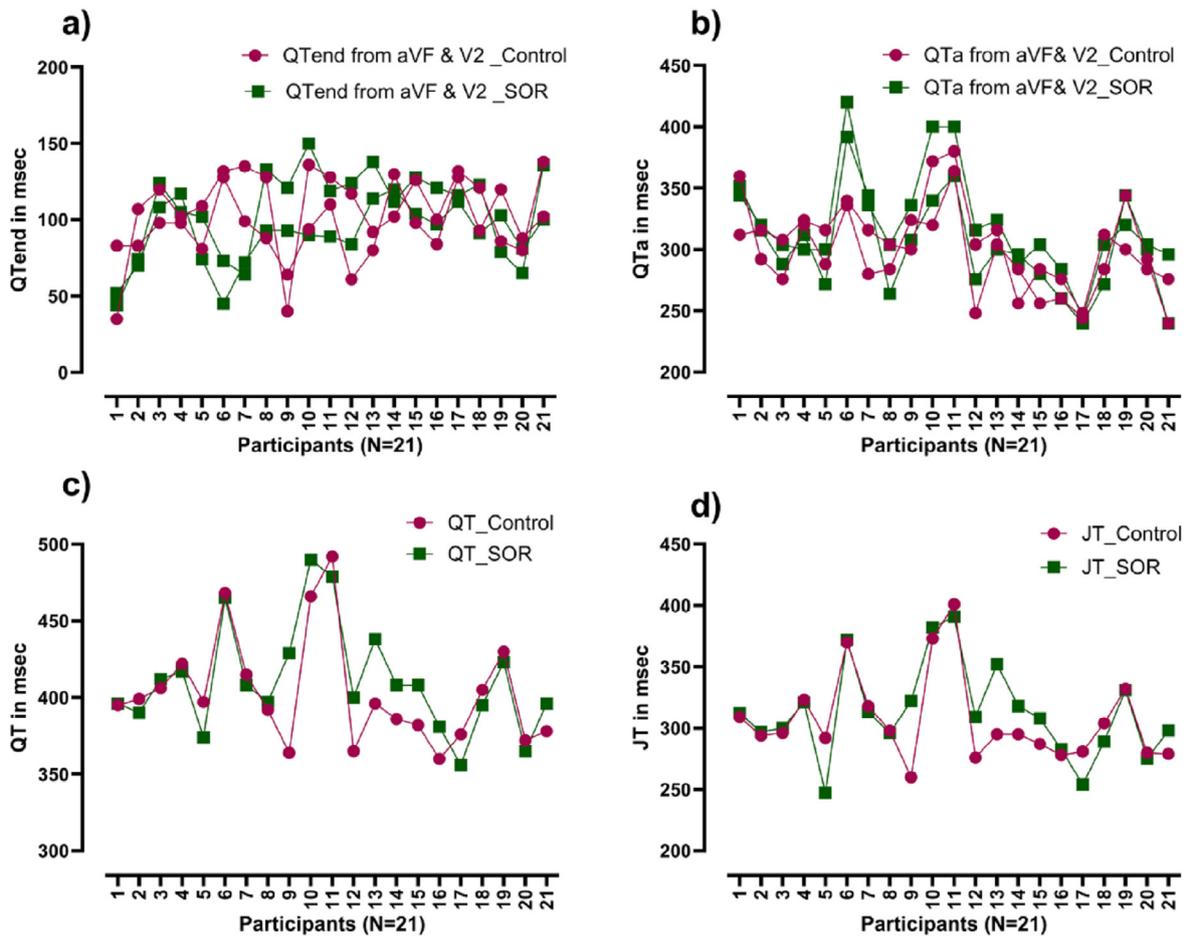


Figure 1: The QTend, QTa, QT, and JT intervals and inhomogeneities

Figure 1: Panels (a-d) display graphical representations of SOR's impact on the dispersion of data points within four distinct time intervals: QTend, QTa, QT, and JT. A comparison of both groups (control vs SOR) to identify any noticeable differences. Panels a and b showcase the QTend and QTa intervals obtained from leads aVF and V2. The X-axis shows data points collected from all 21 participants. Panel a show heterogeneous QTend values in contrast to the other intervals.

There was an increase in the duration of QTcb in Sham and SOR groups, but the increase has not been statistically significant. However, the variability in QTcb was found to be statistically significant $F[1.410, 28.19] = 0.4429, P = 0.051$, for SOR group as shown in Figure 2.

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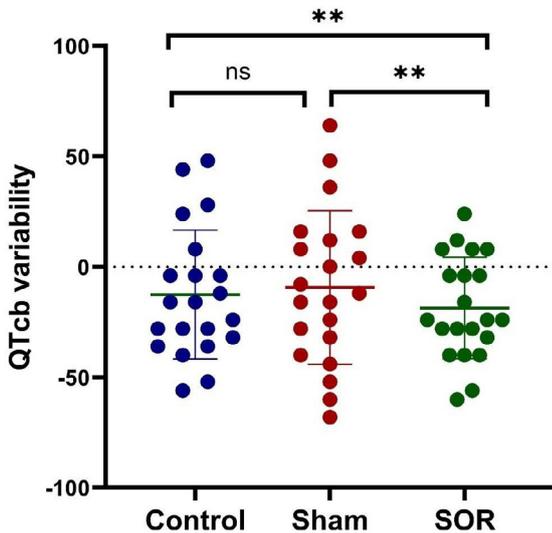


Figure 2: The variance of QTcb in three groups. Non-significant findings are denoted by ns and an asterisk (**) for significance at $p < 0.05$ level.

Figure 2 displays the analysis of variance of QTcb data for all three groups, examining the impact of individual variation on this interval. This suggests that the intervention significantly influenced the variability of QTcb and led to a notable increase in central tendency compared to the other groups, indicating its effectiveness in moderating individual variations of the QTcb interval. The decreased dispersion of QTcb may assist in amplifying the alternating patterns of swift deceleration and acceleration of heart rate, which could be directly associated with the activation of the parasympathetic nervous system.

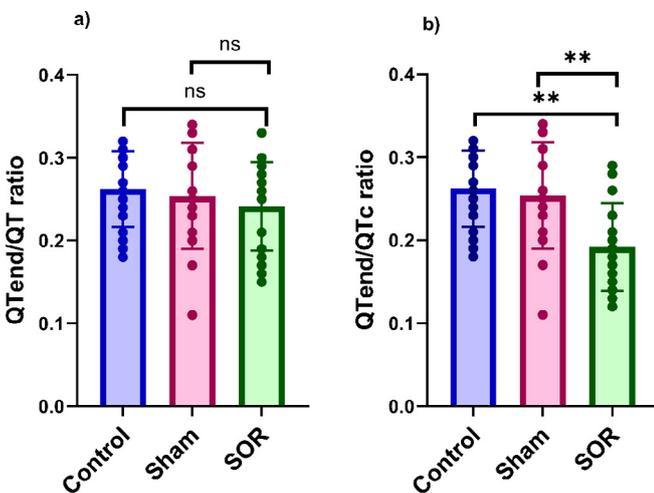


Figure 3: QTend/QT and QTend/QTc ratios in three groups

Panel a): The figures depict the variations in the QTend/QT ratio across three groups. The "ns" indicates that there were no significant differences. Panel b): The QTend/QTc ratios for each of the three groups were computed and compared. A significant decrease in the QTend/QT ratio compared to the other two groups. These results, as indicated by (**), are statistically significant with a p-value of < 0.05 . Abbreviations: QTend represents the duration, measured in msec, from the start of T wave peak to its end; QTc is the corrected QT interval adjusted for heart rate using Bezzet's formula.

The ratio of QTend to QT or QTc interval provides a more precise measurement of relative variation compared to QTend alone. This may be due to its consideration of variations in heart rate and individual discrepancies in the duration of the QT interval. The mean ratio values (Mean±SEM) for the control, sham, and SOR groups were recorded at 0.26 ± 0.01 , 0.25 ± 0.01 , and 0.19 ± 0.1 respectively, providing further support this observation. Notably, a significant decrease in the ratio was observed in the SOR group when compared to the other groups [$F (2,60) = 10.35, P = 0.03$].

Figure 4: The iCEB index obtained from QT, QTc, and JT intervals. Non-significant results are marked with ns, while significant levels ($P > 0.05$) are denoted by (**).

Figure 4 illustrates the ratios computed from various parameters of the QT. The numerator of the ratio predominantly encompasses repolarization, exemplified by values for the QT, QTc, and JT intervals as displayed in panels a-c respectively. The denominator reflects QRS duration, which is an indicator of depolarization. This ratio serves to signify the balance of cardiac electrophysiology, a derived measure from these values. This ratio was found to be significant for the QTc/QRS ratio 4.28 ± 0.08 vs 4.61 ± 0.07 (mean± SEM) in control vs SOR groups respectively [$F (2,60) = 1.58.097, P = 0.05$].

Figure 5: The Bland-Altman plot, which was utilized to assess the level of agreement between QTend values among both sham-treated subjects and controls. This involved pairing the QTend values from both groups to determine if there was any discrepancy (or bias) in their repeated measurements. The limits of agreement are represented by the upper and lower horizontal dashed lines in the plot, which show the range within which the true mean difference is likely to fall. The Y axis represents the mean difference between outcomes for control and sham-treated subjects. X-axis is the mean of two measurements.

The agreement between the control and sham groups was assessed by constructing a Bland-Altman plot as shown in Figure 6. The calculated Bias or Bland-Altman index of agreement was found to be -0.86 , indicating minimal difference between the two groups in terms of their QTend

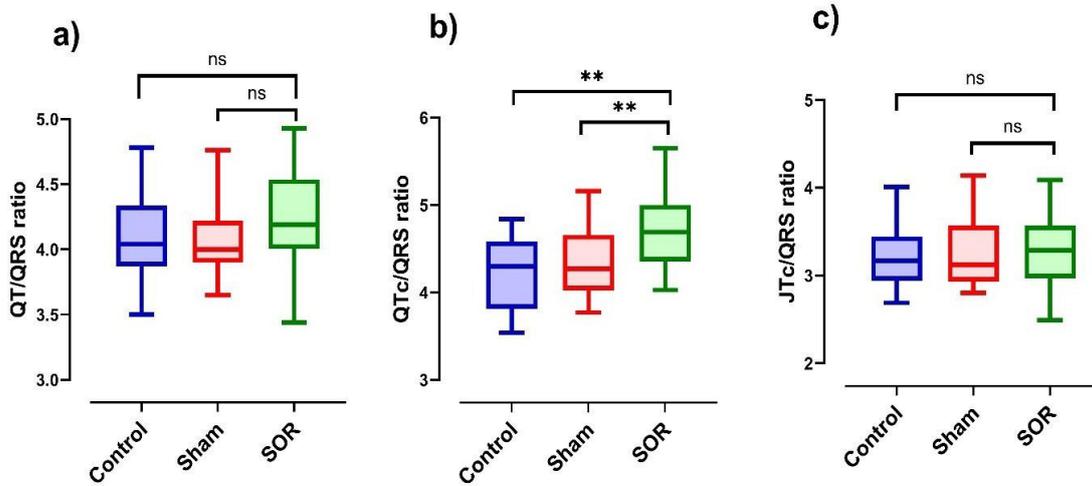


Figure 4: The index of cardiac-electrophysiological balance (iCEB) derived from the QT variables.

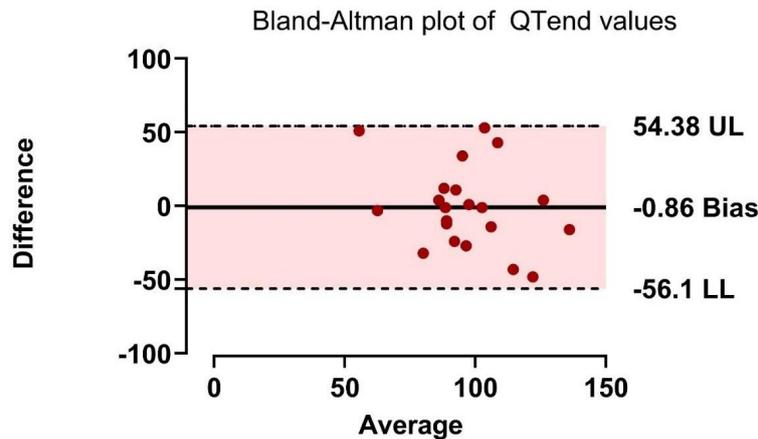


Figure 5: Bland-Altman plot for QTend

values. This suggests that any significant differences observed in the SOR group were solely caused by the sub occipital manipulation and not influenced by touch or other external factors. Therefore, it may be concluded that the manipulation technique might have been responsible for the observed effects on QTend values in the SOR group.

Discussions

Although there is limited research, evidence suggests that OMT may lead to positive clinical outcomes, such as reduced pain severity and stress levels through modulation of the ANS [22,23]. The parasympathetic branch of the ANS possesses both anti-inflammatory and anti-nociceptive properties. Acetylcholine released by vagal endings has a specific binding effect on alpha-7 nicotinic receptors of macrophages, inhibiting the production of pro-inflammatory cytokines [24]. This demonstrates the anti-inflammatory effects of the parasympathetic branch. On the other hand, the sympathetic branch has pro-inflammatory effect, which may potentially increase pain levels. Norepinephrine released by

sympathetic nerve terminals induces interleukin-6 release, which is mediated through β_2 -adrenergic receptors. This further illustrates the pro-inflammatory role of this branch [25].

Multiple studies have shown that OMT techniques effectively enhance parasympathetic nervous system activity as measured by HRV analysis. Ruffini et al. reported a significant increase in high frequency HRV after OMT was administered [23]. Fornari et al. observed heightened high frequency HRV during an arithmetic stress test in individuals who received craniosacral OMT compared to those who did not receive such treatment [26]. Curi et al. demonstrated that fourth ventricular compression reduced blood pressure in hypertensive patients and this antihypertensive effect demonstrated to increase parasympathetic activity [27]. OMT has been shown to exert an influence on the ANS, with variations in the response depending on the stimulation site and type. Specifically, a greater parasympathetic response is observed when stimulation is performed in the cervical and lumbar regions, whereas a greater sympathetic response

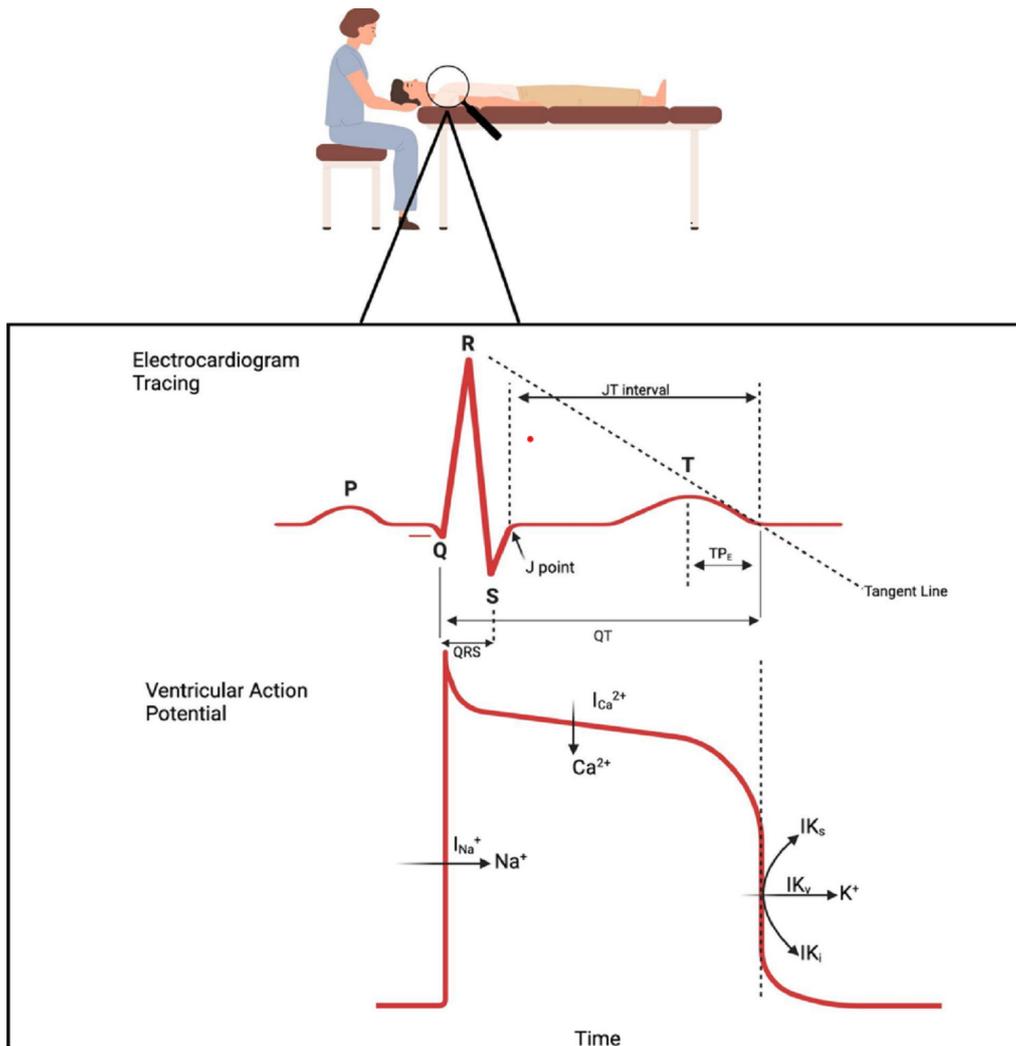


Figure 6: Modulation of QT interval by SOR and temporal relationship with action potential in ventricular myocytes

is observed when stimulation is performed in the thoracic region. [28].

The proximity of the vagus nerve to the musculoskeletal structures in the suboccipital region is a crucial aspect to consider in diagnosing and treating somatic dysfunctions. The study conducted by Courties et al. highlights the link between local inflammation or spasms in this area and its potential impact on the functioning of the vagus nerve [29,30]. This can impair its optimal functioning and autonomic control of the cardiovascular system, resulting in altered EKG variables, particularly the QT interval, which serves as a surrogate measure in this study. The duration of QT is a critical factor in assessing the electrical activity of cardiac ventricles, encompassing the entire process of depolarization and repolarization [31,32]. QT interval variability has emerged as a promising measure of ventricular sympathetic activity. Recent evidence suggests a clear link between QT interval variability and changes in sympathetic tone, particularly in pathophysiologic states where sympathetic activity is elevated, and vagal tone is decreased [33]. Vagal

tone was found to have a greater effect on QT intervals as cycle length increases, while sympathetic tone did not appear to have a significant impact [34]. When beta blockers were used, it was found that the effects of vagal tone were directly exerted on the electrophysiological substrate of the ventricles [35]. The activation of the parasympathetic nervous system, indicated by an increase in vagal tone, can potentially lead to a decrease in heart rate and an increase in APD [36]. In this study, we observed a slight increase in both QT and QTcb measurements among participants who underwent SOR, as well as a reduction in the variability of QTcb values (Table 1 and Figure 2). Recent research has highlighted the importance of QT variability, which refers to variations in interlead QT measurements as a non-invasive method for measuring underlying dispersion [37]. This measure serves as an indicator of ventricular recovery, making it a useful tool for evaluating duration of APD.

The QT interval is a key indicator for detecting variations in its components, which can provide important insights into underlying changes in the action potential [38]. The QRS

complex represents ventricular depolarization, while the QTa and JT intervals largely cover the repolarization phase of the ventricles. Furthermore, the QT interval is closely tied to heart rate due to its effects on autonomic nervous system activity and repolarization reserve[39]. The observed QT interval variations could be linked to heightened vagal activity during Phase 3 of the ventricular action potential. (Figure 6).

Figure 6: Shows the correlation of an EKG and the different electrical events comprising the ventricular action potential. The QT variable subsegments associated with specific electrical events within the action potential based on their ionic basis. The EKG interval measurement is depicted through vertical dashed lines that mark important points, including the QRS onset and offset (J point), T wave peak, and T wave offset. The action potential is divided into five phases (0-4), which result from changes in the balance of inward and outward ionic currents.

The iCEBc (Index of Cardiac Electrophysiological Balance) is a valuable noninvasive tool that has the potential to provide insight into the underlying factors leading to imbalances in the depolarization-to-repolarization ratio [40,41]. The QT interval primarily reflects ventricular repolarization, while the QRS complex represents ventricular depolarization. By analyzing these components in three groups, a cardiac electrophysiological balance could be determined. As depicted in Figure 5, the QTcb/QRS ratio was found to be significantly different in the SOR group compared to other measures like JT intervals (specifically JT/QRS). This finding indicates that a specific section of the repolarization segment may be responsible for this imbalance. The observed increase in ratio may be attributed to the inhomogeneities in the repolarization phase of cardiac cation potential, which is most impacted during periods of decreased heart rate in the SOR group. The results of Figure 3 support our hypothesis that the activation of the parasympathetic system contributes to changes in QTend and QTcb duration. The significantly lower QTend/QTcb ratio indicates a decrease in QTend duration, while the increase in QTcb duration further strengthens our argument for increased parasympathetic activity. This heightened vagal stimulation would have had the effect of decreasing heart rate and prolonging the repolarization phase, resulting in a longer QTcb interval. Therefore, it can be concluded that the observed decrease in QTend/QTcb ratio is most likely due to an increase in parasympathetic activity. In the SOR group, a reduction in QTend duration was observed may act as a protective measure against QT dispersion - a known precursor to potentially hazardous ventricular arrhythmias [42,43].

Our study has provided initial evidence linking parasympathetic-induced QTend shortening to SOR intervention in healthy adults. While these findings have important implications for understanding cardiac

electrophysiology and autonomic control, further research is necessary to confirm and expand upon our results. A larger sample size and diverse population would add more weight to our findings and help us fully understand the complex interaction between SOR intervention and QTend shortening. This provides a strong framework for future research in this field, paving the way for further exploration into the correlation between autonomic regulation and cardiac performance in OMT practice.

Conclusions

The effects of SOR manipulation on QT metrics showed variation in QT intervals and shortening of QTend intervals. There was a slight increase in QT and QTcb length, which may be due to changes in repolarization or a temporary decrease in heart rate caused by parasympathetic stimulation. Shortened QTend intervals can have a positive impact on heart stability by preventing QT dispersion. Post-SOR data revealed changes in repolarization parameters decoupling of QTe from JT intervals, leading to shorter QTend intervals, indicating an increase in parasympathetic activity. This, combined with a moderate increase in QTcb, can result in a longer effective refractory period and improved ventricular relaxation. These results demonstrate the potential benefits of SOR manipulation on cardiac function.

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