Abstract: Objective: To test the null hypothesis that there would be no difference in normalized levels of electromyographic (EMG) activity measured from rectus capitis posterior major (RCPM) muscles with the head held in a self-defined neutral position and with the head held in a retracted position (chin tuck).

Design: A 2x2 within-subjects factorial research design was used. Disposable bipolar fine wire hooked electrodes were used to collect EMG data from asymptomatic subjects. Mixed effects beta regression models were used to analyze the data.

Setting: Michigan State University Center for Orthopedic Research.

Participants: Asymptomatic subjects between the ages of 20-40 years.

Interventions: Not applicable.

Main Outcome Measures: Changes in normalized levels of EMG activity as a function of several covariates including head position.

Results: Normalized EMG activity of RCPM muscles, collected with the head held in a retracted position, was significantly higher (p < 0.0001) than normalized EMG activity collected with the subject's head held in a self-defined neutral position.

Conclusions: RCPM muscles are active when the head is held in a self-defined neutral position and show a significant increase in activity as the head is moved to a retracted position (chin tuck).

Keywords: rectus capitis posterior; headache; neck pain; biomechanics; chin tuck

List of Abbreviations:

OA occipito-atlantal
AA atlanto-axial
RCPm rectus capitis posterior minor
RCPM rectus capitis posterior major
RCP rectus capitis posterior
EMG electromyographic
FI fatty infiltration
FHP forward head position
NHP neutral head position
RHP retracted head position
MVIC maximum voluntary isometric contraction
WAD whiplash-associated disorders
<table>
<thead>
<tr>
<th>Acronym</th>
<th>Description</th>
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<tr>
<td>BMI</td>
<td>body mass index</td>
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<tr>
<td>OR</td>
<td>odds-ratio</td>
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<tr>
<td>MOR</td>
<td>median odds-ratio</td>
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<tr>
<td>LR</td>
<td>likelihood ratio</td>
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<tr>
<td>PRE</td>
<td>proportional reduction in error</td>
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<tr>
<td>ΔAIC</td>
<td>change in the Akaike information criterion</td>
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</table>
Allen W. Heinemann, PhD, ABPP
Editor, Archives of Physical Medicine and Rehabilitation

Dear Dr. Heinemann:

I would like to submit the manuscript entitled, “Clinical Implications of Activation of RCPm and RCPM Muscles During Voluntary Retraction of the Head (Chin Tuck)”. The work is original and has not been previously published or is it currently submitted to another journal. I confirm that I have had full access to all the data in the study and that I take responsibility for the integrity of the data and the accuracy of the data analysis as well as the decision to submit for publication.

The objective of the study was to test the null hypothesis that there would be no difference in normalized levels of EMG activity measured from RCPM muscles with the head in a self-defined neutral position and with the head in a retracted position (chin tuck). Activation levels of RCPM muscles with the head held in a self-defined neutral position and with the head held in a retracted position have not been previously reported.

We observed that normalized EMG activity of RCPM muscles, collected with the head held in a retracted position, was significantly higher ($p < 0.0001$) than normalized EMG activity collected with the subject’s head held in a self-defined neutral position. We hypothesize that a chin tuck protocol may contribute to the relief of head and neck pain by both stretching and strengthening RCP muscles.

Written permissions have been obtained and subject consent forms were collected.

Sincerely yours,

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Clinical Implications of Activation of RCPm and RCPM Muscles

During Voluntary Retraction of the Head (Chin Tuck)

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Acknowledgments

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Clinical Implications of Activation of RCPm and RCPM Muscles During Voluntary Retraction of the Head (Chin Tuck)

Abstract

Objective: To test the null hypothesis that there would be no difference in normalized levels of electromyographic (EMG) activity measured from rectus capitis posterior major (RCPM) muscles with the head held in a self-defined neutral position and with the head held in a retracted position (chin tuck).

Design: A 2x2 within-subjects factorial research design was used. Disposable bipolar fine wire hooked electrodes were used to collect EMG data from asymptomatic subjects. Mixed effects beta regression models were used to analyze the data.

Setting: Michigan State University Center for Orthopedic Research.

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Main Outcome Measures: Changes in normalized levels of EMG activity as a function of several covariates including head position.

Results: Normalized EMG activity of RCPM muscles, collected with the head held in a retracted position, was significantly higher ($p < 0.0001$) than normalized EMG activity collected with the subject’s head held in a self-defined neutral position.

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MVIC  maximum voluntary isometric contraction
WAD  whiplash-associated disorders
BMI  body mass index
OR  odds-ratio
MOR  median odds-ratio
LR  likelihood ratio
PRE  proportional reduction in error
ΔAIC  change in the Akaike information criterion
Introduction

Voluntary retraction of the head, commonly referred to as a chin tuck, is a physiologic motion involving posterior translation of the head within a sagittal plane without rotation that is commonly used in the rehabilitation clinic to address a variety of pain related syndromes. Voluntary retraction of the head results in flexion of both the occipitoatlantal (OA) and the atlantoaxial (AA) joints and selective stretching of rectus capitis posterior minor (RCPm) and rectus capitis posterior major (RCPM) muscles\(^1,2\) (See fig. 1).

The functional role of the rectus capitis posterior (RCP) muscles in performing daily activities is poorly understood. Bilateral contraction of RCP muscles is purported to result in extension of the head.\(^2\) However, their small size, relative to larger muscles acting in parallel with them, would minimize their contribution to gross extension of the head and neck.\(^3,4\) It is intriguing that RCP muscle pathology has been associated with syndromes that are characterized by chronic head and neck pain and yet little effort has been expended in understanding the functional significance of these muscles.

Fatty infiltration (FI) of RCP muscles has been reported in some whiplash patients.\(^5,6,7\) While the cause of the fatty infiltration in these patients is unknown, it has been reported that headache intensity, duration, and frequency increase as the cross sectional area of the muscles decreases\(^8\), and that the progression of FI over time is directly related to pain and disability.\(^9\) FI would be expected to result in changes in both the active and passive characteristics of the muscle. The loss of functional capability/capacity would compromise the ability of these muscles to contribute to the performance of daily activities as a result of mechanical impairment and/or loss of proprioceptive components.
Functional connections between the pain sensitive spinal dura and RCP muscles have been described.\textsuperscript{10,11,12,13} Functional connections between the pain sensitive spinal dura and RCP muscles might be expected to reduce the ability of RCP muscles to limit and/or prevent dural enfolding during daily activities, and could result in clinical manifestations such as head and neck pain.\textsuperscript{14}

Head posture, specifically a forward head posture (FHP), is one of several factors that are associated with head and neck pain\textsuperscript{15,16}, and it has been shown that people with chronic neck pain have a reduced ability to maintain a neutral cervical posture when seated.\textsuperscript{17} Trigger points in RCP muscles are associated with chronic, tension-type headache intensity and frequency and are directly correlated with a FHP.\textsuperscript{18,19} It is unknown if FHP is a consequence of head and neck pain or if it is in part responsible for head and neck pain. Treatment protocols such as a chin tuck are purported to reduce head and neck pain by promoting a neutral cervical posture. Reports estimate that 10-15\% of level of pain and disability could be attributed to head posture if a causative relationship could be established.\textsuperscript{20}

Normalized levels of EMG activity in RCPm muscles have been shown to significantly increase as subjects move their head from a self-defined neutral head position (NHP) to a retracted head position (RHP)\textsuperscript{21} (See fig. 2). Activation levels of RCPM muscles with the head in a self defined neutral position and with the head in a chin tuck position have not been previously reported. A better understanding of the response of RCP muscles in asymptomatic subjects as they perform a chin tuck protocol would help clarify why a chin tuck is associated with reducing chronic head and neck pain in some groups of chronic head and neck pain patients. With this in mind, we set out to test the null hypothesis that there would be no difference in normalized levels of EMG activity measured from RCPM muscles with the head in a neutral position and with the head in a retracted position.

\textbf{Materials and Methods}
Research Design

We used a 2 x 2 within-subjects factorial research design, supplemented by two additional covariates—namely sex and body mass index (BMI)—to study RCPM muscle activation. We recorded EMG data from the RCPM muscles during multiple trials in each head position (NHP vs. RHP) and on each side of the body (left vs. right) for each participant.

Participant Enrollment

We recruited participants by advertising the study to 200 second year osteopathic medicine students at Michigan State University via e-mail. Forty-three students responded to the advertisement, but only 20 met the enrollment eligibility criteria for this study. To ensure that study results would pertain to the population of adults with normal functioning of the upper cervical spine, our inclusion criteria required subjects who (1) had no head and neck pain at the time of participation, (2) had full range of motion for the upper cervical spine (i.e., no significant restrictions), and (3) were 20-40 years of age. The age criterion excludes adults who may have begun to develop age-related muscle atrophy, which can reduce muscle mass by 30-50% among people 45-80 years old. Michigan State University’s Institutional Review Board approved this study and the consent forms used in enrollment.

After the principal investigator (RCH) explained the study protocol to eligible subjects and answered any questions, participants who signed the informed consent form were enrolled. All enrolled participants received $150, even if they were unable to complete the full protocol. We enrolled 20 participants (13 males and 7 females), whose means and standard deviations on several characteristics can be summarized as follows: age 25±2 years; height 177±12 cm; weight 74±15 kg; and body mass index (BMI) 24±4.

Instrumentation
Data collection occurred at the Michigan State University Center for Orthopedic Research. Physical data (e.g., sex, height, weight) were collected after completing the enrollment procedure described above. Each participant was then seated in a cervical flexion/extension device\(^a\). The seat’s height and anterior/posterior position were adjusted to align the articular pillars of C3 with the rotational axis of the device’s movement arm.\(^{21}\)

Each participant was then placed prone on a standard examination table with a towel under his or her chest to flex the head and neck slightly and increase access to the suboccipital region. Next, the physicians (LLP and JJR) used a color Doppler ultrasound unit to screen for congenital blood vessel anomalies that might put participants at risk during electrode insertion. They then used a standardized protocol to insert hooked EMG electrodes\(^b\) made of 25 gauge disposable bipolar stainless steel fine wire into both the right and left RCPM muscles.\(^{23}\) The physicians also placed a ground electrode just above the middle of the spine of the right scapula. Some data that we collected from the RCPm muscles of these same participants have already been published,\(^{21}\) and we plan to use additional data from other muscles (See fig. 3) in future papers.

Muscle function is often estimated from electromyographic (EMG) voltages. Voltages collected from surface electrodes tend to be influenced by cross talk either from adjacent or more superficial muscles, so intramuscular electrodes are the preferred method for quantifying EMG voltages from small, deep muscles. Naturally, the challenge is to accurately insert electrodes into the muscles one wants to study. Fortunately, palpable, anatomic landmarks can be used to safely guide insertion of intramuscular electrodes into the RCPM muscles of human participants with high accuracy\(^{23}\).

The external wires connecting the electrodes to the recording equipment were securely taped to the participant’s body prior to returning the participant to the cervical flexion/extension device. Torso movement was minimized by adjusting the device’s restraints to press the upper torso against the back of the seat.\(^{21}\) Prior to data collection,
we tested the electrodes by asking participants to lightly push their heads back against the experimenter’s hand while we monitored the signal voltage. Noisy or missing signals were then noted and corrected when possible.

Data Collection

Our goal was to examine normalized activation levels of RCPM muscles as a function of head position. Normalization provides a metric for measurements that facilitates comparing data across different muscles and participants. Each participant performed 3 maximal voluntary isometric contraction (MVIC) trials, 5 neutral head position (NHP) trials, and 4 retracted head position (RHP) trials. Each trial produced separate observations on both sides of the body, yielding 2 observations per subject.

Signal Processing

Raw EMG voltages were digitized (3,000 samples/sec) after amplifying them and passing them through a 20 Hz to 1500 Hz bandpass filter. A Matlab® program was used to digitally rectify the data, to removed offset voltages and QRS waves, and to apply a dual pass, 4th order Butterworth filter at a low-pass cut-off frequency of 0.5 Hz.

Establishment of a Self-Defined, Neutral Head Position

Data from five trials where the participants maintained a self-defined, neutral head position (NHP) provided the reference against which we compared muscle activation observed while the head was retracted. In order to establish a self-defined, neutral head position, each participant grasped the seat handles, closed both eyes, rocked his or her head forward and backward across a comfortable full range of motion, then gradually decreased the range of movement until the head settled into a comfortable resting position that was defined as
the NHP.\textsuperscript{26,27} We collected 3 seconds of EMG voltage data from both the left and right RCPM muscles for each of the 5 trials.

Maximum Voluntary Isometric Contraction

We adjusted the cervical device movement arm to place the resistance pad just superior to the participant’s inion and used a head strap to secure the participant’s head against the pad.\textsuperscript{21} Starting with the participant’s head in a NHP, we locked the movement arm in place and asked the participant to perform three MVIC trials against the fixed resistance pad, with 10 seconds of rest between trials. EMG data was collected for 5 seconds per MVIC trial. We used the maximum EMG voltage observed for each side of the body across these trials as the denominator for normalizing the muscle activation data from both NHP and retracted head position (RHP) trials. After completing the MVIC trials, the cervical flexion/extension device was adjusted to permit unrestricted head retraction before moving on to the remaining trials.

Neutral and Retracted Head Position Trials

Each participant alternated between a NHP and a RHP, performing a total of four RHP trials. During these trials, each participant grasped the seat handles, then glided their head from the NHP into a RHP (chin-in) and held that position before returning to the NHP. We collected EMG voltage data from both the left and right RCPM muscles for 5 seconds per trial while the participant maintained the RHP. After completion of all NHP and RHP trials, the participant exited the cervical flexion/extension device and returned to the examination table, where the physicians (LLP and JJR) removed all the electrodes.

Outcome Measure: Muscle Activation

Normalized muscle activation was the proportion resulting from dividing the raw voltage observed during a trial by the relevant MVIC; these values spanned the range [0.01133, 0.98549]. We used Zimprich’s Equation 3\textsuperscript{28} to
rescale those values onto the open unit interval (0,1) prior to analysis, thereby compressing the distribution to
prevent values of exactly zero or one without altering its basic shape. This transformation facilitated applying
beta regression in our previous paper\textsuperscript{21}, so we applied it here for methodological consistency. Rescaled muscle
activation spanned the range \([0.01323, 0.98361]\).

\textit{Predictors}

Both head position (0=NHP, 1=RHP) and side of the body (0=left, 1=right) were binary variables. Head position
is the focal predictor in this study. Because the RCPM muscles are paired structures, collecting data on both
groups of the body during each trial increased the efficiency of the research design, allowed us to test for
asymmetric muscle activation, and to determine whether the effect of head position depends on which side of
the body one observes. As in our previous paper\textsuperscript{21}, we used sex (0=female, 1=male) and body mass index (BMI)
as additional covariates to predict muscle activation. We grand-mean centered BMI to avoid numerical
estimation problems\textsuperscript{29} and simplify interpreting the results.\textsuperscript{30}

\textit{Data Screening}

Each participant could contribute up to 18 observations to the dataset (9 trials x 2 sides of the body x 20
participants = 360 observations possible), but missing or unusable data reduced the sample size. We analyzed
258 observations from 15 participants, which means we retained 75\% of the 342 observations collected from the
19 participants who could tolerate the electrodes. We had complete, usable data from 12 participants, partial
data from another 3 participants, and entirely missing or unusable data for 5 participants (#2, #3, #7, #9, and
#10). We describe our screening criteria below, which are similar to those used in our previous paper.\textsuperscript{21}

Participant #2 was unable to tolerate insertion of the electrodes at all, so we could not collect any data. All data
from participants #3, #7, #9, and #10 were excluded from the analysis due to what we later concluded were
either dislodged or improperly placed electrodes (probably not in the RCPM muscles) or other equipment malfunctions. All voltages for participant #3’s RHP trials were implausibly large outliers, as were all the MVIC voltages on the left side for this participant. Participant #7’s RHP trials all yielded muscle activation greater than 100% of MVIC and all but 1 of the corresponding NHP trials yielded unsustainably high levels of static muscle activation. All the NHP trials for participants # 9 and #10 also yielded unsustainably high levels of static muscle activation.

We discarded all trials associated with a particular electrode when (1) the MVIC voltage was very close to zero, (2) all NHP and RHP voltages recorded were either larger than MVIC, or (3) or all NHP trials resulted in muscle activation greater than 30% MVIC. We reasoned that these situations were the result of inaccurate MVIC voltages. Consequently, left side data for participant #19 were discarded. Finally, we also discarded a few observations that we believe reflected problems affecting only single trials. Specifically, we discarded two NHP observations (left side for #11, right side for #19) that yielded muscle activation greater than 40% MVIC and one RHP trial (right side for #13) that yielded muscle activation greater than 100% MVIC.

Statistical Analysis

We applied a mixed effects beta regression model to explicitly account for (1) measuring muscle activation as a proportion normalized by MVIC, (2) the non-independence of the observations, (3) the skewed distribution of muscle activation, and (4) the greater variance among RHP trials than among NHP trials. This is a two-part model. Our location submodel predicts mean muscle activation as a function of head position, side, sex, and BMI, plus a head position X side interaction. We added a random intercept in the location submodel to account for the correlation among the observations from the same person. The skewed, heteroscedastic distributions for the muscle activation data (See fig. 4) illustrate why our dispersion submodel predicts the variance of muscle activation as a function of head position, side, and a head position X side interaction.
We tested whether the full model fit better than a null model containing only fixed intercepts via a likelihood ratio (LR) test and examining change in the Akaike information criterion (\( \Delta AIC \)). We report the pseudo-\( R^2 \) to quantify variance explained by the location submodel and the proportional reduction in error (\( PRE \)) relative to the null model as another, possibly superior, measure of global model fit. Both pseudo-\( R^2 \) and PRE always have values of zero for a null model.

We set the criterion for statistical significance at \( \alpha = .05 \), measured each predictor’s effect on mean muscle activation via an odds-ratio (OR), and report the median odds-ratio (MOR) to interpret the location submodel random intercept. Graphs of the predicted means and standard deviations of muscle activation—broken down by head position, side of the body, and sex—illustrate our results. The predicted values used for graphing represent results for a participant who is average with respect to both BMI and the random effect on the location submodel intercept.

Results

The aim of this project was to test the null hypothesis that there would be no difference in normalized EMG activation levels of RCPM muscles with the head in a self-defined neutral position and with the head in a retracted position. Table shows the results of the mixed beta regression model we used to test that hypothesis. That model fit the data far better than the null model, LR(9) = 309.8, \( p < .0001 \), \( \Delta AIC = 291.8 \). It explains 62% of the variance in muscle activation (pseudo-\( R^2 = 0.62 \)) and it reduces prediction error by 51% (\( PRE = 0.51 \)). The predicted values are consistent with the pattern of the medians, the skewness, and the heteroscedasticity of the raw data. The significant random intercept (\( \sigma_{u0} = 0.56 \) on the logit scale, \( p = .0001 \), MOR = 1.71) tells us that there is moderate variability in average muscle activation from person to person. For any pair of randomly selected participants, one will typically have about 71% higher odds of muscle activation than the other.
Head position had a strong main effect on mean level of muscle activation, \((p < .0001, \text{ OR} = 3.86)\). Figure 5 shows that mean levels of muscle activation were substantially higher during RHP trials (ranging from 26% to 37% of MVIC) than during NHP trials (ranging from 10% to 15% of MVIC). Neither side of the body nor sex had significant effects on mean muscle activation. There was no evidence for a head position X side interaction effect, so the head position effect is similar regardless of which side of the body one observes. However, BMI had a small, negative effect on mean levels of muscle activation \((p = .0185, \text{ OR}=0.92)\), indicating that increasing BMI is associated with decreased mean levels of muscle activation.

Consistent with the heteroscedasticity shown in Figure 4, all effects in the dispersion submodel were significant. Figure 6 illustrates the significant head position X side interaction \((p < .0001)\) effect on the variance of muscle activation. The interaction effect is perhaps best described by noting that the standard deviation for RHP trials on the left side (20% to 21%) is 6.3 to 6.7 times larger than the one for NHP trials on the left side (3%), but on the right side of the body the standard deviation for RHP trials (12% to 13%) is only about 1.9 to 2.0 times larger than the one for NHP trials (6% to 7%).

**Discussion**

A chin tuck protocol is known to selectively stretch the RCP muscles\(^1,2\) and is commonly used in the rehabilitation clinic to address a variety of head and neck pain issues. The response of RCP muscles as they are stretched during a chin tuck protocol has not been extensively researched. We have shown, for the first time, that normalized EMG activity recorded from RCPM muscles significantly increases from a level of 10% to 15% of MVIC with the head held in a NHP to a level of 26% to 37% of MVIC \((p < 0.0001)\) as a chin tuck is performed. Normalized levels of EMG activity in RCPm muscles have previously been shown to significantly increase as subjects move their head from a self-defined NHP to a RHP.\(^{21}\) These two observations show that a
chin tuck results in eccentric contraction of RCP muscles. Exercises involving eccentric contraction are designed to force a muscle to lengthen as it is activated, and are known to enhance muscle size, strength, and composition.\textsuperscript{35,36} This suggests that chin tuck exercises would both stretch and strengthen RCP muscles.

Biomedical models have shown that short, deep muscles such as the multifidi are necessary to maintain stability of the lumbar spine.\textsuperscript{37} We have shown that RCP muscles are active at a level of 10% to 15% of the MVIC when the head is held in a self-defined NHP, suggesting that they contribute to maintaining a normal, neutral head posture and guard against movements caused by unexpected external forces.\textsuperscript{38,39,40}

Head posture, specifically a forward head posture (FHP), is one of several factors that are associated with head and neck pain\textsuperscript{15,16}, and it has been shown that people with chronic neck pain have a reduced ability to maintain a neutral cervical posture when seated.\textsuperscript{17} Trigger points in RCP muscles are associated with chronic, tension-type headache intensity and frequency and are directly correlated with a FHP.\textsuperscript{18,19} It has been suggested that FHP in these patients is a consequence of RCP muscle hypertonicity that may result in the patient protruding the chin to relieve stress in RCP muscles.\textsuperscript{41,42} A retracted head position may reduce hypertonicity in these muscles and resolve the active trigger points by stretching of the RCP muscles. Resolution of the trigger points would be expected to be accompanied by patients regaining a normal neutral head posture as nociceptive input from the trigger points into the trigeminal nerve is reduced.\textsuperscript{43}

FI of RCP muscles has been reported in some patients diagnosed with whiplash-associated disorders (WAD).\textsuperscript{5,6,7} While the cause of FI in these patients is unknown, it has been reported that headache intensity, duration, and frequency increase as the cross sectional area of the muscles decrease.\textsuperscript{8} Progression of FI over time has been shown to be directly related to pain and disability.\textsuperscript{9} Balance disturbances indicative of somatosensory impairment have been reported in patients diagnosed with WAD.\textsuperscript{44} The high density of muscle
spindles found in RCP muscles would suggest that they provide significant proprioceptive feedback to the CNS that is directly related to position and motion of the head.\(^{39,40,45}\) It is possible that muscle weakness and kinesthetic deficits are both a consequence of FI.\(^{46}\) If FI in RCP muscles is not due to neurogenic atrophy, a chin tuck exercise protocol might be expected to reduce balance disturbances associated with WAD as weakened RCP muscles are activated and strengthened.

Functional connections between the pain sensitive spinal dura and RCP muscles have been described.\(^{10,11,12,13}\) Subsequent work has suggested that this myodural bridge may function to prevent or limit enfolding of the pain sensitive dura mater during normal movements of the head.\(^{47,48,49}\) FI of RCP muscles would be expected to result in muscle weakness that may reduce their ability to limit and/or prevent dural enfolding during daily activities. A chin tuck exercise protocol which restores a normal functional relationship between the RCP muscles and the spinal dura might reduce head and neck pain originating from the spinal dura.

The study has several weaknesses. One would be the absence of EMG data from deep cervical flexors such as the rectus capitis anterior (RCA) and rectus capitis lateralis (RCL) muscles. Access to these muscles with fine wire electrodes was not considered to be feasible because of either an unacceptable level of risk related to hitting a critical vascular structure or that the protocol would not be tolerated by the subjects. Another weakness was the size of the final data set. We ended up discarding 25% of the data due to dislodged/noisy electrodes or implausible values. Finally, we do not know the actual magnitude of the angle of either the OA or the AA joints with the head in the NHP nor do we know the actual increase in these angles as the head is retracted.

**Conclusions**

RCPM muscles are active when the head is held in a self-defined neutral position and show a significant increase in activity as the head is moved to a retracted position (chin tuck). We hypothesize that the clinical
benefit of a chin tuck protocol is to both stretch and strengthen the RCPm and RCPM muscles. Future work will attempt to quantify the clinical benefit of a chin tuck exercise to strengthen RCP muscles in head and neck pain patients.
References


http://dx.doi.org/10.1016/j.spinee.2013.06.011.


**Suppliers:**

a. Atlantis Engineering, 14232 Marsh Lane, Suite 408, Addison, Texas 75001

b. Chalgren Enterprises, Inc., 380 Tomkins Court, Gilroy, CA 95020-3631

c. The Mathworks, 3 Apple Hill Drive, Natick, MA 01760-2098

**Figure Legends:**
Figure 1. The image on the left shows the orientation of C0, C1, and C2 with the head held in a self-defined neutral position. The image on the right shows the head in a retracted position accompanied by flexion of the OA and AA joints, and elongation of RCPm and RCPM muscles.

Figure 2. Processed EMG voltages from right and left RCPm muscles during voluntary retraction of the head from a NHP. The signals form an alternating sequence of 5 NHP trials (valleys) and 4 RHP trials (peaks) from Subject #8 is shown.

Figure 3. Securing of electrode wires prior to collection of EMG data.

Figure 4. Boxplots of muscle activation by side of the body and head position. Filled circles mark the medians. Asymmetry around the medians indicates skewed distributions; varying heights of the boxplots highlight non-constant variance (i.e., heteroscedasticity). RCPM = rectus capitis posterior major.

Figure 5. Predicted means and 95% confidence intervals by side of the body, head position, and sex. RCPM = rectus capitis posterior major.

Figure 6. Predicted standard deviations and 95% confidence intervals by side of the body, head position, and sex. Sex is included as a factor because variances estimated by a beta regression model are indirectly influenced by the predictors in the location submodel. RCPM = rectus capitis posterior major.
Figure 1: The image illustrates the difference between a neutral head position and a retracted head position. In the neutral position, the head is in its natural orientation, while in the retracted position, the head is pulled back, as indicated by the "RCPMi" label. The transition from neutral to retracted is represented by the arrow.
Figure 4

Box plots showing rescaled muscle activation for Left RCPM and Right RCPM in neutral and retracted head positions.

- **Left RCPM**
  - Neutral: Lower activation
  - Retracted: Higher activation

- **Right RCPM**
  - Neutral: Lower activation
  - Retracted: Slightly higher activation
Table. Parameter estimates, confidence intervals, and odds-ratios from the mixed effects beta regression model. The random intercept parameter was modeled as a standard deviation to guarantee a positive value for the corresponding variance. \( N = 258 \) observations; \( J = 15 \) participants. AIC = Akaike information criterion; \( df = \) degrees of freedom; Full = full model; LR = likelihood ratio; Null = null model; PRE = proportional reduction in error; SE = standard error; \( t = t \)-statistic.

* \( p < .05 \), ** \( p < .01 \), *** \( p < .001 \), **** \( p < .0001 \).

\( ^a \) This is the median odds-ratio (MOR) and its confidence interval.

<table>
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<tr>
<th>Parameter</th>
<th>Estimate</th>
<th>SE</th>
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<th>( t (df = 16) )</th>
<th>OR</th>
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<tr>
<td>( \beta_0 ): Intercept</td>
<td>-1.87</td>
<td>0.24</td>
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<td>0.09</td>
<td>[0.09, 0.26]</td>
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<tr>
<td>( \beta_1 ): Head position</td>
<td>1.35</td>
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<td>[1.07, 1.63]</td>
<td>10.45 ****</td>
<td>4.56</td>
<td>[2.93, 5.09]</td>
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<td>(HP)</td>
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<td>( \beta_2 ): Side of the body (S)</td>
<td>0.10</td>
<td>0.08</td>
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<td>1.27</td>
<td>1.43</td>
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<td>( \beta_3 ): Sex (Male)</td>
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<td>0.30</td>
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<td>( \beta_4 ): Centered BMI</td>
<td>-0.09</td>
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<td>-2.66 *</td>
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<td>0.11</td>
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<td>1.57</td>
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Basic Science

Electromyographic activity of rectus capitis posterior minor muscles associated with voluntary retraction of the head

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Abstract

BACKGROUND CONTEXT: The functional role of rectus capitis posterior minor (RCPm) muscles is not well defined. To the best of our knowledge, electromyographic (EMG) data from RCPm muscles in humans have never been collected and analyzed.

PURPOSE: To test the null hypothesis that there will be no difference in normalized levels of EMG activity measured from RCPm muscles with the head in a neutral position and with the head in a retracted position.

STUDY DESIGN: A repeated measures design intended to quantify normalized levels of EMG activity measured from RCPm muscles.

METHODS: Disposable 25-gauge, bipolar wire hooked electrodes were used to collect EMG data from both right and left RCPm muscles from 17 asymptomatic subjects. Data were collected while subjects performed five trials with the head maintained in a neutral position; performed three maximal voluntary isometric contraction efforts; performed four trials with the head maintained in a retracted position. Mixed effects beta regression models were used to analyze the data.

RESULTS: Normalized EMG activity of RCPm muscles collected with the subject’s head held in a retracted position was significantly higher ($p<.0001$) than normalized EMG activity collected with the subject’s head held in a self-selected, neutral position.

CONCLUSIONS: Rectus capitis posterior minor muscles are active when the head is held in a neutral position and show a significant increase in activity when the head is held in a retracted position. © 2013 Elsevier Inc. All rights reserved.

Keywords: Rectus capitis posterior; Electromyography; Neck muscles; Functional role; Head retraction

Introduction

Rectus capitis posterior minor (RCPm) muscles arise from the posterior tubercle on the posterior arch of the atlas and insert on the occipital bone inferior to the inferior nuchal line and lateral to the midline [1]. The orientation of the RCPm muscles is such that there is no significant wrapping around bony or muscular structures. Rectus capitis posterior minor muscles are the only muscles that attach...
to the posterior arch of the atlas. Based on their anatomical location, rectus capitis anterior muscles would appear to be antagonist to RCPm muscles.

The functional significance of RCPm muscles has not been clearly defined. Bilateral contraction of RCPm muscles is purported to result in extension of the head [2]. However, their small size, relative to larger posterior muscles acting in parallel, would seem to minimize their contribution to gross extension of the head and neck [3,4]. The high density of muscle spindles found in RCPm muscles in both the cat and humans would suggest that they provide significant proprioceptive feedback to the central nervous system, related to position and motion of the head [5–8]. It has also been suggested that a fibrous connection to the spinal dura enables RCPm muscles to monitor/control dural tension during flexion and extension of the head and neck [9,10].

Voluntary retraction of the head is a physiologic motion involving posterior translation of the head within a sagittal plane without rotation (Fig. 1). This motion increases posterior loading of the superior articular facets of the atlas, resulting in flexion of the occipitoatlantal (OA) joint and elongation of the RCPm muscles [11,12]. Comparison of RCPm muscle activity when the head is held in a neutral position with muscle activity when the head is held in a retracted position would help to clarify the functional role of these muscles. Increased understanding of the functional role of RCPm muscles should provide insight into head and neck pain that may be related to pathology resulting from fatty infiltration of these muscles.

To the best of our knowledge, electromyographic (EMG) data from RCPm muscles in humans have never been collected and analyzed. The goal of this project was to test the null hypothesis that there will be no difference in normalized activation levels of RCPm muscles with the head in a neutral position and with the head in a retracted position.

Materials and methods

Subject enrollment

Potential subjects were recruited from within the second year student population of the College of Osteopathic Medicine at Michigan State University. We sent an e-mail describing the project to 200 students. Forty-three individuals responded, of which 20 were eligible to enroll in the study. Our inclusion criteria required subjects to be free of headache and neck pain and have no significant motion restrictions in the region of the upper cervical spine. We also required subjects to be between the ages of 20 to 40 years because muscle atrophy due to aging results in a 30% to 50% decrease in muscle mass between the ages of 45 to 80 years [13]. The study was approved by Michigan State University’s Institutional Review Board.

The project leader (RCH) explained the research protocol to each potential subject and addressed their questions. Subjects willing to proceed with the study signed an institutional review board–approved informed consent form. We paid consenting subjects $150 for participation in the study, regardless of whether they completed the protocol or not. The study cohort consisted of 13 males and seven females. The means and standard deviations for demographic variables were as follows: weight, 74±15 kg; height, 177±12 cm; age, 25±2 years; and body mass index (BMI), 24±4.

Subject instrumentation

The study was conducted at the MSU Center for Orthopedic Research. On arrival at the center, the research protocol was reviewed with the subject and physical data were collected (eg, sex, height, weight). The subject was then seated in a PRseries EWC-40, cervical flexion/extension device (Atlantis Engineering, Dallas, TX, USA). The height and anterior/posterior position of the seat were adjusted so that the articular pillars of C3 were in approximate alignment with the axis of rotation of the device’s movement arm (Fig. 2).

The subject then exited the cervical flexion/extension device and assumed a prone position on a standard examination table. A towel was placed between the subject’s

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Fig. 1. Orientation of the occiput, the atlas, and the axis with the head in a self-defined, neutral head position and full voluntary retraction of the head [11]. RCPm, rectus capitis posterior minor.

Fig. 2. Subject position for maximum voluntary isometric contraction efforts from the neutral head position.
chest and the examination table so that the head and neck were slightly flexed. This facilitated insertion of the fine wire, EMG electrodes by increasing access to the suboccipital region. A color Doppler ultrasound unit was used by the physicians (LLP and JJR) to confirm that the subject did not have congenital blood vessel anomalies that might put them at risk during insertion of the fine wire EMG electrodes. After prepping the suboccipital region with rubbing alcohol, a standardized protocol was used to guide insertion of 25-gauge, disposable bipolar stainless steel fine wire hooked EMG electrodes (Chalgren Enterprises, Inc., Gilroy, CA, USA) into right and left RCPm muscles [14]. A ground electrode was placed just above the middle of the spine of the right scapula. Data were simultaneously collected from several other muscles, but those data are not included in this article.

Electromyographic voltages are commonly used to evaluate muscle function in both healthy and injured individuals. In contrast to surface electrodes, intramuscular electrodes help to minimize cross talk from adjacent and/or more superficial muscles, a desirable characteristic when attempting to quantify EMG voltages from small, deep muscles. Insertion of intramuscular electrodes into RCPm muscles of human subjects using readily palpable, common anatomic landmarks has been reported to be both accurate (81% of 181 insertions) and safe [14]. Ultrasound-guided insertion of intramuscular electrodes into obliquus capitis inferior muscles of the nonhuman primate has recently been reported [15].

After insertion of the electrodes, the external wires were securely taped to the subject’s body, and the subject returned to the cervical flexion/extension device. The torso restraint was adjusted to force the upper torso against the back of the seat to minimize movement of the torso (Fig. 2). Viability of the inserted electrodes was assessed by having the subject push lightly against a hand placed behind their head. Lack of signal or excessive noise on the signal was noted and corrected when possible.

Collection of data

We collected EMG data to quantify changes in normalized activation levels of RCPm muscles as a function of voluntary positioning of the head. With a subject in the seated position, data were collected while each subject:

- Maintained a self-selected, neutral head position (NHP).
- Maintained maximal voluntary isometric contraction (MVIC) efforts.
- Maintained a retracted head position (RHP).

Raw EMG voltages were amplified, passed through a bandpass filter (20 to 1,500 Hz), and digitized at 3,000 samples/sec. Software, written for Matlab (The Mathworks, Natick, MA, USA), was used to digitally rectify the data, remove offset voltages and QRS waves, and filter at a low-pass cutoff frequency of 0.5 Hz (dual pass, 4th order Butterworth filter). Maximum values obtained from the three MVIC efforts (MVIC-MaxLeft and MVIC-MaxRight) for each subject were used to normalize (%) MVIC data collected with the subject’s head in NHP and with the subject’s head in RHP to facilitate the comparison of data between muscles and among subjects [16,17].

Self-selected neutral head position

A self-selected NHP was used as a reference position for all tests. While maintaining a light grasp on the hand holds, subjects were instructed to close their eyes and nod their head in a forward and backward direction. The objective was to start from a comfortable full range of motion, and, by incrementally decreasing the range of movement, gradually return the head to a comfortable position, which was defined as NHP [18,19]. Each subject was instructed to maintain NHP for 3 seconds while EMG data were collected. Electromyographic voltages, which were recorded from the left and right sides during these trials, were normalized using MVIC-MaxLeft and MVIC-MaxRight (see below).

Maximum voluntary isometric contraction

We adjusted the resistance pad on the cervical device movement arm so that it was at a level just superior to the inion. A head strap held the resistance pad securely to the subject’s head. The subject was instructed to assume NHP. The movement arm was locked in place and subjects were instructed to perform a series of three MVIC efforts against the fixed resistance pad. Each of these efforts was held for a period of 5 seconds while data were collected. The subject was allowed a 10-second rest period between each of these three efforts. For each subject, we treated the largest EMG voltage (MVIC-Max) observed across these three trials as the maximum voltage that the subject could attain during an MVIC effort. We measured MVIC-Max separately for the left and right RCPm muscles in each subject, then used those voltages to normalize muscle activation levels for both the neutral and retraction trials.

Head retraction

The movement arm, along with the resistance pad, was moved into a position that would allow unrestricted retraction of the head. The subject was instructed to lightly grip the handles by the sides of the seat and assume NHP. The subject was then instructed to glide their head into a “chin-in” position for a retraction trial. The subject was instructed to hold this position for 5 seconds. The subject then returned to NHP for 5 seconds. These motions were repeated four times for each trial. Data were collected continuously during the period required to complete the protocol. After completion of all trials, the subject exited
the cervical flexion/extension device, was positioned in a prone position, and the fine wire electrodes were removed. Each pair of electrodes was examined to confirm that both wires had been removed.

Research design

We used a full factorial, within-subjects design: 2 head positions (NHP vs. RHP) × 2 sides of the body (left vs. right). Each subject completed trials in all four cells of the 2 × 2 design. We also used subject sex and BMI as covariates.

Outcome measure: muscle activation

We normalized muscle activation by dividing the raw voltage measurements observed during each trial by the MVIC-Max voltages to convert them into proportions (multiplying by 100 would thus make them percentages). Because muscle activation values are proportions, they are strictly bounded to fall inside the closed unit interval (0,1). Values of exactly zero and one are valid values for raw proportions. We rescaled muscle activation to fall on the open unit interval (0,1) using Zimpich’s equation 3 [20]. This linear transformation compresses the values slightly (values of exactly zero or one can no longer occur) and makes it possible to analyze the data with beta regression models [20–22] but does not otherwise alter the shape of the distribution. Before rescaling, the muscle activation values ranged from 0.00000 to 0.98464; the rescaled values ranged from 0.00195 to 0.98276.

Predictors

We included several predictors of muscle activation in the analysis. Head position, the focal variable of interest, was a binary variable (0=NHP, 1=RHP). Including this variable allowed us to compare muscle activation between the neutral and retracted positions. We included side of the body (0=left, 1=right) because data were collected from both the left and right RCpms muscles in each subject during each trial. This predictor allowed us to test for lateral asymmetry in both muscle activation levels observed on each side of the body (ie, a main effect for side) and the size of muscle activation differences between neutral and retracted positions (ie, a direction × side interaction), which could reveal whether the effect of head retraction on muscle activation depends on the side of the body observed.

We controlled for possible sex differences with respect to levels of muscle activation by including sex (0=female, 1=male) as a covariate. Similarly, we reasoned that because BMI may be correlated with physical fitness [23], it may also be correlated with muscle activation. Therefore, we used a grand-mean centered version of BMI as a continuous covariate. We centered BMI to avoid numerical estimation problems associated with running beta regressions containing predictors that have absolute values greater than 30 [22] and make the intercept coefficients more interpretable [24,25].

Data screening

Each subject performed five NHP trials and four RHP trials. Each trial produced separate observations on both sides of the body, yielding 18 observations per subject. One of the 20 subjects enrolled in the study (#2) was unable to tolerate insertion of the fine wire EMG electrodes. All data for two subjects (#3 and #19) were excluded from the final analysis. All the neutral trials for subject #3 yielded plausible muscle activation values. However, all the retraction trials yielded voltages a couple orders of magnitude larger than those observed in other subjects and also yielded muscle activation greater than 100% MVIC-Max. Meanwhile, all but one subject #19’s neutral trials yielded muscle activation greater than 40% MVIC-Max (which is an unsustainable long-term, static level of activation [26]), and all of subject #19’s retraction trials yielded muscle activation greater than 100% MVIC-Max. We concluded that either the electrodes were not properly placed in the RCpm muscles or the equipment malfunctioned during administration of the protocol for these two subjects. Therefore, we discarded those data before conducting our analyses.

When a MVIC-Max value was very close to zero, or all voltages recorded from the same electrode were either larger than MVIC-Max, or all neutral trials resulted in muscle activation greater than 30% MVIC-Max, we concluded that the MVIC-Max value was probably inaccurate. We then discarded all trials associated with that electrode (ie, all trials on that side of the body for that subject) in any of these situations. Consequently, left side data for subjects #5, #6, #7, and #10 were discarded for these reasons, along with right side data for subject #9.

We also discarded a few other individual observations because of implausible values for muscle activation. Three retraction observations yielded muscle activation greater than 100% MVIC-Max (one trial on the left for #8; two trials on the right for #11). One neutral trial (#11, left side) that yielded muscle activation greater than 40% MVIC-Max was also rejected.

Ultimately, we included 257 observations from 17 different subjects in the analyses reported below. That means 75% of the observations from those subjects were retained and 25% were discarded.

Statistical analysis

Each subject completed multiple trials in each cell of the within-subjects design, so the observations were not independent. Furthermore, muscle activation values were both positively skewed and heteroscedastic (ie, their variance is not constant across cells in the research design).
Retraction trials had larger skewness and variance than the neutral trials. So, we used a mixed effects beta regression to analyze the data. Beta regression is a rather new method [21] but an excellent choice for analyzing these data for several reasons. First, it always produces fitted values that respect the bounded nature of proportions [21]. Second, the results can be easily transformed back into the original metric of muscle activation [21]. Third, using a beta distribution for the residuals accommodates skewed distributions and explicitly modeling heteroscedasticity [21,22]. Finally, mixed effects variations of this model can properly account for nonindependent observations [20].

A beta regression model consists of a location submodel and a dispersion submodel. The location submodel models the mean of the outcome as a function of a set of predictors [21], and the dispersion submodel accounts for heteroscedasticity by modeling the variance of the outcome as a function of a set of predictors [21]. Our location submodel included fixed main effects for direction, side, sex, and BMI, plus a direction×side interaction. We added a random intercept in the location submodel to account for the nonindependence in our data. This allowed our model to separately estimate a between-subjects variance based on the normal distribution and a within-subjects residual variance based on the beta distribution [20]. Our dispersion submodel included fixed main effects for head position and side, plus a head position×side interaction to reflect the nonconstant variances observed in our exploratory graphs.

We reported several model fit indices. We used both a likelihood ratio test and the Akaike information criterion (AIC) to compare the full model to a null model containing only fixed intercepts (ie, no random effects and no predictors). A difference of 10 or more points indicates that the model with the smaller AIC fits the data better than the competing model [27]. The pseudo-\( R^2 \) [21] aims to measure the proportion of variance in muscle activation explained by the model, although this measure does not account for the predictors in the dispersion submodel [22]. Finally, we reported the proportional reduction in error (PRE) relative to the null model, which may be a better measure of global model fit for beta regression models than the pseudo-\( R^2 \) [22]. For the null model, both pseudo-\( R^2 \) and PRE have values of zero by definition.

A residual plot demonstrated good model fit and no violations of key assumptions. Therefore, we interpreted the results, using \( \alpha=0.05 \) as the criterion for statistical significance. We used the median odds ratio [28] to interpret the random intercept in the location submodel. This statistic represents the typical difference in propensity for muscle activation between two different, randomly selected subjects when we observe them in trials sharing the same head position and on the same side of the body. The median odds ratio will be 1 (its lower bound) when there is no between-subjects variance and greater than 1 when there is considerable between-subject variance. We used odds ratios (ORs) to measure effect sizes for the predictors in the location submodel; ORs greater than 1 indicate increased propensity for muscle activation as a result of switching from the reference category to the other category for the binary predictors (head position, side of the body, or sex) or of increasing BMI by one point. We also plotted 95% confidence intervals for the predicted means and standard deviations of muscle activation at various combinations of values on the predictors.

We used R 2.15.0 (R Foundation for Statistical Computing, Vienna, Austria) [29] for exploratory analyses and the lattice (Deepayan Sarkar, Indian Statistical Institute, New Delhi, India) and Hmisc (Frank E. Harrell, Jr., Vanderbilt University, Nashville, TN, USA) R packages [30,31] for most of our graphing, but used SAS 9.3 (SAS Institute, Inc., Cary, NC, USA) [32] PROC NLMIXED to estimate the final model and a SAS macro (Christopher J. Swearingen, University of Arkansas for Medical Sciences, Little Rock, AR, USA) [33] to obtain a residual plot, because the only available R package for beta regression does not yet support including random effects [34]. We used maximum likelihood estimation with dual quasi-Newton optimization and adaptive Gaussian quadrature integration (over 20 quadrature points). The model converged after 23 iterations. The SAS program used to run the model and the R code for graphing the results are available from the authors on request.

**Results**

Normalized EMG activity from RCPm muscles, collected with the subject's head in a retracted position, was significantly higher (\( p<.0001 \)) than normalized EMG activity collected with the subject's head in a self-selected neutral position.

**Descriptive analyses**

The boxplots in Fig. 3 illustrate both the skewness and heteroscedasticity of the rescaled muscle activation distributions. The distributions were somewhat more skewed for RHP trials than for NHP trials within each side of the body. In each head position, the distribution for the left side of the body was more skewed than the distribution for the right side because of a few outliers. The heights of the boxplots clearly show that RHP trials had noticeably larger variances than NHP trials on both sides of the body, although the difference is larger on the right side. This is what prompted us to include the dispersion submodel containing an interaction in the mixed beta regression model.

**Mixed beta regression**

The model fit statistics (Table) all reinforce the conclusion that the full model fit the data far better than the null model, likelihood ratio (df=9)=248.9, \( p<.0001 \). The \( \Delta AIC = 230.9 \) vastly exceeds the 10-point threshold usually used for model selection. The pseudo-\( R^2 \) suggests that the
There was also moderate nonindependence in the data, as evidenced by the significant random effect of subjects on the location intercept ($t(16)=5.17, p<.01, \text{MOR}=1.54$). The between-subjects standard deviation ($\sigma_{\text{SD}}=0.45$, on the logit scale, median odds ratio=1.54) suggests that for two randomly selected subjects, the one with the higher propensity for muscle activation will have about 54% higher odds of muscle activation than the other subject.

The location fixed effect parameter estimates shown in the Table indicate that there was a strong main effect for head position, $t(16)=8.63, p<.0001, \text{OR}=4.56$, such that average levels of RCPm muscle activation are substantially higher during RHP trials (M=28% on right) than during NHP trials (M=8% on left, 11% on right). There was also a weaker main effect for side of the body ($t(16)=2.65, p<.001, \text{OR}=1.43$), indicating that average levels of RCPm muscle activation are higher on the right than on the left (11% vs. 8% in NHP, 37% vs. 28% in RHP). The nonsignificant position$\times$side interaction effect, $t(16)=0.16, ns, \text{OR}=1.03$, shows that the size of the head position effect on mean levels of muscle activation does not strongly depend on the side of the body observed. Neither sex nor BMI had a significant effect on mean levels of muscle activation ($t(16)=0.11, ns,$

| Parameter estimates, CIs, and ORs for the mixed effects beta regression model |
|---------------------------------|-----------------|-------------------|------------------|---------------|
| Parameter                        | Estimate        | SE (95% CI)       | t (df=16)        | OR (95% CI)   |
| Location fixed effects           |                 |                   |                  |               |
| $\beta_0$: Intercept              | $-2.46$         | $0.23 (-2.94, -1.98)$ | $-10.79^*$       | $0.09 (0.05, 0.14)$ |
| $\beta_1$: HP                     | $-2.52$         | $0.18 (1.15, 1.89)$ | $8.63^*$         | $4.56 (3.14, 6.63)$ |
| $\beta_2$: S                      | $-2.03$         | $0.14 (0.64, 0.7)$  | $2.65^*$         | $1.43 (0.71, 1.91)$ |
| $\beta_3$: Sex (male)             | $-0.03$         | $0.24 (0.49, 0.54)$ | $0.11$           | $1.03 (0.61, 1.72)$ |
| $\beta_4$: Centered BMI           | $-0.01$         | $0.03 (0.07, 0.05)$ | $-0.42$          | $0.99 (0.93, 1.05)$ |
| $\beta_5$: HP$\times$S interaction | $0.03$         | $0.21 (0.42, 0.49)$ | $0.16$           | $1.03 (0.66, 1.63)$ |
| Location random effect            |                 |                   |                  |               |
| $\sigma_{\text{SD}}$: Intercept (SD) | $0.45$         | $0.09 (0.27, 0.63)$ | $5.17^*$         | $1.57 (1.3, 1.89)^*$ |
| Dispersion fixed effects          |                 |                   |                  |               |
| $\gamma_0$: Intercept             | $2.60$          | $0.21 (2.15, 3.06)$ | $12.18^*$        | $0.12 (0.1, 1.2)$ |
| $\gamma_1$: HP                    | $-0.96$         | $0.27 (-1.55, -0.38)$ | $-3.51^*$        | $0.2 (0.1, 0.65)$ |
| $\gamma_2$: S                     | $1.38$          | $0.36 (0.63, 2.13)$ | $3.88^*$         | $1.2 (0.9, 1.6)$ |
| $\gamma_3$: HP$\times$S interaction | $-1.57$       | $0.39 (-2.4, -0.74)$ | $-4.01^*$        | $0.2 (0.1, 0.7)$ |

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SE, standard error; CI, confidence interval; $t$, t-statistic; df, degrees of freedom; OR, odds ratio; HP, head position; S, side of the body; BMI, body mass index; SD, standard deviation; Null, null model; Full, full model; LR, likelihood ratio; AIC, akaike information criterion; PRE, proportional reduction in error.

Note that the parameter for the random intercept was an SD rather than a variance (this parameterization guarantees a positive value for the corresponding variance). N, 257 observations; J, 17 subjects.

$^*$ $p<.0001$.

$^1$ $p<.05$.

$^2$ For the random effect, we report the median OR and the corresponding CI.

$^5$ $p<.01$.

$^11$ $p<.001$. 

Fig. 3. Boxplots of rescaled muscle activation data as a function of side of the body and head position. Filled circles are medians and empty circles are outliers. Asymmetrical whiskers around boxes illustrate skewed distributions; the differing heights of the boxplots demonstrate that the variance is not constant across the combinations of head position and side of the body (i.e., there is noticeable heteroscedasticity). NHP, neutral head position; RHP, retracted head position; RCPm, rectus capitis posterior minor.
predictor and sex is a (nonsignificant) predictor in the location submodel. On the left side, the standard deviation for RHP trials (SD=18%) is 2.6 times larger than the one for NHP trials (SD=7%). However, the difference is quite a bit larger on the right side, where the standard deviation for RHP trials (SD=21%) is 5.0 times larger than the one for NHP trials (SD=4%).

Finally, the boxplots in Fig. 6 show the overall distributions of the predicted values after accounting for all the fixed and random effects in the model. The predicted values from a good model should reproduce the key distributional characteristics of the raw data. Comparing Figs. 3 and 6 reveals that the predicted values in Fig. 6 clearly capture the basic pattern of the medians quite well. They also reproduce some, but not quite all, of the heteroscedasticity apparent in Fig. 3. The distributions of the predicted values are compressed to narrower ranges than the corresponding raw data, but they do capture the skewed nature of the distributions. The pseudo-$R^2$ and PRE statistics indicate that there is room for improvement in overall model fit, but on the whole the results appear acceptable, although still imperfect.

**Discussion**

The aim of this project was to test the null hypothesis that there would be no difference in normalized activation levels of RCPm muscles with the head in a neutral position and with the head in a retracted position.

We found that RCPm muscles are active when the head is held in a self-defined NHP. The range of the mean level of activity with the head in an NHP position for both males and females was between 7.9% and 11.2% of MVIC. An activity level of 7.9% of MVIC has been reported to be a level that is consistent with sustained, static contraction of muscle for 60 minutes [26]. It is possible that the slightly higher levels that we recorded were related to subject stress.

![Fig. 4. Predicted means for muscle activation and corresponding 95% confidence intervals as a function of side of the body, head position, and sex. These estimates illustrate the model results for hypothetical subjects who are average with respect to both body mass index and the random effect of between-subject variability. NHP, neutral head position; RHP, retracted head position; RCPm, rectus capitis posterior minor.](image)

![Fig. 5. Predicted within-subject, residual standard deviations for muscle activation and corresponding 95% confidence intervals as a function of side of the body, head position, and sex. These estimates illustrate the model results for hypothetical subjects who are average with respect to both body mass index and the random effect of between-subject variability. NHP, neutral head position; RHP, retracted head position; RCPm, rectus capitis posterior minor.](image)

![Fig. 6. Boxplots of predicted muscle activation as a function of side of the body and head position. Filled circles are medians and empty circles are outliers. Comparison with Fig. 3 reveals that the distributions of the fitted values from the model are acceptable but imperfect approximations of the skewed, heteroscedastic distributions observed in the raw data. NHP, neutral head position; RHP, retracted head position; RCPm, rectus capitis posterior minor.](image)
due to the invasive nature of the experiments. Biomedical models have shown that short, deep muscles are necessary to maintain spinal stability [35]. In the cervical spine, they function to stabilize the head and neck, maintaining posture and guarding against movements caused by undesirable external forces [6]. We propose that RCPm muscles may function to orient/stabilize the superior articular facets of the atlas relative to the occipital condyles when the head is held in a neutral position.

We found that RCPm muscle activity significantly increased as the head was retracted (p<.0001). The range of the mean level of activity with the head in a retracted position for both males and females was between 28.0% and 37.2% of MVIC. An activity level of 30.0% of MVIC has been reported to be consistent with sustained, static contraction of muscle for approximately 1.0 to 2.5 minutes [26,36]. Voluntary retraction of the head has been reported to increase posterior loading of the superior articular facets of the atlas, resulting in flexion of the OA joint and elongation of the RCPm muscles [11,12]. While the atlas has been characterized as a passive translational element whose orientation is simply driven by head position [37], increased levels of RCPm muscle activation during retraction of the head would be expected to oppose backward rotation of the atlas, an action that would be inconsistent with the definition of the atlas as a simple, passive translational element.

Fatty infiltration of RCPm muscles has been reported in some whiplash patients [38-40]. While the cause of the fatty infiltration in these patients is unknown, it has been reported that the progression of fatty infiltration over time is directly related to pain and disability [41]. Fatty infiltration results in a decrease in muscle mass that is accompanied by a decrease in the ability of a muscle to generate both normal levels of force and proprioceptive information. In the upper cervical spine, these compromise an individual’s ability to precisely coordinate eye/head movements and respond in a protective manner to sudden external forces [42]. It has recently been reported that incongruence between motor output and sensory feedback results in an exacerbation of symptoms in patients with whiplash-associated disorders [43].

A connective link between the RCPm muscles and the cervical dura mater was originally reported by Hack et al. [9]. Subsequent work has confirmed the existence of this connection and has suggested that it may function to prevent or limit enfolding of the pain sensitive dura mater during normal movements of the head [44,45]. Injury-related, fatty infiltration of RCPm muscles would be expected to reduce the ability of these muscles to limit and/or prevent dural enfolding during daily activities and could result in clinical manifestations such as head and neck pain [10].

Retraction of the head has been used in clinical settings to assess and treat patients with neck pain. Head retraction exercises, which incorporate flexion of the craniovertebral junction, have been shown to significantly reduce headache frequency and intensity [46]. While it is known that RCPm muscles are stretched during retraction of the head [11,12], there have been no studies that have quantified the active response of these muscles during retraction. The significant increase in EMG activity reported in the current study suggests that head retraction exercises could result in strengthening of the RCPm muscles.

The study has several weaknesses. One would be the absence of data from rectus capitis anterior muscles. Access to these muscles with wire fine wire electrodes was not considered to be feasible because of either an unacceptable level of risk related to hitting a critical vascular structure or that the protocol would not be tolerated by the subjects. Another weakness was the size of the final data set. We ended up discarding 25% of the data because of dislodged/noisy electrodes or implausible values. Finally, we do not know the actual magnitude of the angle of the OA joint with the head in NHP, nor do we know the actual increase in this angle that occurs when the head is retracted.

Conclusion

We have shown that RCPm muscles are active when the head is held in a self-defined NHP, suggesting that they help orient/stabilize the superior articular facets of the atlas relative to the occipital condyles. Muscle activity was found to significantly increase when the head was voluntarily retracted. This increase in muscle activation would suggest that RCPm muscles are an active element that could influence the magnitude of flexion of the OA joint that occurs during voluntary retraction of the head and may contribute to stabilization of the OA joint during normal daily activities. Future work will continue to investigate the functional significance of RCPm muscles and the clinical relevance of RCPm muscle pathology.

Acknowledgments

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References


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