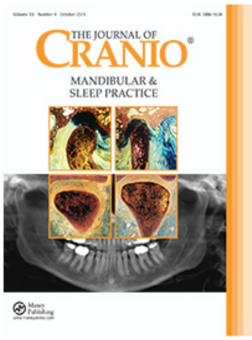

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Simona Tecco, Vincenzo Salini, Stefano Teté, Felice Festa & Christian Colucci

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Simona Tecco, D.D.S.; Vincenzo Salini, M.D.; Stefano Teté, D.D.S.;
Felice Festa, M.D., D.D.S., Ph.D.

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Address for correspondence:
Dr. Simona Tecco
Via Le Mainarde, 26
65121, Pescara
Italy
E-mail: simtecco@tin.it

ABSTRACT: This study evaluated the that effects a pathology of the knee, due to an anterior cruciate ligament (ACL) injury, has on muscular activity of neck, head, and trunk muscles. Twenty-five (25) subjects (mean age 28±9 years) with ACL injury of the left knee were compared with a control no-pathology group. Surface electromyography (sEMG) at mandibular rest position and maximal voluntary clenching (MVC) wasused to evaluate muscular activity of the areas: masseter, anterior temporalis, posterior cervicals, sternocleidomastoid (SCM), and upper and lower trapezius. The sEMG activity of each muscle, as well as the asymmetry index between the right and the left sides, were compared between the two groups. Subjects in the study group showed a significant increase in the asymmetry index of the sEMG activity of the anterior temporalis at mandibular rest position ($p<0.05$). At rest, the areas of anterior temporalis and masseter in the control group showed a significantly lower sEMG activity compared with subjects in the study group, both in the right and the left sides ($p<0.05$). The same was found for the sEMG activity of the areas of SCM and lower trapezius. At MVC, the right areas of anterior temporalis and masseter in the study subjects showed a significantly lower sEMG activity compared with the control group. The same was observed for the lower trapezius area, both in the right and the left sides. In general, ACL injury appears to provide a change in the sEMG activity of head, neck and trunk muscles.

Dr. Simona Tecco *received her D.D.S. degree in 1999 from the faculty of Dentistry, University of Chieti, Italy. Since 1999, she has been a staff member of the Department of Orthodontics and Gnathology, School of Dentistry at the University of Chieti. She is currently working toward a Ph.D. in oral science at the same university.*

The existence of connections between the masticatory system and body posture has been discussed previously.[1] To date, the dental literature includes several articles about the influence of head and body posture on mandibular rest position,[2-3] the range of functional mandibular movements,[4-5] and facial morphology.[6-7] Mintz[8] also observed that temporomandibular joint (TMJ) syndromes can develop based on an orthopedic postural problem. He found that a severe back injury or chronic lower back problems may alter the position of the cranium and result in a mandibular malposture, which can affect the muscles of mastication and cause spasm or chronic irritation. By this mechanism, a TMJ problem could also be caused by repetitive maintenance of an unorthodox position in a job or exercise that may tend to over-stimulate some of the muscles in the previously described chain reaction. Other authors[5,9] suggest that body postural control does not influence mandibular rest position in young adults without occlusal problems, since the range of body postural changes that can be compensated is generally greater in subjects without occlusal problems, than in patients with occlusal problems. Finally, evidence has been presented for a functional and anatomic correlation between the stomatognathic system and the postural regulation system in cats[10] and in rats.[11]

Somatosensory information from the lower limbs was found to strongly contribute to postural control of the body.[12] Anterior cruciate ligament (ACL) injury has repeatedly been reported to compromise knee proprioception, presumably due to destruction of mechanoreceptors within the ligament.[13-18] This has lead researchers to also study the effects of ACL injury on measures of standing balance.[19-23]

Regarding a neurologic connection between the lower extremities (mainly the knee) and the CNS, it is noted that neuromotor control of the knee involves the coordinated activity of surrounding muscles, in particular, the quadriceps. This coordinated activity provides active stability to the knee joint during weight-bearing activities.[24-25] The reverse also being true that knee joint pro-prioception seems to be essential to neuromotor control.

Additionally, proprioceptive afferent information from mechanoreceptors in the muscles, ligaments, capsule, menisci, and skin contributes at the spinal level to influence muscular reflexes which play a large part in dynamic joint stability.[15,26-27] This information seems also to convey to supraspinal centers,[28] where it is integral to motor learning and the ongoing programming of complex movements.

Consequently, it was hypothesized that proprioceptive impairment could be influenced by pain and disability associated with the disease and, in view of the influence of supraspinal centers, some authors have also demonstrated a link between knee disease, proprioceptive impairment, and either physical function[29] or pain[30] in individuals with musculoskeletal knee complaints. Others have failed to do so.[31-32]

Simplistically, chemical substances produced during the pain response may sensitize the nerve endings resulting in abnormal discharge of pain afferents. Via influences of gamma-motorneurons, muscle spindle afferent activity may be altered, thus interfering with proprioceptive input.[33-34] However, evidence that an ACL injury could influence the sEMG activity of head, neck and trunk muscles and in which way is still lacking. The aim of this study was to investigate the surface electromyographic (sEMG) activity of masticatory, neck, and trunk muscles in adult patients who underwent ACL injury. This could be of great interest for researchers, as it could contribute to clarification of the nature of the relationship between body posture and the stomatognathic apparatus.

Materials and Methods

Twenty-seven (27) subjects, Caucasian adult males having undergone ACL injury at the left knee at least six months before testing, and an equal number of voluntary control subjects without any pathology of the knee, were assessed on one occasion of approximately 90 minute duration.

The study subjects were selected from an initial group of 33 patients with ACL injury from the Orthopedic Unit of the University G. D'Annunzio, Chieti, based on the following inclusion criteria, which were also used to select the voluntary control subjects: (i) absence of any previous orthodontic or gnathologic treatment; (ii) presence of a natural dentition and a bilateral molar support with molar and cuspid Angle class I; (iii) absence of any cross-bite; (iv) presence of chronic muscle pain according to the history of signs and symptoms or elicited during palpation of the muscles of the trunk, neck, and stomatognathic area; (v) absence of referred pain due to myofascial trigger points; (vi) absence of whiplash, neurologic, vestibular, oculomotor disorders and/or hip dysymmetry; and (vii) absence of any particular episode of psychosocial and psychological stress profile in the last six months, except for the injury to the knee for the study subjects. Moreover, any patient positive for the diagnosis of pure articular pathology and/or symptoms caused by trauma or surgery was not included in the study or in the control population. Approval was obtained from the University's Review Board for Health Sciences Research involving Human Subjects, and all subjects provided written informed consent before testing.

Subjects underwent sEMG recordings of neck, trunk, and masticatory muscles. Subjects wore gym shorts and shoes for all tests. The study was performed using a Key-Win 2.0 surface electromyograph (Biotronic s.r.l., S. Benedetto Tronto, Ascoli Piceno, Italy) with disposable electrodes (DUO F3010 bipolar - 10 mm, Ag-AgCl, lithium chloride gel, unit distance 22 mm, LTT FIAB Vicchio, Firenze, Italy). The Key-Win 2.0 is an sixty channel electromyograph with a 15-430 Hz band-pass filter, containing a special 60 Hz notch filter to eliminate any of the electrical noise from the recording environment that exceeds the capabilities of the common mode rejection scheme. All monitoring was performed with the patients in a standing position. The subjects were asked to make themselves comfortable, to relax with their arms by their sides, and to look straight ahead and make no head or body movements during the test. The electrodes, which determine to a large extent the quality of the recordings, were placed according to the electrode atlas of Cram and Kasman.[35] Before the electrodes were applied, the skin was thoroughly cleaned with alcohol. The sEMG activity of six muscles was studied bilaterally (i) with the mandible at the rest position, and (ii) during maximal voluntary clenching (MVC). Subjects were instructed to close their jaws in centric occlusion as forcefully as possible. Movement patterns were conducted for at least three repetitions to ascertain stability according to the protocol developed by Donaldson and Donaldson.[36]The first movement patterns were eliminated as the "learning" sequence, as they were frequently observed as dissimilar to the other two repetitions. The third movement was considered the most stable.

The muscular areas tested were: right masseter area (RMM), left masseter area (LMM), right anterior temporal area (RTA) and left anterior temporal area (LTA) as masticatory muscles; right sternocleidomastoid area (RSCM), left sternocleidomastoid area (LSCM), right posterior cervicals area (RPC) and left posterior cervicals area (LPC) as neck muscles; right upper trapezius area (RUTR), left upper trapezius area (LUTR), right lower trapezius area (RLTR) and left lower trapezius area (LL TR) as trunk muscles. The sEMG recording time for each analysis was at least 15 seconds, and the values were expressed in microvolts per second (μV olts/sec).[37]

For the sEMG activity data, with each muscle, the sEMG activity was expressed as mean (standard deviation). Moreover, the corresponding asymmetry index was calculated by using the formula[35]:

$$\frac{\text{sEMG (the higher sEMG between the right and left side) - sEMG (the lower sEMG between the right and left side)}}{\text{sEMG (the higher sEMG between the right and left side)}}$$

For each condition (mandibular rest position and MVC) and each muscle, due to the small size of the sample, a nonparametric test (Mann-Whitney test) was employed to test the significance levels of difference in the asymmetry index between the study and the control groups. To further highlight the differences between the two groups in the sEMG activity of each muscle, a Mann Whitney test was employed to test the significance levels of differences in sEMG activity between the left and right sides and, for each side, between the study and the control groups. All statistical analyses were performed using the Statistical Package for Social Sciences program (SPSS, Inc., Chicago, IL). The 0.05 level was used to denote statistical significance throughout testing.

Results

The clinical results obtained in the present study are summarized in **Tables 1**, **2** and **3**. At rest, the two areas of the masticatory muscles (anterior temporal and masseter) in the control group showed a significantly lower sEMG activity compared with subjects in the study group, both in the right and left sides (**Table 1**). In addition, subjects in the control group showed a significantly lower sEMG activity of the SCM and lower trapezius areas compared with subjects in the study group, both in the right and left sides (**Table 1**). In the MVC, the right areas of the anterior temporal and masseter in the study subjects showed a significantly lower sEMG activity compared with the corresponding areas in the control group (**Table 2**).

There was a significant difference in the asymmety index between the two groups, regarding the anterior temporal area (**Table 3**). With regard to the muscular area of the trunk, subjects in the study group showed a significantly higher sEMG activity of the lower trapezius compared with the control subjects, both in the right and left sides (**Table 2**).

Discussion

In this study, we selected only Caucasian adult males having undergone ACL injury of the left knee at least six months before testing. This inclusion criteria was introduced to exclude subjects with chronic and acute/subacute ACL injuries. It is noted, that the type of ACL injury could influence the knee proprioception, as this seems to be reduced in subjects with chronic injury. By using this inclusion criteria, we tried to obtain an homogeneous group for this characteristic. However, in future studies, it could be useful to investigate two different study groups, including those affected by chronic or acute/subacute ACL diseases.

The current study was designed to evaluate the sEMG activity of the masticatory, neck and trunk muscular areas in subjects who have undergone ACL injury in the knee. Moreover, to objectively monitor the muscles' status, we made use of sEMG recordings. Surface EMG is based on the extra-cellular recording of the motor unit action potentials by means of surface sensors, and the magnitide of energy recorded is in the millivolt range.[35] Reflecting the muscle activity, the sEMG signal is lower at rest and greater under isometric contraction, i.e., maximal voluntary clenching (MVC); therefore, when a muscle is affected by some dysfunction, the signal at rest generally tends to increase, and the sEMG activity tends to decrease during MVC. In the current study, the muscular areas were monitored at both the mandibular rest position and MVC. In order to avoid any occlusal interference on the MVC, the recordings of the MVC were made by using cotton rolls securely tied between the dental arches.

The sEMG activity of the anterior temporal and masseter areas at rest was significantly lower in the control group compared with the study group, both in the right

Table 1
sEMG Activities (μV/s) of the Different Muscles Monitored at Mandibular Rest Position (n=54)

	N	Percentiles		
		25th	50th (Median)	75th
Anterio temporal (left) control group	27	2.15	4.12 § (**)	7.44
Anterior temporal (right) control group	27	3.05	3.78 § (***)	6.12
Anterior temporal (left) study group	27	4.97	7.03	7.62
Anterior temporal (right) study group	27	6.02	7.26	9.26
Masseter (left) control group	27	1.87	2.71 § (***)	3.76
Masseter (right) control group	27	1.67	2.46 § (***)	3.80
Masseter (left) study group	27	3.49	4.89	8.30
Masseter (right) study group	27	3.57	5.20	9.17
SCM (left) control group	27	2.21	2.98 § (***)	4.18
SCM (right) control group	27	2.71	3.46 § (***)	4.19
SCM (left) study group	27	4.26	5.19	6.97
SCM (right) study group	27	4.15	5.75	7.79
Upper trapezius (left) control group	27	3.51	5.93	34.76
Upper trapezius (right) control group	27	5.05	13.22	24.88
Upper trapezius (left) study group	27	5.29	8.30	10.
Upper trapezius (right) study group	27	5.08	7.70	13.15
Lower trapezius (left) control group	27	3.49	4.85 § (**)	8.42
Lower trapezius (right) control group	27	4.36	5.44 § (**)	7.54
Lower trapezius (left) study group	27	7.37	8.97	12.69
Lower trapezius (right) study group	27	5.94	9.30	13.71
Cervicals (left) control group	27	3.71	5.67	8.14
Cervicals (right) control group	27	4.	5.68	9.69
Cervicals (left) study group	27	3.91	7.38	9.55
Cervicals (right) study group	27	4.94	7.80	11.76

† *Significantly different from the corresponding controlateral muscle*
§ Significantly different from the control group
SD Standard Deviation
* P<0.05; ** P<0.01; *** P<0.001

Table 2
sEMG Activities (µV/s) of the Different Muscles Monitored in Maximal Voluntary Clenching (n=54)

	N	Percentiles		
		25th	50th (Median)	75th
Anterior temporal (left) control group	27	77.20	106.54	141.79
Anterior temporal (right) control group	27	78.43	107.08 § (*)	142.35
Anterior temporal (left) study group	27	48.72	91.62	114.01
Anterior temporal (right) study group	27	55.33	84.03	110.79
Masseter (left) control group	27	72.46	106.54	206.53
Masseter (right) control group	27	90.25	151.59 § (*)	201.49
Masseter (left) study group	27	38.89	75.12	109.71
Masseter (right) study group	27	48.09	70.92	121.32
SCM (left) control group	27	4.56	6.57	13.13
SCM (right) control group	27	5.39	6.97	11.11
SCM (left) study group	27	6.40	8.	9.58
SCM (right) study group	27	6.67	8.24	9.51
Upper trapezius (left) control group	27	5.43	17.22	33.56
Upper trapezius (right) control group	27	5.18	21.59	32.63
Upper trapezius (left) study group	27	9.24	12.60	18.
Upper trapezius (right) study group	27	10.	15.	19.36
Lower trapezius (left) control group	27	2.54	4.41 § (***)	6.29
Lower trapezius (right) control group	27	4.18	4.61 § (*)	6.85
Lower trapezius (left) study group	27	10.16	13.12 † (**)	19.50
Lower trapezius (right) study group	27	3.39	9.64	12.
Cervicals (left) control group	27	5.32	6.47	8.90
Cervicals (right) control group	27	4.64	6.88	8.90
Cervicals (left) study group	27	3.90	7.10	9.0
Cervicals (right) study group	27	4.96	6.38	10.0

† *Significantly different from the corresponding controlateral muscle*
§ Significantly different from the control group
SD Standard Deviation
* P<0.05; ** P<0.01; *** P<0.001

Table 3
Asymmetry Index for the Different Muscles Monitored

	N	Mean	Std. Deviation	Range	Minimum	Maximum
MANDIBULAR REST POSITION						
Anterior temporal, study group	27	.45 §	.31	.95	.05	1.
Anterior temporal, control group	27	.29	.19	.67	.03	.70
Masseter, study group	27	.34	.19	.81	.05	.86
Masseter, control group	27	.44	.28	.88	.03	.91
SCM, study group	27	.27	.19	.57	.03	.60
SCM, control group	27	.28	.24	.91	0.0	.91
Upper trapezius, study group	27	.40	.22	.86	.11	.97
Upper Trapezius, control group	27	.47	.28	1.09	-.17	.92
Lower Trapezius, study group	27	.33	.28	.98	0.0	.98
Lower trapezius, control group	27	.35	.28	.84	.02	.85
Cervicals, study group	27	.34	.18	.64	0.0	.64
Cervicals, control group	27	.32	.18	.66	.02	.68
MVC						
Anterior temporal, study group	27	.68	.07	.75	.31	.19
Anterior temporal, control group	27	.99	.	.99	.46	.35
Masseter, study group	27	.82	-.19	.63	.28	.19
Masseter, control group	27	.71	.02	.73	.35	.17
SCM, study group	27	66.98	.02	.67	2.82	12.83
SCM, control group	27	.65	.04	.69	.28	.16
Upper trapezius, study group	27	.66	.	.66	.24	.19
Upper Trapezius, control group	27	.96	.03	.98	.32	.23
Lower Trapezius, study group	27	.89	-.04	.85	.22	.20
Lower trapezius, control group	27	.75	.17	.92	.45	.19
Cervicals, study group	27	.82	.01	.83	.39	.23
Cervicals, control group	27	.83	.03	.86	.32	.21

§ Significantly different from the control group
SD Standard Deviation
* P<0.05; ** P<0.01; *** P<0.001

and left sides (**Table 1**). In the MVC, the same muscular areas showed a significantly higher sEMG activity in the control group compared with the study group, but exclusively in the right side for both considered areas (**Table 1**). This finding seems to suggest that the effects on the muscular tensions produced by the postural pathology of the knee can also extend to some of the masticatory muscles. Since the study subjects showed an increase in the sEMG activity of the masticatory muscles at rest and a decrease of their activity during MVC, we may speculate on a clinically relevant wearing of the masticatory muscles, following a modification in body posture caused by the ACL injury of the knee, which suggests an interdependence between total body posture and occlusion.

Furthermore, the mandibular muscle activities may be influenced by tonic neck and trunk muscles, as previously reported by Darnell.[38] A possible clinical correlation between the stomatognathic system and the rest of the body is based upon the body muscle chains of the neuromuscular system. When a muscle is in spasm or has a myofascial trigger point, it might lead to a modification in postural position.[39] With regard to the sEMG activity of the neck and trunk muscles, the study subjects showed a significantly higher sEMG activity of the SCM and lower trapezius areas at rest in both the right and left sides (**Table 1**) and a significantly lower sEMG activity of the lower trapezius at MVC (**Table 2**), suggesting an interdependence between total body posture and the activity of neck and trunk muscles.

It should also be noted that the sEMG activity can be affected by several factors, i.e., psychological stress. A negative stress profile may have a specific influence on muscle activity. Ruf, et al.[40] found that the sEMG activity of the masticatory muscles during stress situations was significantly higher when compared to that of non-stress situations, and that females have significantly higher ratings than males. In the current study, even though the patients themselves said there was no particularly significant positive or negative psychophysiological stress in their lives, this was deemed unreliable.

Finally, it is of interest to note that in the MVC, the significant discrepancy between the study and the control subjects was observed exclusively on the right side for both the masseter and the anterior trapezius areas, while the differences observed at rest included both the right and the left sides. It is difficult to explain this observation, since it seems to suggest a correlation between the side of the ACL injury (the left knee in all subjects in the study group) and the isometric contraction of the masticatory muscles, suggesting a role of counter-balance of the muscular chains of the other side of the body (with respect to the side where the postural problem was introduced) and their consequent weakness.

The foregoing information seems also to agree with a statement from the clinical orthopedic information, that the lower extremity joints are intimately related and function as a unit. Therefore, intuitively, changes in loading and structure occurring at one joint or subunit of the lower extremity could result in neuromuscular alterations at the other lower extremity joints.

Using mechanical power methods, McGibbon[41] characterized mechanical alterations present in 13 subjects with knee osteoarthritis and focused on the hip and ankle joints in addition to the affected knee. Subjects had reduced ankle power, increased power absorption at the hip, and an absent positive peak in knee power, so it was concluded that the alterations in loading may affect the pattern of evolution of lower extremity osteoarthritis.

A recent study also indicated that osteoarthritis showed a nonrandom pattern in the evolution of lower extremity osteoarthritis. Subjects with end stage unilateral knee osteoarthritis were found to develop osteoarthritis in the hip contralateral to the side of the affected knee significantly more often than they did at the ipsilateral hip.[42]

Similarly, subjects with end stage unilateral hip osteoarthritis were found to develop knee osteoarthritis at the knee contralateral to the side of the affected hip significantly more often than they did in the ipsilateral knee.[42]

Interestingly, the asymmetry in loading of the hips in these subjects with early osteoarthritis diminished with analgesic relief of their knee pain. Thus, this asymmetry in dynamic joint loading, favoring the contralateral side, seems to reflect an interrelationship between the lower extremities and the body posture.

No conclusion was possible regarding the “mechanism at work,” since the cross-sectional construction of the current study avoided the finding of statistically significant associations. The results obtained in this study warrant further longitudinal investigation in order to clarify the mechanism of the observed significant associations. The current cross-sectional study only demonstrated that a postural problem due to an ACL injury in the knee should be associated with a significant change in the sEMG activity of the areas which correspond to the masticatory muscles (anterior, temporal, and masseter) and neck and trunk muscles (SCM and lower trapezius).

The data could be used in research to better understand the complex body posture system and its relationship to the force of gravity, but they cannot explain the significant associations observed.

In the future, in view of a complete understanding of the relationship between ACL injury and muscle activity, it would be interesting to evaluate sEMG activity of the upper and lower leg muscles affected by an ACL injury. In this investigation, this was not considered, since the primary interest of the researchers was to evaluate the head and neck areas.

The data could also contribute to a clarification of the complex nature of the relationship between head, neck, and trunk muscles and ACL injury, primarily concerning postural or neural linkages. An investigation to evaluate the sEMG activity of the legs, head, neck, and trunk muscles, after a local anesthetic block of the ACL could also be very useful. In addition, sEMG activity of the head, neck, and trunk muscles could be evaluated after knee brace stabilization and ACL surgical repair.

From a clinical standpoint, the current study seems to suggest that it is important to determine the clinical outcome resulting from these findings, in terms of understanding whether some stomatognathic muscular symptoms occur in patients who undergo ACL injury and subsequent ACL surgical repair, and what type of influence the ACL surgical repair could have on stomatognathic symptoms. The study of muscular stomatognathic symptoms will be necessary for completion of the study in this research area.

The current study's results can be added to several other observations, which indicate that a specialist in a single discipline (for example, orthopedics or gnathology) cannot always resolve or understand a patient's problem and underscores the importance of a multidisciplinary approach. Dentists, physicians, orthopedists, physiotherapists, osteopaths, etc., each contribute their own specific knowledge in order to reach a differential diagnosis that allows correct treatment planning. Clinical success (in order to understand the origin of the problem), therefore, depends on the ability of each specialist to analyze the different aspects of the same problem. A teamwork structure may be the best option to obtain a good functional state of the stomatognathic system and the rest of the body after therapy.

Conclusions

The results of the current study indicate that an ACL injury can be associated with a change in the sEMG activity of the areas corresponding to the masticatory muscles (anterior temporal and masseter) and to the neck and trunk muscles (SCM and lower trapezius). However, further investigations are required to better understand the subsequent changes in the sEMG activity of these muscles, after a surgical procedure and implementation of a rehabilitative program to bring about a complete knee recovery.

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Dr. Vincenzo Salini *received his M.D. degree in 1991 from the Faculty of Medicine, University of Chieti, Italy and degrees in orthopedics and sports medicine in 1996 and 2000, respectively. Dr. Salini is an orthopedic doctor in the orthopedic division at the Hospital of Chieti, Italy.*

Dr. Christian Colucci *is completing a fellowship in orthopedics at the University of G. d'Annunzio, Chieti-Pescara, Italy.*

Dr. Felice Festa Dr. Felice Festa *is director of the Department of Orthodontics and Gnathology, School of Dentistry and a professor of orthodontics at the University of Chieti, Italy. He is also director of the postgraduate program in clinical gnathology and orthodontics. He received his M.D. and D.D.S. degrees at the University of Rome in 1979 and 1982 respectively. He earned an M.S. degree in orthodontics in 1985 at the University of Cagliari. In 2001 he was nominated National Referee Professor in orthodontics at the National College of Professors.*
Dr. Festa has many published research articles in his field.