The Relationship between Muscle Weakness and Activation of the Cerebral Cortex Early after Unicompartmental Knee Arthroplasty

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Abstract. [Purpose] This study investigated the relationship between muscle weakness and activation of the cerebral cortex in the early period after unicompartmental knee arthroplasty (UKA). [Subjects] The subjects were 11 patients who underwent UKA. [Methods] They performed maximum isometric knee extension using a muscle force measurement device before, and 1 and 2 weeks after UKA. They were simultaneously measured for changes in the concentrations of oxyhemoglobin, deoxyhemoglobin, and total hemoglobin in the bilateral sensorimotor leg areas using functional multi-channel near-infrared spectroscopy. [Results] The muscle force decreased after UKA, and the active region of the sensorimotor leg area also narrowed. The severity of knee pain with muscle contraction 2 weeks after UKA did not change significantly compared with the preoperative level. [Conclusions] The patients treated with UKA displayed early postoperative muscle weakness, and the bilateral sensorimotor areas that were activated during maximum isometric knee extension were narrowed, suggesting that early postoperative muscle weakness is influenced by the central nervous system.

Key words: Unicompartmental knee arthroplasty, Muscle weakness, Cerebral cortex

(INTRODUCTION

Unicompartmental knee arthroplasty (UKA) is indicated for osteoarthritis of the knee (knee OA) and femoral condylar necrosis localized in the medial or lateral condyle. As only the medial (lateral) joint is replaced with a prosthesis in UKA, it is less invasive than total knee arthroplasty (TKA), in which the whole degenerated knee joint is replaced with a prosthesis. UKA results in rapid recovery of the range of motion of the affected joint compared with TKA, and has been suggested to shorten the patient's hospital stay and improve their functional score1). Changes in the strength of the quadriceps femoris muscle on the treated side during maximum isometric knee extension after UKA and TKA have been investigated by several studies, and postoperative muscle weakness was observed2–4). The causes of muscle weakness after TKA include pain5), joint injury5), and arthropgenic muscle inhibition (AMI), in which quadriceps femoris muscle inhibition is induced by exudate and swelling-associated changes in the intra-articular pressure transmitted from the articular receptor via the spinal cord5–9). In the acute postoperative period, patients lose approximately half of their preoperative quadriceps strength in the first month after TKA4,10). It was reported that muscle weakness early after surgery is mainly due to the failure of voluntary muscle activation, not due to pain11). The failure of voluntary muscle activation is due to a reduction in the maximal force output resulting from inability to recruit all of the muscle’s motor units or to attain the maximal discharge rate from the motor units that are recruited12). Some studies have also reported that postoperative muscle weakness is a result of central nervous system influence2,4). Muscle output during voluntary physical movement is controlled by the nervous system. Due to improvements in imaging techniques, studies of human brain function during voluntary movement have recently been performed13–17), and the relationship between muscle output and brain function has also been investigated18–21). However, no studies have investigated the contribution of lesser cerebral activation to muscle weakness after UKA. An investigation of the relationship between muscle weakness and activation of the cerebral cortex during the early period after UKA would be useful for designing more effective rehabilitation programs after knee replacement, because...
only strength training is carried out for muscle hypertrophy at present. Functional near-infrared spectroscopy (fNIRS) measures changes in the hemoglobin (Hb) level and nerve activity-coupled cerebral blood flow, which allows the state of brain activity to be examined. Various brain functions have been measured by fNIRS, and recently there has been explosive growth in optical imaging research focusing on the complex motor behaviors affecting health and disease, including studies of the cortical contribution to everyday tasks such as walking and running, motor skill learning, cortical hemodynamics associated with athletic motor performance, and the cortical compensation associated with recovery from cerebrovascular accidents. Therefore, in this study, we investigated whether the central nervous system is involved in the muscle weakness experienced after surgery by examining the function of the cerebral cortex using fNIRS during voluntary movement of a replacement knee during the early postoperative period.

The purpose of the present study was to examine whether: the change in cerebral cortex activation accounts for the greater loss of quadriceps strength; the worsening of knee pain compared with the preoperative level accounts for a large portion of the worsening of voluntary activation after UKA; and the scalp blood flow influences fNIRS values of the sensorimotor area.

**SUBJECTS AND METHODS**

**Table 1. Patients' characteristics**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Body mass index (kg/m²)</th>
<th>Disease</th>
<th>Operated side</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Male</td>
<td>82</td>
<td>27.7</td>
<td>Femoral medial condylar necrosis</td>
<td>Left</td>
</tr>
<tr>
<td>2</td>
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<td>78</td>
<td>28.3</td>
<td>Knee OA</td>
<td>Left</td>
</tr>
<tr>
<td>3</td>
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<td>69</td>
<td>26.4</td>
<td>Knee OA</td>
<td>Left</td>
</tr>
<tr>
<td>4</td>
<td>Male</td>
<td>70</td>
<td>31.3</td>
<td>Knee OA</td>
<td>Right</td>
</tr>
<tr>
<td>5</td>
<td>Female</td>
<td>67</td>
<td>29.1</td>
<td>Knee OA</td>
<td>Right</td>
</tr>
<tr>
<td>6</td>
<td>Female</td>
<td>73</td>
<td>29.7</td>
<td>Knee OA</td>
<td>Right</td>
</tr>
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<td>7</td>
<td>Female</td>
<td>72</td>
<td>24.4</td>
<td>Knee OA</td>
<td>Right</td>
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<tr>
<td>8</td>
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<td>80</td>
<td>31.4</td>
<td>Knee OA</td>
<td>Right</td>
</tr>
<tr>
<td>9</td>
<td>Male</td>
<td>70</td>
<td>22.5</td>
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<td>Female</td>
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<td>23.8</td>
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<tr>
<td>11</td>
<td>Female</td>
<td>77</td>
<td>24.8</td>
<td>Knee OA</td>
<td>Right</td>
</tr>
</tbody>
</table>

Knee OA: osteoarthritis of the knee

Study 1: The subjects were 11 patients (4 males and 7 females; mean age ± standard deviation [SD]: 74.3 ± 5.1 years [67–82 years]) with a mean body mass index ± SD of 27.2 ± 3.0 kg/m² (22.5–31.4 kg/m²) who underwent UKA at Kagawa University Hospital (Table 1). Ten patients had osteoarthritis of the knee (knee OA) (left side in 3 and right side in 7), and one patient suffered from femoral medial condylar necrosis (left side). Informed consent to participate in the study was received from the subjects after the study’s objective had been explained to them. The patients sat on the edge of a bed with their hip and knee joints at flexion angles of 90° and 70°, respectively, and performed maximum isometric extension of their treated knee, and the muscle force generated during this exercise was measured. A task consisting of resting for 20 seconds and maximum isometric extension of the treated knee for 5 seconds, was consecutively repeated 3 times on the treated side with an additional 20 seconds of rest allowed after the last repetition (total duration: 95 seconds). The subjects also performed another task (rest) comprised of resting in the sitting position without maximum isometric extension for 95 seconds. The muscle force generated during maximum isometric knee extension was measured using a μTas F-1 device (ANIMA Co., Japan). This device contains a thin sensor applied to the measurement site and measures the isometric muscle force generated during an exercise. A belt holds the device in place. The strength of the force generated during isometric knee extension per kg body weight (kgf/kg) is calculated as the mean of 3 measurements. A visual analog scale (VAS) was used to quantify knee pain during testing. Patients showed in the pain level on a scale from 0 (mm) to 100 (mm) using a ruler, with 0 (mm) representing no pain and 100 (mm) representing the worst pain imaginable. The knee pain rating was assigned after 3 maximum isometric extensions. A 3-wavelength (780, 805, and 830 nm) fNIRS system (OMM-3000 NIRStation, Shimadzu Co., Japan) was utilized to measure changes (mM•cm) in the concentrations of oxyhemoglobin, deoxyhemoglobin, and total hemoglobin ([oxyHb], [deoxyHb], and [totalHb], respectively; [totalHb] = [oxyHb]+[deoxyHb]) in the cortex by applying the modified Beer-Lambert law to the acquired data. fNIRS data were collected at 10-Hz sampling intervals.

A probe holder that was specially designed for the probe arrangement employed in this study was attached to the subject’s scalp, and 12 probes (6 light-emitting and 6 light-receiving fibers) were positioned according to the 1–17 measurement channel arrangement. The probes were arranged at an inter-optode distance of 3 cm around the sensorimotor leg area (95 × 60 mm) with the Cz position as specified in the international 10–20 system used as the baseline (Fig. 1). We principally used changes in [oxyHb] as a marker of regional activation, since previous findings have shown that oxyHb is more sensitive to locomotion-related...
During the task, and comparisons of these values were determined for each channel during each of the 3 repetitions and that for the corresponding 2 seconds during the rest were determined for the 2 seconds from 3 to 5 seconds after exercise initiation. For all 11 subjects, the mean \([\text{oxyHb}]\) values from the measurement data to correct for the baseline data in the 15-second resting period before motion was subtracted. As in study 1, using the fNIRS system, the 12 probes were attached to the subject’s scalp (Fig. 1). In addition, we principally used changes in \([\text{oxyHb}]\) as a marker of regional activation.

The fNIRS data obtained at channels 5 and 12 were regarded as those of the contralateral sensorimotor leg area, and the data obtained at channels 6 and 13 as those of the ipsilateral sensorimotor leg area. For each of the 7 subjects, the changes in \([\text{oxyHb}]\) measured during the 3 repetitions of the task were averaged for each channel. A linear equation with a slope of 0 passing the mean at a time constant of 0.1 seconds, and the values measured in the 3 sets of the task were averaged. A linear equation with a slope of 0 passing the mean of 150 data points in the 15-second resting period before the task motion was subtracted from the measurement data to correct for the baseline. As in study 1, using the fNIRS system, the 12 probes were attached to the subject’s scalp (Fig. 1). In addition, we principally used changes in \([\text{oxyHb}]\) as a marker of regional activation.

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[\text{oxyHb}] in each channel and the skin tissue blood flow after task initiation were analyzed using the paired t-test.

**RESULTS**

Study 1: Figure 2 shows topographical image patterns of averages of the time courses of [\text{oxyHb}], [\text{deoxyHb}], and [\text{totalHb}] in the bilateral temporo-parietal region during maximum isometric extension at 1 week after UKA in patient 4. It shows that increments in [\text{oxyHb}] and [\text{totalHb}] occurred mainly in the bilateral posterior sensorimotor areas during the task. Figure 3 shows averages of changes in [\text{oxyHb}], [\text{deoxyHb}], and [\text{totalHb}] in the anterior and posterior contra- and ipsilateral sensorimotor leg areas during maximum isometric knee extension before, and 1 and 2 weeks after UKA in the same subject. Before UKA, [\text{oxyHb}] increased and [\text{deoxyHb}] decreased during the task in all areas. The [\text{totalHb}] increased during the task in the posterior contra- and ipsilateral sensorimotor leg areas, and it decreased during the task in the anterior contra- and ipsilateral sensorimotor leg areas. However, [\text{oxyHb}] and [\text{totalHb}] increased during the task in only the bilateral posterior sensorimotor areas, and [\text{oxyHb}] and [\text{totalHb}] decreased in the bilateral anterior sensorimotor areas at 1 and 2 weeks after UKA.

A summary of [\text{oxyHb}] detected during maximum isometric extension in the anterior and posterior contra- and ipsilateral sensorimotor leg areas before, 1 week after, and 2 weeks after UKA is shown in Table 2. Before UKA, in all of the channels corresponding to the anterior and posterior contralateral and ipsilateral sensorimotor areas, [\text{oxyHb}] increased with time from 0 seconds (motion initiation). Compared to those at rest, the mean [\text{oxyHb}] for the 2 seconds of maximum isometric extension increased in all of the channels corresponding to the anterior and posterior contralateral and ipsilateral sensorimotor areas. At 1 week after UKA, [\text{oxyHb}] increased in the posterior contralateral and ipsilateral sensorimotor areas, but not in the anterior contralateral or ipsilateral sensorimotor area. In addition, the activated regions of the bilateral sensorimotor areas had narrowed compared to before UKA. At 2 weeks after UKA, the changes were similar to those observed at 1 week after UKA.

The muscle force (mean ± SD) generated during maximum isometric knee extension was 0.32 ± 0.16 (kgf/kg) before UKA and 0.16 ± 0.08 and 0.20 ± 0.07 (kgf/kg) at 1 and 2 weeks after UKA, i.e., the muscle force was reduced at 1 week (p = 0.004) and 2 weeks (p = 0.032) after, compared with before UKA. The knee pain (VAS) during maximum isometric knee extension was 9.3 ± 14.1 (mm) before UKA and 36.4 ± 24.3 (mm) and 24.2 ± 22.2 (mm) at 1 and 2 weeks after UKA, i.e., knee pain had increased at 1 week (p = 0.012) after, compared with before UKA; however, there was no significant difference between before and 2 weeks after UKA.

Study 2: Each of the 7 subjects showed patterns of mean changes in skin tissue blood flow in the scalp and [\text{oxyHb}] at channel 5 during maximum isometric knee extension (Fig. 4). The skin tissue blood flow and [\text{oxyHb}] simultaneously increased after task initiation; however, [\text{oxyHb}] reached a peak value during the task, and the skin blood flow reached its peak value at the end of the task. The time required after the initiation of maximum isometric knee extension to reach the peak value was compared between [\text{oxyHb}] of each channel and the skin blood flow. The time required to reach the maximum value of skin blood flow was significantly longer than that of [\text{oxyHb}] (Table 3).

**DISCUSSION**

The muscle force generated during maximum isometric knee extension was significantly decreased at 1 and 2 weeks after UKA compared with that before surgery. Weakness of the quadriceps femoris muscle after UKA and TKA has previously been reported\(^{2-4}\), and it was suggested that muscle weakness occurs early after surgery. Stevens et al.\(^{7}\) suggested that, at a mean of 3.5 weeks after TKA, the quadriceps strength had decreased by 60%. More than half of the strength loss was explained by an increase in AMI, and pain during contraction accounted for less than 25% of the increase in AMI from before to after surgery. Perhonen et al.\(^{10}\) detected quadriceps atrophy of less than 5% at 3 weeks after TKA, while other studies found quadriceps atrophy of 16–21% in healthy adults due to disuse or immobilization over a similar period of time. Therefore, muscle atrophy is not considered to cause the muscle weakness experienced after UKA and TKA. Rather, surgery-associated muscle weakness has been suggested to be the cause, due to inhibition of the central nervous system, based on studies of the central activation ratio of the quadriceps femoris muscle following TKA\(^ {5-7}\). However, the changes in cerebral cortical activation have not previously been investigated.

Two weeks after UKA, the postoperative score for pain during isometric knee extension was not significantly different from the preoperative score, whereas muscle weakness had developed. Minzer et al.\(^ {3}\) reported that knee pain with muscle contraction after TKA plays a small role in the reduction of muscle activation, and the muscle weakness is due to the failure of voluntary muscle activation.

In this study, the [\text{oxyHb}] levels in the bilateral sensorimotor areas increased before UKA, but the areas displaying increased [\text{oxyHb}] levels were narrowed at 1 and 2 weeks after UKA. Animal and human studies have suggested that changes in physical afferent information or motor output result in alterations in the neuroplasticity of the sensorimotor areas of nervous system\(^ {35, 36}\), and physical inactivity has also been found to induce muscle weakness\(^ {37-39}\). Liepert et al.\(^ {40}\) showed that the anterior tibial muscle-controlling region in the primary motor area narrowed after plaster fixation of the ankle joint for complex fracture of the distal tibia or talus, suggesting an association between inactivity-induced muscle weakness and reduced primary motor area activation. The muscle force generated during maximum isometric knee extension decreased after UKA. We assumed that changes in the afferent information supplied by the knee joint were caused by surgical stress and inhibited the activation of the relevant sensorimotor area, thereby, narrowing the active regions in the bilateral sensorimotor
Fig. 2. Dynamic optograhic image of [oxyHb], [deoxyHb], and [totalHb] in the bilateral parieto-temporal region of patient 4 during maximum isometric extension 1 week after UKA. Images were taken at 1-sec intervals from 0 to 5 sec after the start of the task.

Fig. 3. Example of the anterior and posterior contra- and ipsilateral sensorimotor leg areas showing the time-course of [oxyHb], [deoxyHb], and [totalHb] during maximum isometric knee extension before and 1 and 2 weeks after UKA in one subject (Patient 4). a) anterior contralateral sensorimotor areas; b) posterior contralateral sensorimotor areas; c) anterior ipsilateral sensorimotor areas; d) posterior ipsilateral sensorimotor areas. The arrow indicates the 5-second maximum isometric knee extension period. The red, blue, and green indicate [oxyHb], [deoxyHb], and [totalHb], respectively.
areas at 1 and 2 weeks after UKA.

Since light-emitting and receiving fibers are directly applied to the scalp, fNIRS reflects oxygen metabolism and blood flow in tissues that can be penetrated by near-infrared radiation. Heartbeat, respiration, and blood pressure rhythm have been reported to influence fNIRS measurement \(^{41, 42}\). Therefore, it is necessity to confirm the influence of scalp blood flow on fNIRS measurements. In fNIRS during treadmill walking at different speeds \(^{43}\) and a perception task \(^{24}\), the simultaneously measured scalp (forehead) blood flow increased during the task, but the variation and level of increase varied among subjects. In this study, during maximum isometric knee extension, the time required to reach the maximum value of skin tissue blood flow and [oxyHb] of each of the individual channels were different. In addition, [oxyHb] in all of the sensorimotor areas during the task increased before UKA, but the increased areas narrowed after UKA. We suggest that the skin tissue blood flow is associated with changes in [oxyHb] measured by fNIRS during maximum isometric knee extension; however, cerebral cortex activation can be assessed using the analysis of the time course of [oxyHb].

In rehabilitative muscle strength training, the muscle strength can be increased by motor imagery and mental practice \(^{44–46}\), and it may prove to be a useful adjunct to traditional treatment options aimed at increasing the muscle strength \(^{47}\). Mental training reportedly enhances the cortical output signal, which drives the muscles to a higher activation level and increases strength \(^{48}\). The results of this study suggest that it is necessary to investigate the utility of muscle strength training that activates cerebral areas, such as motor imagery and mental practice, for early rehabilitation following knee arthroplasty.

**ACKNOWLEDGEMENTS**

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**REFERENCES**


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**Table 2.** [OxyHb] changes in sensorimotor leg cortical areas during isometric knee extension before and after UKA

<table>
<thead>
<tr>
<th>Time</th>
<th>Area</th>
<th>Contralateral</th>
<th>Ipsilateral</th>
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<tr>
<td></td>
<td>Mean maximum value (mM · cm)</td>
<td>Time (sec)</td>
<td>Mean maximum value (mM · cm)</td>
</tr>
<tr>
<td>Before UKA</td>
<td>Anterior</td>
<td>0.014 ± 0.013 (^{†})</td>
<td>2.8–5.0*</td>
</tr>
<tr>
<td></td>
<td>Posterior</td>
<td>0.020 ± 0.020 (^{†})</td>
<td>2.6–5.0*</td>
</tr>
<tr>
<td>1 week after UKA</td>
<td>Anterior</td>
<td>0.007 ± 0.017</td>
<td>n.s.</td>
</tr>
<tr>
<td></td>
<td>Posterior</td>
<td>0.015 ± 0.010 (^{†})</td>
<td>1.8–4.6*</td>
</tr>
<tr>
<td>2 weeks after UKA</td>
<td>Anterior</td>
<td>0.012 ± 0.021</td>
<td>n.s.</td>
</tr>
<tr>
<td></td>
<td>Posterior</td>
<td>0.016 ± 0.024 (^{†})</td>
<td>3.4–5.0*</td>
</tr>
</tbody>
</table>

* A significant increase in the oxyHb level was detected vs. the value at 0 seconds p < 0.05. † Mean oxyHb level for the 2 seconds of maximum isometric extension vs. mean oxyHb for the corresponding 2 seconds of rest, p < 0.05. n.s.: not significant

**Table 3.** The time needed to reach the maximum value of [oxyHb] in each channel and the skin tissue blood flow during maximum isometric knee extension

<table>
<thead>
<tr>
<th>Time (sec)</th>
<th>Skin tissue blood flow</th>
<th>OxyHb of channel 5</th>
<th>OxyHb of channel 6</th>
<th>OxyHb of channel 12</th>
<th>OxyHb of channel 13</th>
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<tbody>
<tr>
<td>5.1 ± 0.6</td>
<td>3.7 ± 0.7*</td>
<td>4.0 ± 0.6*</td>
<td>4.1 ± 0.6*</td>
<td>4.1 ± 0.6*</td>
<td></td>
</tr>
</tbody>
</table>

* Significant difference between the time points for the maximum oxyHb at each channel and skin tissue blood flow (p < 0.05)


6) Hurley MV: The effects of joint damage on muscle function, proprioception and rehabilitation. Man Ther, 1997, 2: 11–17. [Medline] [CrossRef]


