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BODY WEIGHT SUPPORT REDUCED PAIN AND NORMALIZED MUSCLE ACTIVATION STRATEGY IN PATIENTS WITH KNEE OSTEOARTHRITIS

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SUMMARY

The purpose was to study muscle activation strategy in patients with severe knee osteoarthritis (OA) and to elucidate if their activation strategy is related to pain. Weight support with lower body positive pressure was used as a dynamic exercise model. Seven male patients (13 OA knees) with severe OA and 9 healthy controls participated. They walked 3 km/hour at 100, 80, 60, 40 and 20% body weight (BW). EMG was recorded from knee extensor and flexor muscles.
Our data showed higher relative levels of muscle activation (mean EMG and co-activation index) in affected and less-affected knees with osteoarthritis during gait at 3 km/h compared to healthy controls. Likewise, a medio-lateral activation strategy towards increased lateral activation was found in the patients. However, these differences were diminished when the knee load was reduced with BW support. We concluded that the patients muscle activation strategy is a load dependent compensatory strategy due to the disease as unloading of the joint decreased pain and normalized muscle activation.

INTRODUCTION

Knee osteoarthritis (OA) is a common chronic degenerative joint disease primary affecting elderly. The most frequently affected compartment in the knee is the medial tibiofemoral compartment, e.g. [1]. OA is a major course of pain and physical disability e.g. in walking in elderly. Medical treatment includes pharmacological pain reduction and in severe cases total knee replacement. Advanced age, sex, overweight, joint injury and biomechanical factors are reported risk factors for development of the disease. Different muscle activation strategy, increased external knee adduction moment during stance and reduction in walking speed all of which are associated with knee joint loading has been reported in patients with OA e.g .[2]. However, it is not known if the identified biomechanical changes are associated with the development of OA or a compensatory mechanism due to the disease.

We used weight supported gait as a dynamic exercise model to study muscle activation strategy in patients with knee OA. If knee pain is reduced with increasing BW support in the present model, it allows us to study if a changed muscle activation strategy in the patients during gait is likely to be a compensatory strategy due to pain or a patient specific pattern independent of the presence of pain.

The purpose was to study the effect of body weight support on muscle activation strategy in patients with severe knee osteoarthritis and to elucidate if their activation strategy is related to pain.

METHODS

The study included 7 patients (60.9 years, BMI 29.8 kg/m^2) with severe medial knee OA (7 affected knees and 6 less-affected knees) and 9 healthy controls (52.7 years, BMI 25.1 kg/m^2). The patients were scheduled for total replacement of the knee. All participants walked 3 km/hour at 100 %BW, 80 %BW, 60 %BW, 40 %BW and 20 %BW on an anti-gravity treadmill (G-trainer, Alter-G, USA) [3]. The anti-gravity treadmill is constructed as a treadmill surrounded by a pressure chamber. The user wears a special pair of shorts and zips the waist into a pressurized airtight enclosure, which is suspended over the treadmill surface. Increased pressure in the chamber provides an up-ward force to the body. By controlling the pressure in the chamber it is possible to reduce the body weight of the individual accurately to 20 % of your normal body weight in 1% increments. The mobility is unrestricted and allows a natural gait pattern. Vertical ground reaction force was measured with built-in force transducers.

Surface EMG was recorded (1KHz) bilaterally from knee extensors (m. vastus lateralis and m. vastus medialis) and knee flexors (m. biceps femoris and m. semitendinosus) during gait. EMG during gait was analysed (30-s periods) as RMS values (21-ms RMS moved in 1-ms steps) and expressed as %EMG measured during maximum isometric knee extensions and knee flexions (best of three contractions). Extensor muscle activation, flexor muscle activation, flexion-extension co-activation index, lateral co-activation index (LQH) and medial co-activation index (MQH) were calculated. The co-activation index [4] was calculated as:

$[\Sigma_{i=1-n}$ (lowerEMG$_i$/higherEMG$_i$) x (lowerEMG$_i$+higher EMG$_i$)]/n, where lowerEMG is the activity of the less active muscle and higherEMG is the activity of the more active muscle. This method allows calculation of the instantaneous co-activation during gait. The co-activation index was calculated as the average instantaneous co-activation index across 30 s of gait.
Knee pain (left and right) during gait was measured on a VAS scale.

RESULTS AND DISCUSSION

Knee extension strength of the affected leg was reduced by 35% compared to the healthy controls whereas knee extension of the less-affected leg did not differ significantly from the control legs although a trend towards reduced muscle strength was found.
Peak vertical ground reaction force during gait decreased significantly from 12 N/kg at 100%BW to 4.1 N/kg at 20%BW (all legs). No differences were found between legs.

Knee pain in the affected knee during gait was reduced significantly when BW was supported. In the less-affected leg only two of the patients experienced pain during gait at 3 km/h. Pain was reduced in these two patients when BW was supported.

Mean extensor muscle activity was higher for affected (12.4(SE 2.1)%EMGmax and the less-affected (14.0(SE 3.2)%EMGmax legs than the control legs (5.1(SE 1.0)%EMGmax during gait at 100 %BW. BW support did not change extensor muscle activity in controls whereas a significant gradual decrease was found for the affected and the less-affected legs with increasing BW support. At 20 %BW no difference was found between extensor muscle activity in the OA legs and the control legs indicating normalization of muscle activation at low loads where the knee pain was significantly reduced.

Mean flexion-extension co-activation index was higher for the affected and the less-affected legs compared to the control legs. Thus, the flexion-extension co-activation index was 11.0(SE 2.8)%EMGmax (affected), 12.7(SE 4.5)%EMGmax (less-affected) and 5.2(SE 1.0)%EMGmax (control) during gait at 100%BW. The response to increasing BW support corresponded to the response for the mean extensor muscle activity. Thus, normalization of EMG was found at high levels of BW support.

The ratio between the LQH-index and MQH-index showed no medio-lateral side differences in the control legs at any of the investigated BW's. However, in the OA legs higher LQH-index was found at 100%BW ($p=0.014$) and 80%BW ($p=0.039$). A tendency to medio-lateral side difference was found at 60%BW ($p=0.67$) whereas at 40%BW and 20%BW no medial-lateral side differences were found in the OA legs. Thus, the medial-lateral muscle activation strategy, towards increased lateral muscle activation as found in the patient group is load dependent and may be a compensatory strategy to unload the medial tibiofemoral compartment of the knee. Our findings showed that the patients muscle activation strategy is related to pain rather than a fixed patient specific muscle activity pattern.

CONCLUSIONS

The knee OA patients showed higher levels of muscle activation (%EMGmax and co-activation index) than healthy controls at 100%BW indicating increased knee joint stabilization in the patients. Unloading the knee with increasing BW support decreased knee pain and knee muscle activation significantly in the patients. Furthermore, the medio-lateral muscle activation strategy towards larger LQH-index as found in the patients was normalized with increasing BW support. Thus, our results suggest that the patients muscle activation strategy is a load dependent compensatory strategy due to the disease as unloading of the joint decreased pain and normalized muscle activation.

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