Mechanisms for Work Related Disorders Among Computer Workers

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Abstract. Work related musculoskeletal disorders are common among computer workers, especially in the neck/shoulder region and the upper extremities. The relation between physical and psychosocial work load and generation of pain is still unclear. In this paper we describe five models. According to the often addressed Cinderella hypothesis, the pain is due to an overuse of low threshold muscle fibres. In a series of studies including intramuscular electromyography from the trapezius muscle, we have found several motor units that were active throughout coarse arm movements, during prolonged computer work tasks, and in both voluntary and stress induced contractions. Furthermore we have seen that motor unit statistics varies significantly between repeated measurements in one individual, hence the method would be inappropriate for group comparisons. Finally, we discuss a model based on a general model, literature studies, and own research.

Keywords: Mechanisms, chronic pain, Cinderella hypothesis, intramuscular electromyography, motor units, trapezius muscle.

1 Introduction

Work related musculoskeletal disorders are common among computer workers, especially in the neck/shoulder region and the upper extremities [1, 2]. The work tasks are often monotonous, and the muscle loads are normally relatively low and static. Although the question of how the pain is generated has been in focus in several research projects, there is still not one model that is generally accepted or proven. Instead the research has lead to several suggestions of mechanisms (for recent reviews see [3-5]). In this paper we shortly described proposed models for the generation of pain among i.a. computer workers. Then, we summarise our own work concerning the Cinderella hypothesis. Finally, we discuss a model based on a general model [6], literature studies, and own research.

2 Models for Pain Generation

In this chapter, five models for pain generation are described. They are all assuming the causes of static work at low muscular load, and cognitive tasks/psychosocial work factors as the original sources for the pain generation.

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2.1 The Blood Vessel-Nociceptor Interaction Hypothesis

According to a model proposed by Knardahl [7], the pain may originate from nociceptive interactions (stimulation of pain receptors) in the connective blood vessels that supply the muscle fibres. Three potential mechanisms may interact: (1) extraction of blood vessels (vasodilatation), (2) vascular production of pain producing substances, e.g. prostaglandins and nitric oxide, and (3) blood vessel inflammation. Concerning (1), Knardahl refers to an association between vasodilatation and pain in migraine patients, and on the pain effect of different vasodilating drugs.

2.2 The Hyperventilation Theory

A hyperventilation theory was developed by Schleifer and co-workers [8]; hyperventilation causes a drop in arterial CO_2, which causes a decrease of carbonic acid in the blood, which results in respiratory alkalosis, i.e. a rise in plasma pH above 7.45. This disruption in acid-base equilibrium will then in turn trigger a neuronal excitation, causing increased muscle tension and muscle spasms, with adverse effects for muscle tissue health. In support of the model, Schleifer and Ley [9] found low end-tidal CO_2 during sustained repetitive computer work, and with discrimination between high and low mental demands.

2.3 The Vicious Circle Hypothesis

A model describing the genesis and spread of muscular tension in patients with musculoskeletal pain syndromes was suggested by Johansson and Sojka [10], and further developed by Johansson [11] and Bergenheim [12]; a repetitive, static muscle contraction (in primary muscles) causes a production of metabolites, which activates gamma-motoneurons projecting to both primary and secondary muscles. This activation increases the static and dynamic stretch sensitivity and discharges of primary and secondary muscle spindle afferents (MSA). The increased primary and secondary MSA activities will increase the reflex-mediated component of the muscle stiffness, which in turn causes a further production of metabolites in primary and secondary muscles. An increased primary MSA activity will also hamper the proprioception, which could possibly lead to increased co-contractions. The result is a vicious circle that spreads and increases the sensations of pain throughout a body region.

There are e.g. proprioception-studies that support the hypothesis as well studies that contradict parts of the model.

2.4 The Nitric Oxide/Oxygen Ratio Hypothesis

Eriksen [13] assumes that neck myalgia is evoked when low-level contractions in the trapezius muscle are combined with psychological stress or prolonged head-down neck flexion at work; increased sympathetic nerve activity leads to arterial

vasoconstriction, which causes reduced capillary flow and reduced intracellular O_2 in the skeletal muscle. The sympathetic vasoconstriction seems to be preserved as long as the muscle activity is of low intensity. The vasoconstriction will also reduce the vascular removal of NO produced during the muscle fibre excitation-contraction process. A sharp rise in the NO to O_2-ratio will occur, which via biochemical processes will cause myalgic pain.

2.5 The Cinderella Hypothesis

The pain may be due to an overuse of low threshold muscle fibres causing damage at the muscle cell level. These fibres belong to low threshold motor units (MUs), which are recruited at the onset of muscle activation and which are firing continuously until the muscle is relaxed completely. This so-called Cinderella hypothesis [14,15] is based on a prescribed MU recruitment and de-recruitment size-principle order [16] and is supported by findings from cell morphology studies in myalgic muscles.

3 Our Own Investigations of the Possibility for the Cinderella Hypothesis to Explain the Pain Generation

When the Cinderella hypothesis was formulated the empirical data were taken from experiments including short term static contractions. One condition, among others, that should be met for the hypothesis to be accepted is that there are MUs that are continuously active under occupationally relevant conditions. Our group has in a series of experiments including intramuscular electromyography (EMG) from the trapezius muscle investigated the presence of continuously active low-threshold MUs.

We have also investigated the reproducibility of the method and its possibility to e.g. to be used in future studies comparing Cinderella MUs in different populations, for example in pain and no pain groups.

3.1 Intramuscular Electromyography in Experiments

Electrodes made of 0.05 mm diameter Teflon isolated stainless steel wires were used for intramuscular EMG from the right trapezius muscle. Figure 1 shows the setup in an experiment of computer work. A semi-automatic classification program was used for MUAP decomposition [e.g. 3, 17]. It utilises the entire signal complexes in each channel and compares MUAP morphologies. The algorithm has been developed facilitate tracing of MUAP that change shape over time. All experiments were carried out with the subject sitting in a chair. Maximal voluntary surface electromyographic activity (MVE) were measured in the end of the experiments.

In the reproducibility tests, MU activity from two neighbouring measurement points was measured in three repetitions (with at least two weeks in between) of 20-minute static contractions (trapezius) in 12 subjects. The intra- and inter subject variability in terms of e.g. number of C MUs was analysed.

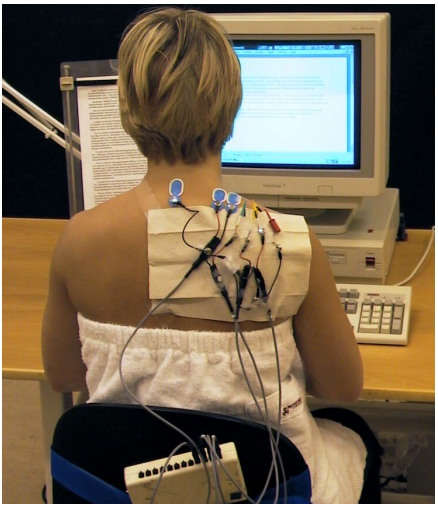

Fig. 1. The experimental set-up in a study of computer work

3.2 Results from Our Intramuscular Experiments

In our experiments including intramuscular EMG, we found motor units (MUs) in very small pick-up volumes, which were active throughout coarse arm movements, at different movement speeds [18]. In a study including mental stressors, we found, in 12 out of 14 subjects, MUs that were active both in voluntary and stress induced contractions (Lundberg et al 2002). In an other experiment including one hour of static low level contraction we found in 3 out of 8 subjects MUs that were continuously active the full hour [17]. Also in one hour computer work there were MUs active for one hour [20]. In most of the experiments there were a close relation between continuous MU activity and a static surface EMG level.

In the reproducibility tests, the intra-subject variances were very high for all MU parameters studied, which indicates low test-retest repeatability. Averaging MU activity results from two measurement points only moderately decreased the variances.

4 Discussion

In our own studies, we have found continuous MU activity (and "substitution") not only in a large variety of pure lab experiments, but also in 1h-computer work sessions. The findings support the Cinderella hypothesis, but they do not represent a final proof. The link from continuous MU activity to pain generation needs still to be further investigated.

The findings of the reproducibility tests show that decomposition of intramuscular wire EMG is an inappropriate technique for comparative studies between groups of individuals and that intra-subject comparisons should be restricted to the same recording point and test occasion.

Each one of the mentioned hypotheses may serve as a relevant explanation for, or contribute to, the mechanisms behind muscle pain in the context of computer work, i.e. monotonous work often under stressful conditions. But none is yet generally accepted. There are additional hypotheses suggested in the literature [4].

The nitric oxide/oxygen ratio hypothesis is still "young"; instead of traditionally hampered blood macrocirculation, it concerns insufficient blood supply on an active muscle fibre level. Since insufficient muscle fibre rest will intensify and speed up this mechanism, there might be a connection between this model and the implications of the Cinderella hypothesis. Findings of mitochondrial disturbances and reduced capillarisation in women may, together with our own findings of continuous and sustained MU activity, support the theory that prolonged low-level workloads and psychological stress lead to localised muscle cell ischemia and lactic acid evoked muscle pain, as described by Eriksen [13].

We here propose a pathway (also described in [3]) between computer workloads and chronic muscle pain. It is based on the more general model proposed by Winkel and Westgaard [6], is schematically described in Figure 2. In many aspects, this pathway independently resembles the conclusions given in a recent review article by Visser and van Dieën [4].

Computer work is often characterised by prolonged, monotonous tasks and with low physical demands. These external exposures can lead to an internal exposure of prolonged low-level muscular activity with few periods of muscle rest. Computer work may also include psychological stress. This exposure primarily lead to an internal exposure/response of mental strain and secondarily to prolonged low-level muscular activity with few periods of muscle rest.

We saw, as described, that prolonged low-level muscular activity and few periods of muscular rest can lead to continuous and sustained activity of selective MUs. As a reaction to the induced mental strain, sympathetic nerve activity may be increased, leading to arterial vasoconstriction and reduced capillary flow, and since the muscular activity is of a low-level character, this sympathetic vasoconstriction can be preserved [21]. That is, a combination of low-level muscular activity and induced mental strain can lead to prolonged vasoconstriction. Furthermore, during low-level muscular activity, the K+ concentration is maintained close to the resting level [22]. Release of intracellular K+ is one important feedback mechanism in fatigue [23]. Thus, low-level muscular activity can lead to an absence of muscular fatigue perception.

The continuous and sustained MU activity may constitute, together with the prolonged vasoconstriction, a risk for localised ischemia in selective muscle fibres. Localised ischemia may, via mitochondrial deficiencies, lead to ATP insufficiency and lactic acid evoked muscle pain [13]. An insufficiency of ATP may also affect the homeostasis of Ca2+ released from the sarcoplasmic reticulum during the muscle fibre excitation-contraction phase [23], leading to skeletal muscle damage; [24, 25]. Thus, the combination of continuous, sustained MU activity and prolonged vasoconstriction may, via an acutely impaired energy metabolism for selective muscle fibres, lead to a production of nociceptives and acute muscle pain.

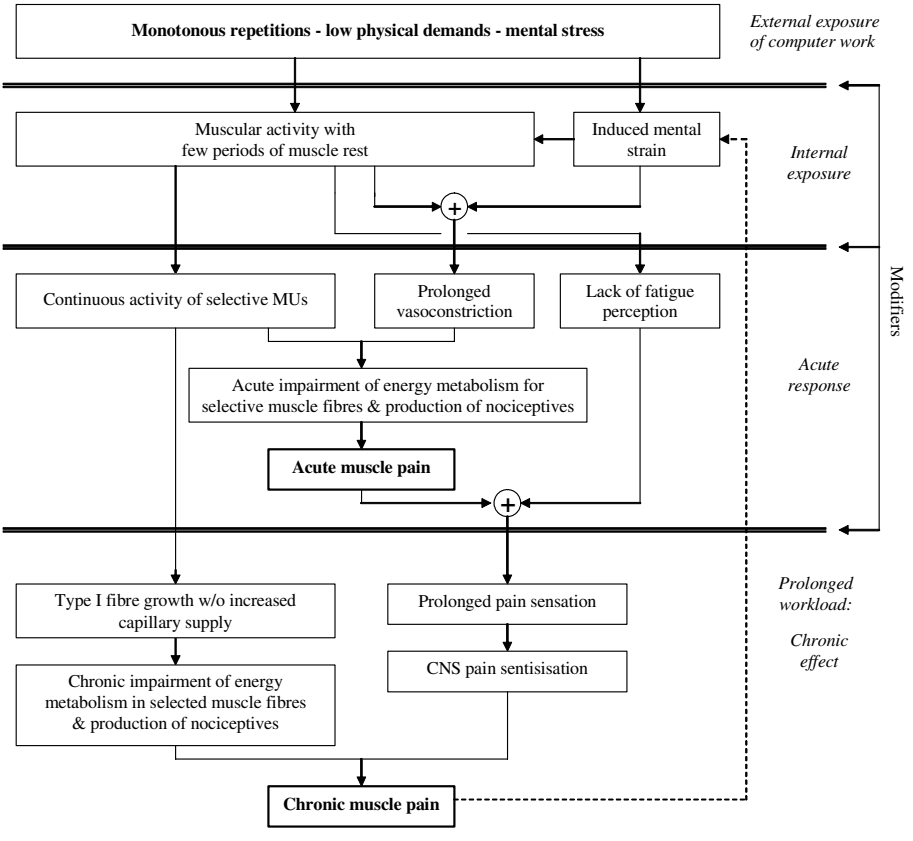

Fig. 2. Proposed pathway between computer work and chronic muscle pain. Double lines indicate areas susceptible to modifiers, e.g. individual factors. The dashed line indicates a feedback loop.

Two ways to chronic pain are suggested, in the case that the workload described above is prolonged. First, there is a risk that an acute pain sensation is prolonged, especially as a perception of muscular fatigue may not be present. Eventually, the prolonged acute pain sensation can, via CNS pain sensitisation, turn into a chronic pain behaviour [26], which does not necessarily need pain stimuli to be maintained. Second, a prolonged workload can also lead to a growth of type I muscle fibres without increased capillary supply, as inferred by Hägg [15]. This is supported by findings of deteriorated microcirculation of blood through specific muscle fibres [27-29], in myalgic trapezius muscles. Rosendal [30] demonstrated that trapezius myalgia was associated with locally increased anaerobic metabolism and increased levels of substances potentially activating peripheral nociceptive processes. Thus, a prolonged workload may, through type I fibre growth and deteriorated microcirculation, lead to a chronic impairment of energy metabolism in selected muscle fibres, production of nociceptives and chronic muscle pain.

Finally, a chronic pain may induce feedback loops that potentially aggravate muscle pain further. Musculoskeletal complaints may also increase the motor response to psychological stress, decreasing the muscle rest [31].

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