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Effects of physical and mental stressors on muscle pain

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Physical and mental stressors as risk factors for pain development are discussed. These multifaceted stressor terms are narrowed down so that physical stressors are represented by muscle activity recorded by electromyography (EMG), while mental stress is considered synonymous with psychosocial stress in vocational studies; in experimental studies cognitive stress is used as a model. Pain in the shoulder and neck are focused and related to EMG recordings of activity in the trapezius muscle. Major challenges in this field include proper risk assessment at low physical work loads and criteria for evaluating stress as a risk factor. A 3-factor conceptual model is presented in which the independent dimensions physical work load, mental stress, and individual sensitivity determine the risk of shoulder and neck complaints. It is pointed out that a predominant reduction in physical work load for many jobs and an increasing interaction between work conditions and the general life situation of workers pose particular challenges for risk assessment.

Key terms electromyography, mental stress, musculoskeletal, physical work load, shoulder and neck.

Effects of physical and mental stressors on muscle pain — a note on terminology

Physical and mental stressors are generally accepted as potential causes of pain development (1). In occupational settings, the evidence is mainly based on studies utilizing a combination of epidemiologic methods and physiological recordings (2), but laboratory studies also contribute to our understanding (3). It is a problem in the attempt to summarize this evidence that different research traditions use different, but overlapping terminology. In this review, a "stressor" is a condition or factor that causes a physiological response. A physical stressor corresponds in most cases to a *mechanical exposure* (appendix in reference 4), but may also include effects of *physical exposures* such as lighting. A mental stressor is considered equivalent to *psychosocial exposure* in an occupational setting, but it is represented by imposed mental demands in laboratory studies. It can be subdivided into cognitive ("task-related") and emotional ("interpersonal") stressors. "Stress" is the nonspecific response to a stressor, consisting of several of the physiological responses in the model of Westgaard & Winkel (5). Both stress and stressors can be *risk factors* for muscle pain symptoms.

Muscle activity as a risk factor for muscle pain

Traditionally, heavy manual handling has been considered the principal cause of musculoskeletal pain in occupational settings. Later, sustained posture and repetitive movement were accepted as important risk factors in many work situations. Even more recently, mental stressors were added to the list. The relative importance of different risk factors is in part dependent on the body region under strain: heavy lifting (low back), walking and standing (legs), and repetitive work tasks (arm and shoulders) are predominantly important as risk factors for musculoskeletal pain at specific body locations. This review focuses on pain in the shoulder and neck, since this body region is best studied with respect to the combined effects of physical and mental stressors.

The physical stressor is represented by surface electromyographic (EMG) recordings of muscle activity in this review. In the epidemiology of work-related musculoskeletal disorders, the main aim is to identify risk factors of the disorders. Muscle activity is, in that sense, not the best representation of occupational risk due to physical stressors, as it also represents a considerable individual element through differences in, for example, motor

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coordination patterns, physical strength, and work technique. Nevertheless, this variable is presumably the most relevant instrumented measurement with which to represent a regional physical stressor that can function as a marker of a physiological process that initiates pain. The implicit query posed by the title of this paper is therefore reformulated into 2 specific queries: (i) is there an association between recorded muscle activity and muscle pain development and (ii) is mental stress as a risk factor represented by elevated muscle activity levels? The aim of this paper is to give an overview of evidence, mainly from the laboratory of my co-workers and I, which provides partial answers to the queries.

Interpreting muscle activity as a risk indicator of muscle pain symptoms

This issue was first examined by recording fatigue effects in sustained contractions. The classic force-fatigue curve of Monod (6) and Rohmert (7) is well known in the ergonomic literature, and it has been further examined in more recent studies (8). These experiments show that a muscle load higher than 8—10% of the maximal voluntary contraction cannot be sustained. Muscle loads at this level or higher are therefore commonly considered a cause of musculoskeletal pain. Queries can be raised regarding the validity of this evaluation as regards occupational work loads, which consist of force-varying rather than of true static contractions. A more critical aspect is that many workers develop disorders at considerably lower work loads (or the equivalent muscle activity levels) than indicated as safe by this criterion (5, 9).

In recent years there has been more focus on the time variables of physical work load. The association between the mechanical exposure and risk of musculoskeletal disorders has been conceptualized as a U-shaped exposure health-effect curve, indicating elevated risk both at high and low exposure (10). It was proposed that mechanical exposure depends on the variables level (amplitude), force variation pattern (repetitiveness), and duration and that risk at low exposure levels is due to long-duration sustained or repetitive exposure patterns. The 3 variables in the model are conceptual entities, and we still struggle to provide good operational definitions. An activity pattern analysis (exposure variation analysis) (11) provides a good overview of exposure variation but results in a matrix of variable values that is difficult to apply as a risk assessment tool. Risk assessment on the basis of the static component of the EMG activity pattern, a suggestion originally made by Jonsson (12), would consider elements of both amplitude and the temporal activity pattern and would be easier to evaluate. The suggested acceptable level of 2% of maximal EMG activity has some support in field studies, at least when workers reporting adverse psychosocial exposure are excluded (2).

Another hypothesis relates the pain induction process to overexertion of the functional units of the muscle, the motor units (13). The hypothesis is based on the Henneman et al (14) recruitment principle, stating that there is a fixed order of recruitment of motor units. Thus low-threshold motor units may show sustained activity patterns even at low overall activity levels. The motor unit hypothesis complements the U-shape exposure-effect curve by emphasizing the time dimension in the risk assessment of mechanical exposure, and it provides a physiological explanation for the lack of association between EMG amplitude and muscle pain symptoms at low activity levels (15). It leaves us with the problem of identifying means with which to perform proper risk assessment, since motor unit activity patterns are not identified in the surface EMG. One approach has been the "EMG gap" analysis, which quantifies the appearance of short, subsecond breaks in the EMG activity pattern. This analysis has distinguished workers with and without pain in some, but not all vocational studies (16—18). Experimental studies with the simultaneous recording of single motor unit activity patterns and surface EMG have indicated that EMG gaps are associated with an increased probability of motor unit substitution (ie, the temporal replacement of a motor unit with another unit of initially higher threshold) (19, 20).

The selective-overexertion hypothesis has indirect support in the finding of "ragged-red fibers" in trapezius muscle (ie, fibers that have a fragmented morphologic appearance and stain for NADH-tetrazolium reductase) indicating mitocondrial dysfunction and disturbed metabolism. They are particularly prominent in the descending regions of the trapezius muscle (21). Whether these fibers are more frequent among workers with sustained or repetitive work tasks is debated (22). Finally, it must be emphasized that, to the extent motor unit activity pattern causes pain development, this mechanism does not invalidate other pain induction mechanisms; instead it supplements them.

Mental stressors as a source of muscle pain

Work-related mental stressors can be subdivided into cognitive and emotional stressors, which are related to worktask demands and to social relationships at work. Whether these aspects represent similar or different qualities of risk in terms of musculoskeletal health effects is not known. In recent work mental stressors have figured as a prominent risk factor in the development of musculoskeletal pain, with the shoulders, neck, and head as the dominant body locations. The best-known model for characterizing mental stressors is the 3-factor model of Karasek & Theorell (23), which was developed to evaluate the risk of cardiovascular disorders in terms of psychological demand, decision latitude, and social support. The same model is increasingly used to indicate risk of musculoskeletal disorders (24). Alternative models with risk indicators that are variations of these themes exist (25). It is a problem that the variables represent a *perception* of the psychosocial environment and are context-sensitive (eg, decision latitude may have different meanings depending on the work setting). This can be one reason why these variables do not show consistent results between studies. A further concern is that the interview questions, on which the 3 constructs in the Karasek-Theorell model are based, may not be appropriate for the work situations studied. In a recent study of shop assistants, one of the reported stressors was customer relation, which we felt was not properly represented in the standardized questionnaire. This uncertainty may cause inconsistent responses to questions. There also appeared to be genuine differences in the perception of the criticality of the different mental stressors; this finding may explain the lack of statistical association between pain and mental stressors (unpublished results). Half the workers nevertheless felt their complaints were due to conditions at work.

Experimental studies of mental stress, muscle responses and pain development

Motor activation through mental stress, both cognitive and emotional, has been described in many experimental psychophysiological studies (26—28). We have explored this phenomenon in laboratory studies to examine whether this type of motor response is associated with pain development due to stress. We have obtained the following results:

1. The trapezius muscle shows a clear motor response to cognitive stress and is among the most responsive muscles in our experimental setting (29).

2. The motor response is dose-dependent (ie, a higher level of stress is associated with higher muscle activity level) (30).

3. Complex tasks with a higher level of information processing are associated with higher muscle activity levels, at least for some subjects (31).

4. The low-variation, largely stable activity pattern observed in surface EMG recordings reflects sustained motor unit activity (32 and figure 1).

Thus the trapezius response to cognitive stress has many features we would look for if the response is to be associated with pain development in the shoulder and neck. Other studies have shown interaction effects when combining physical and mental stressors [ie, added mental stress causes an increase in muscle activity when the same physical work task is performed (33, 34)].

The possible association between stress-induced muscular activity and pain was examined by extending the cognitive stressor test to 1 hour and including more recording sites in the upper body region (ie, bilateral recording from the trapezius, splenius, temporal and frontal muscles). Pain scores (bilateral in the case of the trapezius and temporal sites) were made on a visual analogue scale (VAS) for these locations, with pain reports every 10 minutes (3). Experiments were carried out on normally pain-free subjects and on patients with shoulder myalgia, fibromyalgia, tension headache, and cervicogenic headache (a unilateral headache).

For the referents, pain developed predominantly in the shoulder and neck during the test. The pain response was at a low level, subsiding quickly after the test (3). Those with a pain response in the shoulder of at least 10

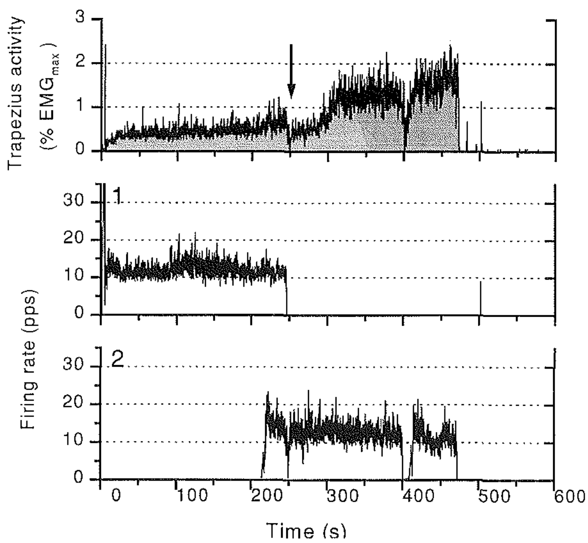

Figure 1. Surface electromyographic (EMG) activity (upper panel) and firing rate (low-pass filtered at 0.5 Hz; two lower panels) of 2 individual motor units during a laboratory experiment with mental stress (20). A low-level, sustained activity pattern is shown in the surface EMG during the first part of the test, derived from sustained firing of individual motor units. In other experiments, many examples of sustained motor-unit firing throughout the 10-minute test period have been observed (32). The figure also illustrates an example of motor unit substitution: a new unit is recruited at 210 seconds when the surface EMG level is slightly increased. Soon after, there is a temporal break in the activity pattern. Both units are silenced, but only the last recruited unit with the initially higher recruitment threshold recovers. The first unit shows signs of reactivation 505 seconds into the experiment. The experiment is further an example of motor-unit substitution coinciding with an EMG gap in the surface EMG. We have observed several such instances, both during controlled, static contractions and during uncontrolled muscle activation like typing (19). Substitution may alternatively happen without gaps in the surface EMG signal.

VAS units responded with higher trapezius EMG activity than those who did not develop pain at this body location. The correlation was statistically significant but weak, which can be understood if sustained motor unit activity is the underlying critical feature in the response. There was, however, no association between muscle activity and pain development in the underlying muscles for the other recording sites.

Similar studies of the patient groups showed more marked pain development than for the pain-free referents. Pain development followed the diagnostic features of the patients (ie, located in the shoulder and neck region for shoulder myalgia patients, widespread in case of fibromyalgia patients, and so on). All the patient groups, including the headache patients, reported pain development in the shoulder region (one-sided for the cervicogenic headache patients). In contrast to the spatial variation in pain responses, the muscle response was stereotypic for all the patient groups, with an enhanced activity level in the trapezius muscle the first 10 minutes of the test and subsiding slowly thereafter. The trapezius response was only present on the symptomatic side for the cervicogenic headache patients (35). Thus, in cases of shoulder pain and trapezius EMG activity, the results of the different series of experiments were supportive of muscle activity involvement in pain initiation (ie, patients who had a higher level of pain development in the shoulder also had a stronger trapezius EMG response). This finding was not the case for the other recording sites. Within the fibromyalgia group, the amplitude of the trapezius response

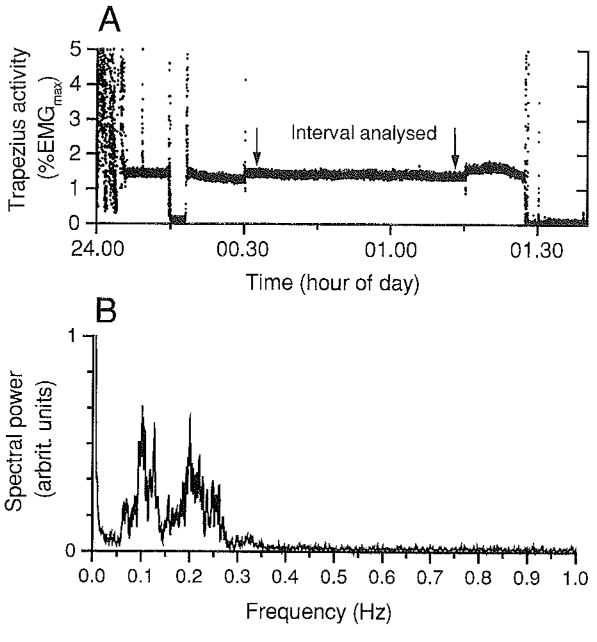

Figure 2. Sustained trapezius activity during the first 2 hours of a sleep period (A). Part of this activity period was analyzed by fast fourier transform (interval between vertical arrows), and it shows rhythmic components corresponding to respiratory and cardiovascular regulatory rhythm (B).

correlated not only with shoulder pain development, but also with pain development at other locations, despite a lack of correlation between the corresponding pain responses (Bansevicius et al, unpublished report). Thus the trapezius EMG response stands out as a special case of stress-induced muscle activation. To the extent muscle activity is related to pain development in a dose-response manner, this is apparently only the case for trapezius and shoulder pain among the sites and muscles we have examined. A hypothesis that aims to explain pain development during stressful conditions as a result of sustained, low-level motor activity must explain these negative results. A pertinent question is whether another hypothesis can be found to match our findings.

One way of resolving apparently conflicting results is by postulating that the trapezius EMG activity is not causal to the development of pain, but is a parallel physiological response. Limbic components of the brain, which control the autonomic nervous system, also have a strong influence on the spinal motor system (including segmental and proprioceptive interneurons). The limbic component of the motor system, termed the "third motor system" by Holstege (36), is concerned with gain setting of motoneurons and triggering mechanisms for rhythmic activity (eg, respiration, shivering, cardiovascular regulation). The influence of the 3rd motor system over the function of striated muscles varies from muscle to muscle, from strong control over respiratory muscles like the diaphragm to relatively minor influence over peripheral flexor muscles. The trapezius may receive relatively strong input from limbic components of the motor system, as indicated by the strong response in our laboratory experiments with induced stress. We have obtained experimental evidence of such influences through 24-hour recordings of muscle activity in the trapezius muscles (figure 2A). During sleep, when somatic motor activity is minimal, a sustained low-level activity pattern, similar to that observed during the cognitive stress test, can be observed in the trapezius muscle. This activity may contain rhythmic components that reflect respiratory rhythm, as well as the slower cardiovascular regulatory rhythm of 0.1 Hz (figure 2B). This occurrence represents sustained strain on individual motor units that may contribute to the development of the ragged-red and moth-eaten fibers in the trapezius muscle. The autonomic influence that activates trapezius motoneurons may, however, still be a parallel phenomenon to other physiological processes of etiologic significance.

Risk evaluation of physical and mental stressors for shoulder and neck pain

First, our attempts to establish risk indicators valid for work situations with low physical work load and mental stress do not invalidate established risk indicators for work situations with high physical work load.

A consideration of risk indicators at a low physical work load must build on the results of field studies. Experimental studies like those described in the previous section yield interesting results but are of practical relevance only if procedures or tools for risk assessment can be found. The issues of the sensitivity and validity of risk assessment in a field setting must be addressed. A main conclusion of our vocational studies is that mental stressors are not well represented in EMG recordings at work. The EMG gap analysis has novel features not fully explored and may work as a risk assessment tool, as a supplement to such traditional approaches as evaluation by the static EMG level. However, the studies only show that workers with many EMG gaps within a group may have less pain than those with few gaps, but there is no criterion for the number of gaps that represents a risk of musculoskeletal pain. We have no evidence to indicate that the gap analysis can detect *stress-induced* muscle pain.

The criterion that a static activity level above 2% of the maximum EMG activity represents a risk of muscle pain should be reexamined. In our experience, this criterion distinguishes workers with shoulder pain from those without pain when workers reporting the presence of mental stressors are excluded. The fact that pain may develop at lower EMG levels, possibly for reasons unrelated to biomechanical exposure, have made this criterion less popular. My belief is that this can be a valid criterion for assessing traditional biomechanical exposure through EMG recording, but it must be supplemented by criteria that address the time dimension of biomechanical exposure (ie, criteria for "intermittent static" work).

We have had some success in using tests with EMG recordings to differentiate between workers with and without shoulder pain (17). These tests can show sustained EMG activity patterns during nominal rest or high activity levels during controlled movement. The specificity of this type of assessment is not good, and such tests do not represent a reasonable approach to occupational risk assessment, since they represent risk through person-based variables that do not reflect the work situation.

Figure 3 presents a conceptual risk analysis model for pain in the shoulder and neck (37). The model shows increasing risk along 3 independent (in risk assessment terms) axes: physical work load, stress and individual sensitivity. Traditional risk assessment is carried out along the axis of physical work load as evaluated in terms of amplitude, repetitiveness, and duration. Individual assessment can be based on muscle activity recordings, while external exposure assessment (description of work tasks, workplace layout, etc) can be used for the group assessment of risk. The stress dimension includes both work stress and stressors outside work and may, at least

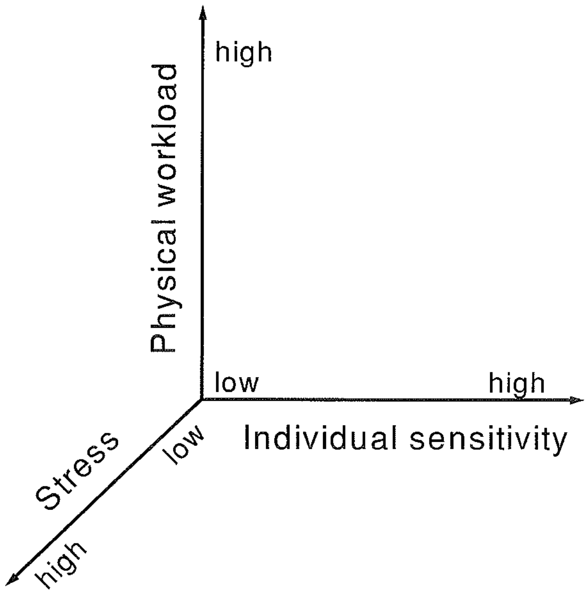

Figure 3. A conceptual model for assessing health risk in the shoulder and neck region. Health risk is increasing along 3 independent dimensions. High scores along 2 of the 3 axes imply a high risk of complaints. See the text for additional details.

in part, be evaluated according to the Karasek-Theorell model. Although physiological responses to stress (including muscle activity) are well documented, it is currently not possible to perform risk evaluation of the stress dimension with physiological measurements. Individual sensitivity was originally indicated by a history of disorders, but other indicators can be included (eg, a tendency of sustained motor unit activation or excessive use of force when not necessary). Loops in the model will exist (eg, sustained stress may in the longer-term cause elevated tension at rest). The inclusion of such considerations would result in a dynamic model for mechanisms of pain induction, but it is beyond the scope of the model in figure 3, which serves as a basis for a one-off risk assessment.

Risk assessment for special purposes would omit parts of the model (eg, the individual component is normally not considered when occupational risk is evaluated). It is, however, an increasingly important challenge to address risk assessment in occupational situations in which the physical work load is low and complaints develop through an interaction between the workplace, leisure-time activities, and individual factors. This challenge is clearly of both a scientific and a policy nature.

References

1. Kroemer KHE, Grandjean E. Fitting the task to the human. London: Taylor and Francis, 1997.

2. Westgaard RH, Jansen T, Jensen C. EMG of neck and shoulder muscles: the relationship between muscle activity and muscle pain in occupational settings. In: Kumar S, Mital A, editors. Electromyography in ergonomics. London: Taylor & Francis, 1996:227—58.
3. Bansevicius D, Westgaard RH, Jensen C. Mental stress of long duration: EMG activity, perceived tension, fatigue, and pain development in pain-free subjects. Headache 1997; 37:499—510.
4. Westgaard RH, Winkel J. Ergonomic intervention research for improved musculoskeletal health: a critical review. Int J Ind Erg 1997;20:463—500.
5. Westgaard RH, Winkel J. Guidelines for occupational musculoskeletal load as a basis for intervention: a critical review. Appl Ergon 1996;27:79—88.
6. Monod H. Contributions á l'étude du travail statique. Paris: Institut National de Securité, 1956.
7. Rohmert W. Ermittlung von Erholungspausen für statische Arbeit des Menschen. Int Z Angew Physiol Einschl Arbeitsphysiol 1960;18:123—64.
8. Björkstèn M, Jonsson B. Endurance limit of force in long-term intermittent static contractions. Scand J Work Environ Health 1977;3:23—7.
9. Westgaard RH. Measurement and evaluation of postural load in occupational work situations. Eur J Appl Physiol 1988; 57:291—304.
10. Winkel J, Westgaard RH. Occupational and individual risk factors for shoulder-neck complaints: part II — the scientific basis (literature review) for the guide. Int J Ind Erg 1992; 10:85—104.
11. Mathiassen SE, Winkel J. Quantifying variation in physical load using exposure-vs-time data. Ergonomics 1991; 34:1455—68.
12. Jonsson B. Kinesiology. With special reference to electromyographic kinesiology. Contemp Clin Neurophysiol (EEG Suppl no 34) 1978;417—28.
13. Hägg GM. Static work loads and occupational myalgia — a new explanation model. In: Anderson PA, Hobart DJ, Danoff JV, editors. Electromyographical kinesiology. Amsterdam: Elsevier, 1991:141—3.
14. Henneman E, Somjen G, Carpenter DO. Excitability and inhibitability of motoneurons of different sizes. J Neurophysiol 1965;28:599—620.
15. Jensen C, Nilsen K, Hansen K, Westgaard RH. Trapezius muscle load as a risk indicator for occupational shoulder-neck complaints. Int Arch Occup Environ Health 1993;64:415—23.
16. Veiersted KB, Westgaard RH, Andersen P. Electromygraphic evaluation of muscular work pattern as a predictor of trapezius myalgia. Scand J Work Environ Health 1993;19:284—90.
17. Vasseljen O, Westgaard RH. A case-control study of trapezius muscle activity in office and manual workers with shoulder and neck pain and symptom-free controls. Int Arch Occup Environ Health 1995;67:11—8.
18. Hägg GM, Åström A. Load pattern and pressure pain threshold in the upper trapezius muscle and psychosocial factors in medical secretaries with and without shoulder/neck disorders. Int Arch Occup Environ Health 1997;69:423—32.
19. Westgaard RH, De Luca CJ. Motor unit substitution during sustained contractions: implications for ergonomics. In: Seppälä P, Luopajärvi T, Nygård C-H, Mattila M, editors. Proceedings of the 13th IEA congress; vol 4 (Musculoskeletal disorders, rehabilitation). Helsinki: Finnish Institute of Occupational Health, 1997:237—9.
20. Westgaard RH, De Luca CJ. Motor unit substitution in long—duration contractions of the human trapezius muscle. J Neurophysiol 1999;82;501—4.
21. Lindman R, Eriksson A, Thornell L—E. Fiber type composition of the human female trapezius muscle: enzyme—histochemical characteristics. Am J Anat 1991;190:385—92.
22. Larsson B, Libelius R, Ohlsson K. Trapezius muscle changes unrelated to static work load. Acta Orthop Scand 1992; 63:203—6.
23. Karasek R; Theorell T. Healthy work. New York (NY): Basic Books, 1990.
24. Toomingas A, Theorell T, Michélsen H, Nordemar R, Stockholm—MUSIC I study group. Associations between self—rated psychosocial work conditions and musculoskeletal symptoms and signs. Scand J Work Environ Health 1997; 23:130—9.
25. Johansson JÅ, Rubenowitz S. Risk indicators in the psychosocial and physical work environment for work—related neck, shoulder and low back symptoms: a study among blue— and white—collar workers in eight companies. Scand J Rehabil Med 1994;26:131—42.
26. Davis RC. Patterns of muscular activity during "mental work" and their constancy. J Exp Psychol 1939;24:451—65.
27. Goldstein IB. Electromyography. A measure of skeletal muscle response. In: Greenfield NS, Sternback RA, editors. Handbook of Psychophysiology. New York (NY): Holt, Rinehart & Win, 1972:329—65.
28. Cacioppo JT, Petty RE, Losch ME, Kim HS. Electromyographic activity over facial muscle regions can differentiate the valence and intensity of affective reactions. J Pers Soc Psychol 1986;50:260—8.
29. Wærsted M, Westgaard RH. Attention—related muscle activity in different body regions during VDU work with minimal physical activity. Ergonomics 1996;39:661—76.
30. Wærsted M, Bjørklund RA, Westgaard RH. The effect of motivation on shoulder—muscle tension in attention—demanding tasks. Ergonomics 1994;37:363—76.
31. Wærsted M, Bjørklund RA, Westgaard RH. Shoulder muscle tension induced by two VDU—based tasks of different complexity. Ergonomics 1991;34:137—50.
32. Wærsted M, Eken T, Westgaard RH. Activity of single motor units in attention—demanding tasks: firing pattern in the human trapezius muscle. Eur J Appl Physiol 1996;72:323—9.
33. Weber A, Fussler C, O'Hanlon JF, Grierer R, Grandjean E. Psychophysiological effects of repetitive tasks. Ergonomics 1980;23:1033—46.
34. Lundberg U, Kadefors R, Melin B, Palmerud G, Hassmén P, Engström M, et al. Psychophysiological stress and EMG activity of the trapezius muscle. Int J Behav Med 1994;1:354—70.
35. Bansevicius D, Sjaastad O. Cervicogenic headache: the influence of mental load on pain level and EMG of shoulder—neck and facial muscles. Headache 1996;36:372—8.
36. Holstege G. Descending motor pathways and the spinal motor system: limbic and non—limbic components. Prog Brain Res 1991;87:307—421.
37. Westgaard RH, Jensen C, Hansen K. Individual and work—related risk factors associated with symptoms of musculoskeletal complaints. Int Arch Occup Environ Health 1993; 64:405—13.