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REVIEW ARTICLE

SHOULDER PAIN IN HEMIPLEGIA REVISITED: CONTRIBUTION OF FUNCTIONAL ELECTRICAL STIMULATION AND OTHER THERAPIES

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Objectives: **Post-stroke shoulder pain is probably the most frequent complication in hemiplegia and has repercussions on motor rehabilitation and the psychological equilibrium of the patient. The strategies for prevention and treatment are presented.**

Aetiology: **Among the various factors contributing to the occurrence of shoulder pain in hemiplegia, some are related to the joint, such as lesion of the rotator cuff tendons, reflex sympathetic dystrophy, inferior-anterior subluxation of the head of the humerus, whereas others are related to the neurologic lesion such as central post-stroke pain, lack of sensibility, unilateral neglect and spasticity.**

Prevention: **Efforts should be made from the start to keep the shoulder in an ideal position at all times and movement of the shoulder and upper limb should be carried out with care.**

Treatment: **Will be aimed to the cause of pain and passive or active range of motion exercises will be encouraged. Physical, medical and surgical treatments have improved over the last few decades. Functional electrical stimulation in patients with shoulder pain and subluxation, applied early after onset of the stroke, has shown beneficial positive effects on subluxation, pain and mobility. Efforts should therefore be made to better understand the post-stroke shoulder pain in order to provide better outcomes of rehabilitation and thus improve quality of life for patients.**

Key words: stroke, shoulder pain, shoulder subluxation, spasticity, prevention, treatment, electrical stimulation.

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INTRODUCTION

According to the World Health Organization, cerebral vascular accident (CVA) represents the second most common cause of death in industrialized countries (1). It is also a major cause of disability, since it results in paralysis and cognitive defects in the surviving population. Data derived from the literature suggest that 25% of the patients suffering from a stroke die within a few weeks (2). Despite organized and appropriate care, one third of the survivors remain dependent or bedridden, another third make a full recovery, leaving the last third with some degree of residual functional incapacity. About half of all hemiplegic survivors will be left with a non-functional arm (3). Lifetime costs of stroke are high. For instance, ischaemic strokes in the USA in 1990 have been estimated to cost US$90,981 per patient (4). In Australia the 1993–94 annual cost was assessed at about Australian $40,000 per patient (5) and these costs increase when complications occur.

One of the most important and frequent complications of stroke is shoulder pain, with a prevalence of 34–84% (6, 7). It is independent of age and sex (8, 9) and occurs in the second week after the stroke. The consequence of shoulder pain for motor rehabilitation and psychological well-being makes it an important factor. This paper reviews the aetiological factors and options for prevention and treatment. It aims to improve understanding of this condition and to promote the potential of prevention and treatment.

AETIOLOGY

Among the various factors that contribute to the occurrence of shoulder pain following CVA, some are related to the neurological lesion and others to the joint.

Neurological factors

Moskowitz & Porter (10) suggested the possible occurrence of a peripheral nerve lesion occurring as a result of traction of the arm, pressure on the brachial plexus or secondary to peripheral nerve trauma. A brachial plexus injury has been described in hemiplegia (11) and suggested as a possible effect of subluxation (12, 13), but this has not been shown to be common in patients (14, 15).

Another cause is secondary to central nervous system mechanisms. In such cases, hemiplegic shoulder pain is often associated with sensory disturbance and/or neglect and cognitive disturbances. In their study, Broeks et al. (2) found that 74% of patients suffered from disordered sensory functioning and Poulin et al. (16) reported significantly more painful hemiplegic shoulders in patients with left-sided hemiplegia.

Spasticity, which frequently follows the hypotonic phase in patients with hemiplegia can be a cause of pain (6). Furthermore, significant spasticity of the upper extremity can greatly interfere with its functional use, in particular when spastic antagonists counteract selective voluntary muscle activity. Shoulder mus-

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cles have been implicated in the concomitant internally rotated and adducted posture of the upper extremity. Spastic muscles are painful when stretched; this could be one of the mechanisms involved in enthesopathy due to increased tendon traction. (17). Spasticity itself can also be painful at rest.

Factors related to the joint

The prevalence of rotator cuff tendon tears in arthrographic studies was reported in 33–40% of hemiplegic patients (18–20), but these studies showed that the incidence of cuff rupture was no greater in hemiplegic patients than in age-matched controls. Other similar work in painful hemiplegic shoulders failed to show any tendon lesion, but demonstrated in 23 of 30 patients signs of an adhesive capsulitis (14). The latter could result from a tendino-bursitis of the shoulder.

A diagnosis of reflex sympathetic dystrophy syndrome is frequently made in patients with hemiplegic shoulder pain. Its prevalence has been reported in the literature as between 12.5% and 27% (6, 21, 22). Recently Daviet et al. (23) presented a study using their scale for this problem and found a prevalence of 34% in a group of 69 hemiplegic patients. However, this scale does not take into account subluxation of the shoulder, which seems to represent an important prognostic factor according to the majority of the authors. For some, the relation between subluxation and shoulder pain in patients with hemiplegia remains controversial, but it appears for many reasons, that treatment of shoulder subluxation continues to be the standard of care in many rehabilitation facilities (24). Antero-inferior subluxation may cause pain directly or indirectly. Evidently, shoulder subluxation is difficult to refute when it represents the only sign of pain. Subluxation may mask or inhibit functional recovery by limiting the range of motion (ROM) and therefore increase disability. Shoulder subluxation is likely to contribute to the pathogenesis of other painful conditions by stretching neurovascular and musculoskeletal tissues, and thus lead to immobilization and atrophy of the rotator muscles. It has been suggested that there is a correlation between early subluxation and the subsequent development of shoulder pain (6, 25) as well as a correlation between subluxation and other types of shoulder pathology seen in hemiplegic patients (2, 3, 14, 15, 24).

Radiographic measurement offers the opportunity for an early diagnosis and for allowing the evolution of the problem through repeated assessment. Several techniques have been described (20, 25–27). Sophisticated techniques, such as three-dimensional X-ray or the use of digitizer and computer, seems to give good correlation with the clinical data (28, 29). The simplest and most available technique remains the plain antero-posterior radiograph, but special attention must be made to the patient's position (preferably standing in the upright position or sitting without any arm support) (25–27).

Finally, it should be remembered that other conditions, such as angina pectoris, fracture or even metastasis of the humerus head or the glenoid cavity, can be responsible for pain, especially in the early stages.

PREVENTION

Efforts must be made to keep the upper extremity in a constantly good position from the onset. Mobilization of the upper limb and, in particular the ROM of the shoulder, should be performed carefully. Nursing care also requires special attention from the onset. Therapists should instruct family members and hospital personnel on methods of handling a patient with a hemiplegic upper extremity (30–32).

As the patient becomes able to sit and transfer from bed to wheelchair, it is important to maintain a good posture for the upper extremity. Elevation of the arm with a pillow is not only used to avoid stretching of soft tissues or hanging of the upper limb, but to maintain the shoulder in its correct position to avoid pain and dependent oedema of the hand. The use of an arm-tray for wheelchair users helps to keep the head of the humerus in good position. During ambulation the flaccid hemiplegic patient should wear a sling, as demonstrated by van Ouwenaller et al. (6) and other authors (33, 34).

Once spasticity appears, this sling becomes contraindicated, since spasticity produces an imbalance in the muscles of the shoulder and arm and contributes to the common clinical picture of an adducted, internally rotated arm with elbow flexion. It is then essential to give adequate exercises and optimize the range of joint motion to maintain a functional pain free range. However, despite every effort to prevent shoulder pain, clinical experience shows that this complication can still develop.

TREATMENT

Treatment must be aimed first at the cause, when this is known.

Neurological factors

Central post-stroke pain. Central post-stroke pain (CPSP) is a common finding in up to 8% of the patients (35) and can be held responsible for a part of shoulder pain. In CPSP, classical oral medication aimed at neuropathic pain should be used. The tricyclic antidepressants, such as amitryptiline or nortryptiline, and the anticonvulsants (carbamazepine, gabapentin, clonazepam and phenytoin), have been shown to be beneficial (36, 37), while the use of selective serotonin reuptake inhibitors is still debated.

Even in neuropathic pain, the use of opioids must not be ruled out. Ketamin, a N-methyl-D-aspartate antagonist has proved useful (38). When using any of these agents, the patient must be made aware of the fact that the benefits of therapy will usually only take place after the onset of side-effects. Regrettably, CPSP is often resistant to treatment.

Hemineglect

Hemineglect is most prevalent in the first weeks post stroke and may decrease spontaneously afterwards. Coincidence with shoulder pain has been reported, but the latter continues to be present even after the hemineglect disappears (2, 16). Rehabilitation may help to reduce hemineglect by drawing the patient's

Table I. *Functional electrical stimulation session characteristics*

Sequence	First	Second	Third
Duration (min)	90	30	10
Current	Rectangular biphasic	Rectangular biphasic	Rectangular biphasic
Frequency (Hz)	8	40	1
Impulsion time (μs)	350	350	350
Contraction ratio (on:off)	1:5	1:5	1:5

attention to the neglected side and stimulating movements contralateral to the lesion. On a very limited set of patients, Prada & Tallis (39) used electrical stimulation on the skin of the affected forearm in order to draw the patient's attention to his neglected arm. A more rapid improvement was demonstrated during the treatment phase. A recent Cochrane Library review evaluated the effects of cognitive rehabilitation in spatial neglect following stroke and shows that, although patients improve their performance on neuropsychological tests, the global effect on disability was inconclusive (40). At present, the treatment of sensory loss is disappointing.

Spasticity

Spasticity after stroke is very common. Although not inevitably, it is a frequent cause of shoulder pain. Physical, medical and surgical management of generalized or focal spasticity has much improved in the last decades (41).

Physical modalities, such as cold or heat application, electrical stimulation, the Bobath approach and others, as well as some drugs (42) will temporarily reduce muscle spasticity without curing it.

A wide range of oral drugs is available (43), most act on the central nervous system (CNS).

Tizanidine is an alpha-2 agonist, acting both at spinal and supraspinal levels, thus acting both in spasticity of spinal or cerebral origin. Oral baclofen, a $GABA_B$ receptor agonist, is mainly indicated in spasticity of spinal origin. Dantrolene inhibits calcium release from the sarcoplasmic reticulum, thus acting outside the CNS. It is particularly beneficial for spasticity of cerebral origin (44). Unfortunately, the incidence of severe hepatotoxicity is significant. The use of diazepam, a $GABA_A$ receptor agonist, is less common nowadays due to its numerous side-effects (e.g. sedation, weakness). Despite this, its existence should not be overlooked, as it is quite a potent antispastic agent, especially for spasticity of spinal origin (45).

Nowadays, the use of cannabinoids seem to be much favoured at least by patients, both for the treatment of pain and for spasticity. However actual trials seem inconclusive (46, 47).

The use of neurolytic agents, such as alcohol or phenol, in the management of focal spasticity has been widely developed by reducing spasticity, improving the range of joint motion and facilitating function (48). Phenol has an immediate and reversible conduction blocking effect as a local anaesthetic (49) and a long-term effect on demyelination and axonal degeneration (50, 51). Alcohol acts differently. It does not paralyse the muscle as phenol does, which also produces muscle atrophy (52, 53), but acts on gamma nerve fibres without producing necrosis of the muscle fibres (54–56). Besides peripheral or intramuscular neurolysis with one or the other products, intramuscular administration of Botulinum toxin type A (BTX-A) is now currently applied for the same purposes. BTX-A weakens the activation of the muscle by selectively blocking the release of acetylcholine at the neuromuscular junction (57–59). In a review of the literature on this subject, Van Kuijk et al. (60) emphasized the importance of adequate patient and goal selection in treating upper extremity spasticity following stroke with BTA-X. Hesse et al. (61) found evidence of a synergistic effect of early electrical stimulation combined with BTX-A injections in the treatment of spastic post-stroke upper extremity.

Neurosurgery for spasticity management is now used in a limited number of cases when non-invasive techniques have failed. Dorsal rhizotomies were performed to decrease regional spasticity and these techniques have developed toward the more precise and sophisticated dorsal root entry zone-otomy (DREZ-otomy) procedures (62, 63). Iatrogenic side effects are frequent (among them decrease of lemniscal sensitivity) and are described in up to 70% of the patients (64).

For more focal spasticity, peripheral neurotomies can be performed. Their results can be first assessed through an anaesthetic block on the nerve bundle involved.

These techniques are destructive and non-reversible. A less invasive and reversible approach is the use of intrathecal baclofen (ITB), which, infused through a subcutaneously implanted programmable pump, can prove useful in some cases. It should not be considered first choice treatment for upper limb spasticity, although it seems much more useful in lower limb spasticity (65–67). Upper extremity spasticity reduction has been achieved in cerebral-palsied children by using synergy between ITB and DREZ-otomy (68). Electrical stimulation of the spinal cord dorsal columns has been used with varying results and can be sometime considered an option (69, 70).

Causes related to the joint

These are more common.

Rotator cuff tendon lesions

Rotator cuff tendon lesions may benefit from physical treatment and in some instances from steroid injections (71). This also applies to tendinitis and capsulitis. Local ultrasound, as well as some modalities of heat (short wave, hot packs) are reported as effective.

Reflex sympathetic dystrophy

Reflex sympathetic dystrophy, now included in the complex regional pain syndrome, is a complicated disorder and is not always as easy to manage. Nevertheless, in the majority of the cases treated very early after their first signs, the prognosis is

Table II. *Functional electrical stimulation treatment, similar results after 1 and 2 years*

Features	FES group ($n = 57$)	Control group ($n = 58$)
Painless (%)	80.7**	55.2
Decreased subluxation (%)	78.9*	58.6
Motor function recovery (%)	82.5**	60.3

* $p < 0.05$, ** $p < 0.01$.

good. In addition to medication, such as oral steroids (72), or non-steroidal anti-inflammatory drugs (32, 73) with or without calcitonin (74), rehabilitation is the treatment of choice in the form of ROM and specific mobilization techniques (75).

Shoulder subluxation

The lack of effective interventions for managing shoulder subluxation has prompted the investigation of neuromuscular electric stimulation for treating this problem in hemiplegia. In 1994 Faghri et al. (76) described the effectiveness of functional electrical stimulation (FES) on shoulder subluxation and shoulder pain in hemiplegic patients. The promising results of this preliminary study enabled us to assess its further use for the same purpose (77). A total of 256 hemiplegic patients were included in a study carried out by the Department of Physical and Rehabilitation Medicine at the University Hospital of Geneva a few years ago. Patients were alternatively assigned to either a control group or a group receiving FES on their hemiplegic shoulder. The FES was administrated by mean of a commercially available Medi-Compex® electrical stimulator, programmed as described in Table I. Patients had 2 hours of daily FES sessions following 3 separate patterns 5 days a week for a total of 5 weeks. After a follow-up of 2 years, the results were in favour of the FES group. The maximal improvement in pain, subluxation and, surprisingly, motor recovery was observed at 6 months in the FES group. These results slightly improved again after 12 months (Table II) and remained constant for up to 24 months. A significant reduction in subluxation in the FES group, as well as a decrease of pain and improvement in the recovery and function of the upper extremity indicates that FES is an effective intervention in early treatment. The diversified program of FES given in the study facilitates the recruitment of different muscle fibres and subsequently to that of adjacent fibres, thereby increasing the shoulder's muscles strength.

These results strongly suggest that FES does not only help in decreasing pain and subluxation, but also speeds up functional improvement. Some authors (78, 79) found the same findings on the effectiveness of FES. Another recent study investigating the feasibility of percutaneous intramuscular neuromuscular electric stimulation for treating shoulder subluxation and pain in patients with hemiplegia confirmed prior results (24). These positive results seem to be encountered in patients treated early with FES, whereas those treated in the chronic phase seem to have no benefit (80). Low frequency transcutaneous electrical nerve stimulation has also proved to be useful in reducing pain and improving motor function in the upper extremity (81–83).

DISCUSSION AND CONCLUSION

Shoulder pain is one of the most frequent complications of hemiplegia. The severity of pain in many patients makes it one of the most devastating sequelae to follow the initial neurological event. The aetiology of shoulder pain may vary and it is very often difficult to isolate a specific cause. More commonly, several factors are involved and this problem represents a multifactorial pathology, which is probably due to the exceedingly complex functional and structural anatomy of the shoulder. Smooth, effortless and complete movement of the shoulder girdle requires synchronous motion of the gleno-humeral and related joints, which will, therefore, invariably be compromised by a neurological event or anatomical defect. To further our understanding of the factors that lead to the development of shoulder pain, the dynamic nature of many conditions seen in hemiplegia must be considered. For instance, Joynt (84) recently suggested that the subacromial area of the shoulder could be the pain-producing site.

Furthermore, many of the predisposing factors that cause shoulder pain in hemiplegia are not usually identified until the spastic phase has evolved. Spasticity in effect is not only a cause of pain itself, but it may potentialize other factors and become a cofactor in pain. The management of spasticity should aim not only at reducing muscle tone, but should be directed at restoration or improvement of function and at reducing carer burden (6).

Besides spasticity, shoulder subluxation is among the most frequent causes contributing to pain, although it, in itself, is not necessarily its cause. It does appear to be evident, that the potential for injury is higher when subluxation is prolonged and it is more difficult to treat in a chronic state (78).

The prime treatment should be thorough prevention. Despite this, clinical experience shows us that a painful shoulder can nevertheless appear.

Different treatments can be applied. Ideally, successful ones depend on identifying the cause of the symptoms. This is not always possible, and sometimes empirical treatment is offered. The validity and reliability of critical outcome measures should be established. The role of adequately performed exercises has been emphasized (85–87), since a higher prevalence of shoulder pain has been reported in patients who did not continue to exercise soon after their discharge from treatment (88).

Among the therapeutic possibilities, different options have been presented. Recently it has been demonstrated that FES, which was first suggested by Faghri et al. (76), plays an important role in acting on or decreasing shoulder pain, reducing disability—in particular shoulder subluxation—and finally improving motor function. Chantraine et al. (77) confirmed the benefit and the efficacy of this treatment in a larger sample of

patients. The efficacy of FES has since been confirmed by other authors (24, 78).

Nevertheless, as encouraging as these studies may be, further investigation is necessary to gain a better understanding of the natural history of shoulder pain in patients with hemiplegia. The mechanism of shoulder pain needs to be better defined, as does the positive action of FES on pain, disability and motor function in the hemiplegic shoulder (89).

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CONTINUING MEDICAL EDUCATION – CME – IN REHABILITATION MEDICINE

AND

EXERCISE FOR DOCTOR'S UNDER SPECIALIST TRAINING

UEMS European Board of Physical and Rehabilitation Medicine

The publication of the review article: Vuagnat H, Chantraine A: Shoulder pain in hemiplagia revisited: Contribution of functional electrical stimulation and other therapies. J Rehabil Med 2003; 35; 1–8 and the CME questions on the following pages have been sponsored by

CME QUESTIONS

Please choose one answer for each question.

1. After a cerebral vascular accident, what is the percentage of patients left with a definitive non-functional arm?

   a. 10%
   b. 20%
   c. 30%
   d. 50%
   e. 60%

2. What is the cause of shoulder pain in hemiplegia?

   a. Tendinitis
   b. Peripheral nerve lesion
   c. Metastasis of the head of the humerus
   d. Antero-inferior subluxation of the shoulder
   e. All of the above

3. Which of the following proposals is not responsible for shoulder pain?

   a. Hemineglect
   b. Sensory loss
   c. Absent reflexes in the arm
   d. Flaccidity
   e. Spasticity

4. In the hemiplegic patient, what is the typical upper extremity position due to spastic muscle imbalance?

   a. Internal rotation, adduction and flexion
   b. Internal rotation, abduction and flexion
   c. Internal rotation, abduction and extension
   d. External rotation, abduction and extension
   e. External rotation, adduction and flexion

5. From the following statements, which is not considered an effective prevention of shoulder pain in hemiplegia?

   a. To keep the shoulder in good position by having the arm elevated
   b. To use a sling in a flaccid hemiplegia
   c. To teach the family members how to handle the involved upper limb
   d. To keep the shoulder in the correct position by leaving the arm to hang down
   e. To maintain gentle range of motion of the upper extremity

6. Which one of the following actions of the phenol used to reduce spasticity is not true?

   a. It causes atrophy by muscle fibre necrosis
   b. It has an immediate and reversible effect as a local anaesthetic conduction blockade
   c. It has a long-term effect on axonal degeneration
   d. It is not painful
   e. It has a long-term effect on demyelination

7. One of the following statements on the use of botulinum toxin type A to reduce hemiplegic upper limb spasticity is not correct:

   a. It is applied by intramuscular injection
   b. It selectively blocks the release of acetylcholine at the neuromuscular junction
   c. Its action is reversible
   d. Its effect is potentialized by early electrical stimulation
   e. It selectively acts on gamma-nerve fibres

8. In the hemiplegic painful subluxed shoulder, the beneficial effects of functional electrical stimulation results in:
   a. A significant reduction of subluxation
   b. A significant decrease of pain
   c. Some reduction in spasticity
   d. An improvement of motor function
   e. All of the above

9. Which of the following statements on functional electrical stimulation for hemiplegic shoulder pain is not true:

   a. The sessions should last at least 2 hours per day
   b. The current frequency should be comprised between 90 and 110 Hertz
   c. The whole treatment should last 5 weeks
   d. The contraction time (ratio on/off) should be 1:5
   e. The impulsion time should be at least 350 μsec

10. Which of the following treatments for the hemiplegic painful shoulder has a very short temporary action on spasticity:

    a. Local application of cold
    b. Alcohol intramuscular injection
    c. Electrical stimulation combined with botulinum toxin type A
    d. Intramuscular neurolysis of phenol
    e. Injection of botulinum toxin type A

11. Which are the most widely used medications for central post-stroke pain?:

    a. Steroids
    b. Opioids
    c. Non-steroidal anti inflammatory
    d. Anticonvulsants and tricyclic anti-depressants
    e. Selective serotonin reuptake inhibitors

12. Among the following treatments used to reduce spasticity, which one does not act on the central nervous system?

    a. Diazepam
    b. Dantrolene
    c. Baclofen
    d. Tizanidine
    e. Clonazepam

Answers

1. d; 2. e; 3. c; 4. a; 5. d; 6. d; 7. e; 8. e; 9. b; 10. a; 11. d; 12. b.