

# Clinical Diagnosis of Muscle Cramp and Muscular Cramp Syndromes

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**Summary.** The diagnosis of muscle cramp is based on clinical features. Algorithms are presented for the diagnosis of muscle cramp and cramp syndromes.

**Key words:** Muscle cramp – Syndromes – Diagnosis

## Introduction

Muscle cramp is an extreme form of muscular contraction. During muscle cramp actual damage to muscle tissue may occur, as shown by the elevated CK levels in blood and the myoglobinuria which frequently follow cramp. Rupture of muscle tissue and even adjacent tendons may take place during muscle cramp, indicating failure of physiologic inhibition mechanisms [1]. Muscle cramp is excruciatingly painful and afterwards the affected muscle may remain sore and tender for a period of days. Persons suffering from nocturnal muscle cramp often go to bed terrified with the prospect of being surprised in their sleep by intensely painful cramp.

Epidemiologic data on muscle cramp are sparse. In a recent neuroepidemiological survey of the adult population of the Netherlands the overall frequency of muscle cramp in 1988 was estimated at 37% [2]. For a symptom so common and distressing, muscle cramp has attracted surprisingly little attention [3]. Cramp syndromes are dealt with very concisely in most textbooks of clinical neurology. Nevertheless, their diagnosis is essentially based upon clinical criteria and electromyography is seldom needed for support. In this short survey, we provide a practical basis for diagnosing muscle cramp as well as several muscular cramp syndromes.

## Clinical Diagnosis of Muscle Cramp

The words muscle cramp and muscle spasm are erroneously used as synonyms. Muscle spasm indicates any

form of involuntary muscular contraction, including those without pain and those of long duration e.g. spasticity, rigidity and muscular hypertonus. Painless muscular spasms like myoclonus, dystonia and dyskinesia are often wrongly called muscle cramp.

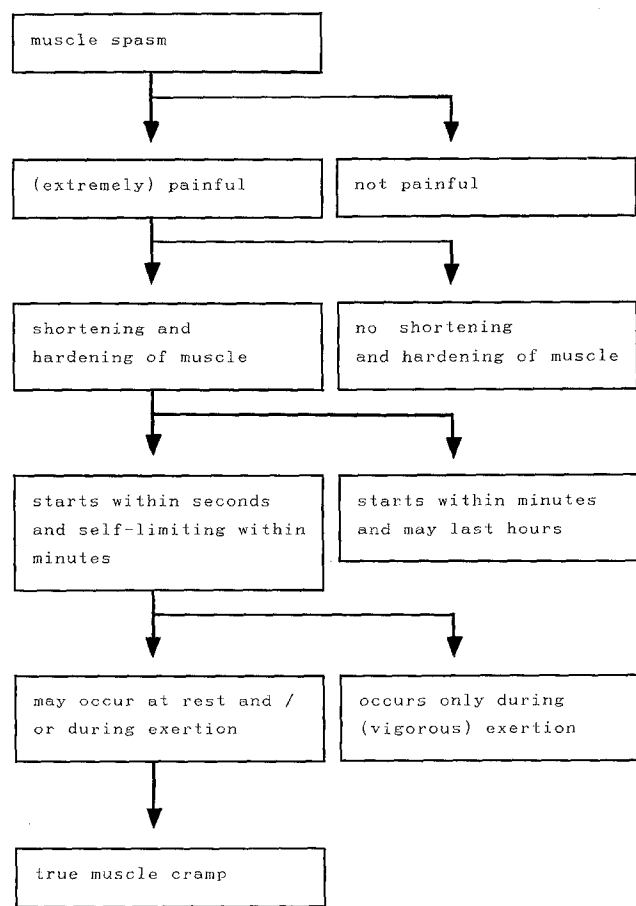
Intestinal colics caused by spasms and narrowing of segments of bowel are frequently called cramps, possibly because of the intense accompanying pain. Painful affections of clearly non-muscular origin e.g. varicosis (in German: Krampfadern), intermittent claudication, neuralgia and arthralgia are sometimes mistaken for muscle cramp.

In a true muscle cramp, there is sudden involuntary and painful shortening of muscle, attended by visible or palpable knotting of muscle, often with abnormal posture of the affected joint, and relieved by passive stretching or massage [4]. Muscle cramp starts and ends with muscle twitching in different parts of the affected muscle. It may occur at rest after trivial movement, especially when the muscle is relaxed and shortened, but also after forceful contraction, especially when the muscle shortens. Ordinary muscle cramp is self-limiting within minutes.

This description separates muscle cramp from other painful muscle disorders without shortening of muscle e.g. myositis and myalgia on the one side and involuntary shortening of muscle without pain e.g. myotonia and tetany on the other side (Fig. 1). Myotonic spasm is in fact a delayed muscular relaxation, lasting no longer than minutes. It does not occur spontaneously at rest like muscle cramp and may be provoked by activity, percussion, electrical or mechanical stimulation of muscle.

Characteristic in tetany is the fixed sequence of clinical symptoms [5]. Sensory nerves giving rise to tingling paraesthesia are more susceptible than motor nerves causing fasciculation and muscle spasm. Symptoms begin distally and spread up the limbs to involve the trunk. The characteristic posture of the hand in carpopedal spasm and the wellknown Chvostek, Erb and Trousseau signs are highly diagnostic. Tetanic spasm may last continuously for hours or days, while muscle cramp is self-limiting within minutes. Tetanic spasms are not usually painful, but may be, especially in prolonged attacks. Hyperventilation tetany may clinically mimic ordinary muscle cramp and muscular cramp syndromes.

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**Fig. 1.** Clinical diagnosis of muscle cramp

Diagnostic difficulty may arise with myopathic muscle contracture and occupational cramp as they cause muscular discomfort and painful stiffness. In few rare metabolic myopathies myopathic muscle contracture may be induced by vigorous exercise or ischaemia. It does not occur spontaneously at rest or with minor movement like muscle cramp and may last an hour or more. The term myopathic muscle contracture rather than contraction points to the marked disproportion between the intense mechanical muscle shortening and the paucity of accompanying electrical activity (EMG-silence) [6]. Electromyography in true muscle cramp shows high-amplitude high-frequency discharges. The pathophysiology of myopathic muscle contracture is related to exhaustion of muscle energy stores and resembles the state of rigor mortis [6].

In clinical practice contracture is defined in a different sense as a limitation of movement at a joint, sometimes traceable to a painless fixed shortening of fully relaxed muscle [7].

Occupational cramp is caused by the involuntary co-contraction of agonists and antagonists and restricted to specific activities that have been practiced to a point that they can be carried out without conscious thought. The resulting abnormal posture interferes with intended highly skilled and complex movements (writing, playing musical instruments, working as a telegraphist). It is considered a form of focal dystonia, although true dys-

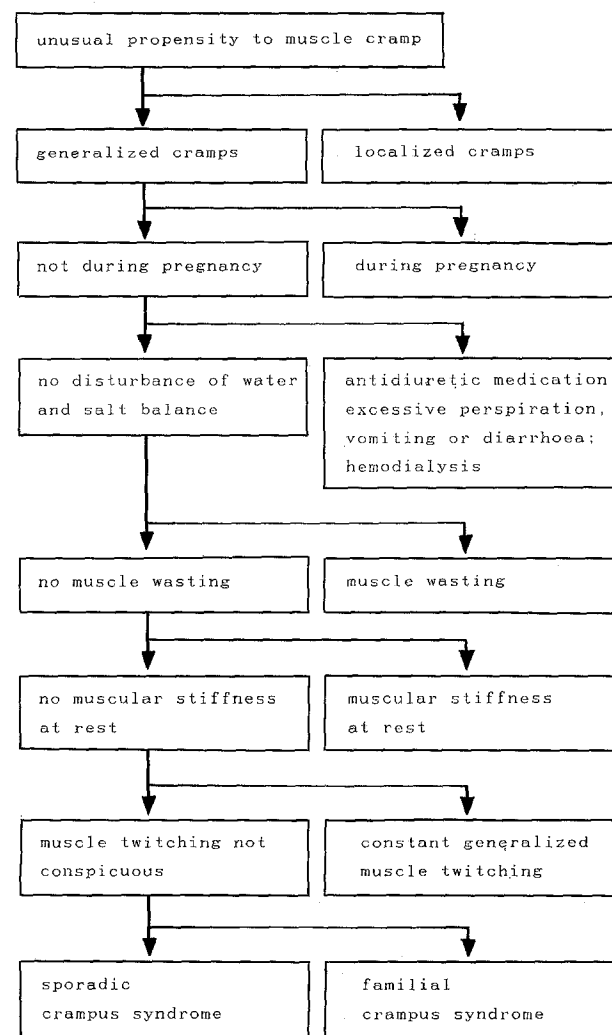
tonia is usually not linked to a specific movement and is not ordinarily painful [4].

### Clinical Diagnosis of Muscular Cramp Syndromes

In most persons muscle cramp occurs rarely, but some people are truly disabled by unusual propensity to cramp [2, 6]. Frequent muscle cramp may be a manifestation of one of several neuromuscular syndromes. Correct diagnosis of these muscular cramp syndromes may have prognostic implications. Some of these syndromes are medically treatable. Their diagnosis is based on taking a careful medical history and neurologic examination. Electromyography, laboratory investigations or muscle biopsy are seldom needed as supporting evidence.

Frequent, strictly localized muscle cramp and fasciculations, often in one calf or foot, may be produced by compression of a nerve root in muscles supplied by that root (Fig. 2) [8]. Generalized neurological affections like motor neuron disease may rarely initially present with localized muscle cramp.

Common riskfactors for generalized muscle cramp should then be asked for. Disturbances of water and salt



**Fig. 2.** Clinical diagnosis of muscular cramp syndromes

balance, especially serum hypo-osmolality, are important, readily treatable causes of frequent muscle cramp [9]. Pregnancy-associated muscle cramp may be provoked by physiological changes in serum during pregnancy [10].

### Muscle Cramp and Muscular Stiffness

Although muscle soreness following cramp may be accompanied by slight muscle spasm, continuous muscular stiffness at rest is not a feature of ordinary muscle cramp. Three cramp syndromes are known to produce marked muscular stiffness at rest: tetanus, strychnine poisoning and the stiff-man syndrome. Care should be taken to exclude the characteristic spasticity of motor neuron disease, that may also accompany muscle cramp and the rigidity of extrapyramidal disease, or the rigid spine syndrome.

*Clostridium tetani* infection is a major health problem in developing countries and correct diagnosis remains of vital importance for patient-management [8]. Tetanus toxin reaches the central nervous system by retrograde intra-axonal transport and blocks presynaptic inhibition of spinal motoneurons. Muscle stiffness is usually the initial symptom and involves first the area of injury and infection and then the muscles of the jaw (trismus). Reflex spasms may be absent in milder cases; they usually begin within 48 h after stiffness is observed. Any external stimulus may precipitate a paroxysmal reflex spasm with sudden simultaneous contraction of agonists and antagonists, while the patient is fully conscious and in intense pain. Paroxysms also occur spontaneously.

Strychnine blocks postsynaptic inhibition in the central nervous system by competing with glycine. In rare cases of strychnine poisoning muscle stiffness occurs first, followed by reflex spasms, that are not as prominent as in tetanus.

The stiff-man syndrome was first described by Moersch and Woltman [11] and resembles a chronic form of tetanus, characterized by persistent muscular stiffness and painful reflex spasms. The illness is sporadic, occurs predominantly in adults and follows a slowly progressive course. Proximal and axial musculature of trunk and neck become increasingly taut and stiff. Walking becomes laborious (monolithic gait). Paroxysmal muscle cramps, often initiated by external stimuli, are superimposed on the persistent stiffness. Trismus is absent, an important clinical distinction from tetanus. Symptoms disappear completely during sleep and anaesthesia. Neurological examination and cerebrospinal fluid are normal. Fasciculation should be absent, clinically as well as electromyographically. Diazepam often offers symptomatic relief.

### Muscle Cramp and Muscular Twitching

In a survey conducted by questionnaire, a quarter of a group of medical personnel reported experiencing more

than one episode of benign fasciculation per month. Eleven out of 539 healthy persons reported the symptom daily [12]. Some persons with widespread benign fasciculations also suffer from frequent muscle cramp. Muscle cramp frequently starts and ends with fasciculatory muscle twitching. Three clinical syndromes include an association of frequent widespread muscle cramp and fasciculatory muscle twitching: the muscular cramp-fasciculation syndrome (Denny-Brown and Foley syndrome), the continuous muscle fibre activity syndrome (Isaacs' syndrome) and lower motor neuron disease.

Denny-Brown stressed the close relations between myokymia (continuous undulating muscle twitching), frequent benign fasciculation and muscle cramp, most prominent in the calf muscles [13]. Wasting, weakness and alteration of reflexes should be absent in this syndrome. Many persons suffering from the muscular cramp-fasciculation syndrome can induce cramps at will. This syndrome has been reported only once to evolve into motor neuron disease with weakness and wasting [14].

Isaacs described the continuous muscle fibre activity syndrome, a sporadic disorder that may begin at any age, characterized clinically by continuous widespread fasciculation and myokymia, muscular cramps, prolonged spasms and pseudomyotonia in the distal limb musculature [15]. Pseudomyotonia differs clinically from true myotonia because delayed muscular relaxation increases instead of decreases with repetitive activity and percussion myotonia is absent. Carpopedal spasm, increased sweating and slight peripheral neuropathy may be present. Symptoms persist during sleep and anaesthesia and are alleviated by carbamazepine.

Lower motor neuron disease may cause generalized muscle cramp and fasciculations. Clinical diagnosis of lower motor neuron disease demands the presence of muscle wasting. This, however, may also be a feature of severe compression of a nerve root. Electromyographic signs of fasciculations as well as of denervation and re-innervation are seen both in lower motor neuron disease and in severe radiculopathy. In the absence of clear signs of radiculopathy only clinical or electromyographic evidence of spreading disease can offer diagnostic certainty.

If frequent muscle cramps occur alone, without other neurologic abnormalities, the name "crampus syndrome" will have to do. Sporadic and familial forms are known [16–18]. In this survey extremely rare cramp syndromes e.g. the Satoyoshi syndrome and atypical variants, often only reported in single patients or families, are omitted for the sake of clarity [19]. In cases that do not fit into clear-cut clinical syndromes, computer-aided literature study is recommended.

The use of phrases like continuous motor activity syndrome and motor unit hyperactivity state should be discouraged. They are used for several clear-cut muscular cramp syndromes as well as many atypical variants, sometimes even characterised by the absence of motor unit activity.

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