

Rest cramps in the elderly

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Clinical experience suggests that muscle cramps are common in elderly people. They can rarely be ascribed to a specific cause and, as they commonly occur during sleep, are usually labelled 'rest cramps' or 'night cramps'. 'For a symptom so common and distressing, night cramps have attracted surprisingly little investigative attention.' So wrote Gootnick in 1943 [1], and his comments are equally pertinent nearly half a century later, so poor is our knowledge of this apparently simple symptom. Why are rest cramps so common in elderly people? Why do they occur most often in the leg, and why at night when the leg muscles are relaxed and warm? This review draws on the available evidence to respond to these questions and also outlines a practical management plan for the condition.

Clinical features

Muscle cramps are a sudden painful involuntary contraction of a muscle or muscle group, the involved muscles becoming transiently locked in a violent spasm of uncoordinated muscular contraction. Cramps need to be distinguished from tetany due to low plasma concentrations of ionised calcium, and also from the restless legs syndrome in which sleep is disturbed by unpleasant sensations in the legs, relieved by movement [2].

Joekes [3] has proposed a practical four-point clinical classification for cramps (Table 1). This emphasises the need to consider underlying neurological or muscular disease and it stresses the role of drugs as precipitating factors. Rest cramp is not just a residual category but constitutes a discrete clinical syndrome mostly confined to elderly people, with cramp episodes occurring at night, usually in the early hours, and generally affecting the foot or calf muscles. Although rest cramps have not attracted much clinical enthusiasm, they are often distressing enough for the patient to be eager to discuss them. It is easy to pass them over as self-limiting and not life-threatening, but for some elderly people life is severely disrupted because of their fear of falling asleep [4].

Pathogenesis

An adequate explanation of rest cramp must account for the muscle fasciculation which is often an antecedent

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Table 1. A clinical classification of cramp (after Joekes).

1. Disease associated	Electrolyte losses Neuromuscular (eg motor neurone disease, peripheral neuropathy)
2. Drug induced	
3. Effort cramp	Swimming etc 'Musician's cramp'
4. Rest cramp	Pregnancy Post-exercise Elderly (nocturnal)

feature, and for the runs of high-frequency discharges ranging from 200 to 300 impulses per second on electromyography recordings [5]. A satisfactory explanation for rest cramp must also encompass the clinical observations that they are nocturnal and predominantly affect distal leg muscles. Moreover, it is a syndrome of the elderly and this implies that age-related changes in key tissues may be relevant in understanding the mechanism(s) for rest cramp.

Neuromuscular age-related changes

Muscle mass and muscle strength decrease with age [6,7]. Few studies have attempted to describe the histopathology or neurophysiology of age-related muscle changes. However, a consistent finding has been evidence of substantial denervation and type II fibre atrophy [8-10], most marked in the distal musculature [9]. Changes are also seen in the peripheral nerves with fall-out of Schwann cells and patchy loss or thinning of myelin sheath [11], compounded by an age-related reduction in density of myelinated peripheral fibres which preferentially affects the larger fibres [12]. This disproportionate loss of larger fibres seems to involve the longer anterior tibial and sural nerves but not the superficial radial nerve [13]. This results in a slowing of nerve conduction [14].

Histopathologically the various ageing changes are consistently seen to be greatest in the longest peripheral nerves and most distal muscles. This finding may be an important component in explaining why rest cramps predominantly affect the foot and calf muscles. A further important age-related change is the fall-out of anterior horn cells which occurs with increasing age [15]. This has

a parallel with motor neurone disease in which fasciculation and nocturnal cramps are common [16].

Vascular age-related changes

Reports of rest cramp have generally emphasised that the peripheral circulation is unimpaired, implying that it need not be an important factor in the mechanism for cramp production [1,4,17]. However, some degree of atherosclerosis is universal in the elderly [18] and clinical signs of impaired circulation in the leg may be misleading; arterial pressure and flow only fall seriously when the cross-section area of a vessel is reduced by 75% [19]. Thus moderate arterial disease may be difficult to quantify by clinical factors alone. Physiological studies of lower limb blood flow using plethysmography or pulse-analysis by doppler have not yet been reported for patients with rest cramp.

It has been suggested that limb circulation may be disturbed in patients with rest cramp due to previous damage to the deep venous system, leading to venous hypertension. The main evidence for this is that nocturnal cramps may be alleviated by elevating the foot of the bed, so improving leg drainage [20]. It is likely, however, that venous hypertension is an aggravating factor rather than a prime mechanism.

Drugs as factors

Muscle pain, stiffness and cramps represent one of several drug-related neuromuscular syndromes [21,22]. Various drugs have been incriminated (Table 2) and knowledge of these is important, for their selective withdrawal offers an opportunity to modify cramp symptoms. In addition, these agents provide potential insight into mechanisms for cramp production. Drug-induced hypokalaemia is one such mechanism common to diuretics, carbenoxalone, purgatives, and amphotericin B. Chronic hypokalaemia most often produces generalised painless weakness of muscles, but muscle aches and cramps may also be a feature and have been attributed to changes in the resting potential and excitability of the muscle cell membrane [21,22]. Enhanced neuromuscular excitability has also been the mechanism suggested for rest cramps due to nifedipine [23], metolazone [24], and the β_2 adrenergic antagonist salbutamol, particularly when it is given orally [25].

Suggested mechanism

Consideration of the factors discussed above allows a tentative unifying explanation for rest cramps to be advanced. It is evident that neuromuscular control is most impaired by ageing changes in the most distal part of the peripheral nervous system. This would explain the predisposition of rest cramp to affect foot and calf muscles. The susceptibility of the distal leg musculature to involuntary contraction is aggravated by certain drug actions and by impaired peripheral circulation. In this latter respect it is relevant to consider the haemodynamic changes occurring during sleep. Increasing depth of sleep

Table 2. Drugs associated with cramp.

Diuretics	}	Due to hypokalaemia
Purgatives		
Liquorice		
Carbenoxalone		
Amphotericin B		
Clofibrate		
Danazol		
Cimetidine		
Metolazone		
Bumetanide		
Lithium		
Labetalol		
Cytotoxics		
β_2 adrenergic agonists		

is accompanied by decreasing blood pressure [26] and cardiac output [27,28], factors that might be expected further to impair the peripheral circulation. Thus, ageing changes and the haemodynamic consequences of sleep (with or without additional drug factors) summate to produce spontaneous tetanic activity resulting in an episode of cramp (Fig. 1).

This model, although consistent with the available facts, is necessarily speculative and requires evaluation. It would, for instance, be of interest to determine if a fall-off in leg circulation preceded an episode of rest cramp. Also, it would be possible to test the prediction that drugs which improve peripheral circulation should be an effective treatment for the disorder.

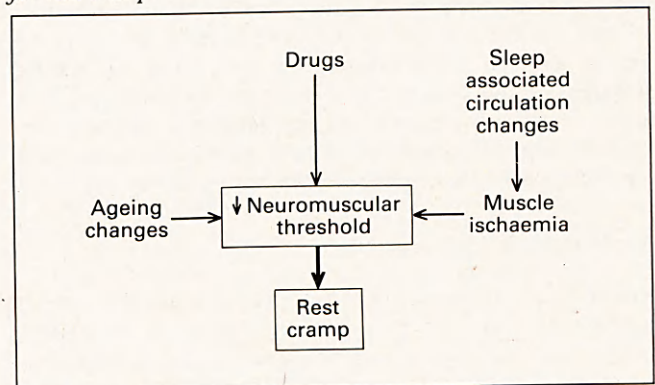
Management

Physical treatment: preventative

Simple elevation of the foot of the bed may be effective in those patients with signs of venous insufficiency in the legs [20,29]. A rather more bizarre but enthusiastically proposed method is to place a magnet between the mattress and lower sheet. The first sign of cramp would immediately disappear if the affected leg was moved over the magnet [30].

Another simple preventative measure is based on stretching the calf muscles [31]. Patients are instructed to

Fig. 1. Suggested relationship between the contributing factors for rest cramp.



stand with their shoes off, face a wall 2-3 feet away and then lean forward, using the hands and arms to regulate their forward tilt and keeping the heels in contact with the floor, until a moderately intense, but not painful, pulling sensation develops in their calf muscles. The stretching is maintained for 10 seconds and repeated after 5 seconds relaxation. The whole sequence is repeated up to three times daily. The ages of the 44 subjects described in this report were not given, but it was inferred that the cramps being treated were post-exercise related. In this situation the underlying muscle pathology is complex [32]. However, it is an attractive method which deserves further study.

Physical treatment: alleviation

Two complementary methods to terminate an episode of cramp have been proposed on the basis of the neurophysiological principle of reciprocal inhibition [33]. The methods involve either passive stretching of the contracting muscle or active contraction of the antagonist muscle. The basis for the passive method is that an attack of calf cramp may be abolished by standing on the foot and bringing the heel down hard to the ground [34]. The continued strong stretching of the calf muscle during this manoeuvre induces the anti-stretch reflex brought about by efferents from the Golgi organs in the tendon mediating a spinal cord inhibitory reflex designed to prevent rupture of contracting muscles. The active method involves voluntary contraction of the opposing muscle group, for example ankle dorsiflexion during spasm of the calf muscle. In this situation the active contraction brings about relaxation of the cramping muscle by an inhibitory spinal cord reflex.

Drug treatments

Quinine sulphate is the recommended drug treatment for rest cramp [35]. It was initially used as a successful treatment for myotonia [36] and was subsequently found to be effective in open studies of rest cramp [1,4]. Quinine increases the threshold of stimulation of the motor end plate, and also prolongs the refractory period of the muscle, resulting in a diminished response to tetanic stimulation.

Quinine has been shown in a controlled study to be an effective agent for reducing the frequency of muscle cramps in patients on haemodialysis [37]. The cause of cramps in this situation is complicated, and suggested factors are blood volume contraction [38], dialysis related sodium deficiency [39], and impaired tissue oxygen delivery due to reduced red blood cell 2,3-DPG concentrations or post-dialysis alkalosis [40]. In dialysis related cramps, therefore, quinine acts at the final common pathway for cramp production, the muscle membrane, rather than influencing putative primary causative mechanisms. This may be an analogous situation with rest cramp in elderly people. Here again there is trial evidence for effectiveness of this agent. In 1983 Jones *et al.* studied 9 elderly people with nocturnal cramps treated with quinine in a double-blind crossover study [17]. Quinine reduced the number

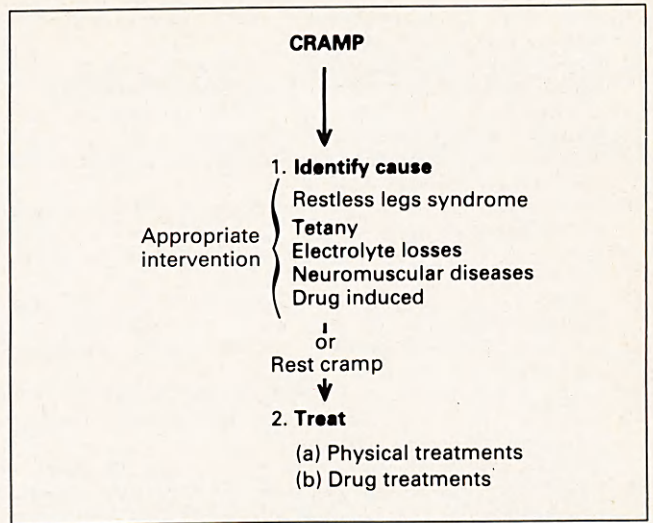


Fig. 2. An approach to the evaluation and management of rest cramps in the elderly.

and severity of cramps when taken before retiring to bed, and probably also decreased the duration of the cramp episode.

Quinine seems to be well tolerated in the low doses used for night cramps, and it has been suggested that the main reason for finding an alternative treatment is the risk of accidental or non-accidental overdose [41]. In overdose it is a highly toxic compound, producing cardiac arrhythmias and blindness (often permanent) [42].

Several other drugs have been cited as potential treatments for cramps: diazepam, dantrolene, phenytoin, procainamide, and diphenhydramine. However, there is no evidence that these agents are effective.

Conclusion

Rest cramps have been almost entirely ignored in general and geriatric medicine texts. Yet much remains to be learnt about this apparently simple but distressing syndrome in elderly people. It is important that cramps are not overlooked as a presenting feature of neuromuscular disease and that potentially aggravating drugs are considered (Fig. 2). Alleviation has mostly centred around treatments with drugs, especially quinine, but simple physical methods to prevent and abolish attacks deserve more detailed evaluation.

Acknowledgement

We are grateful for helpful comments from Dr J. A. Twomey, consultant neurophysiologist, in preparing this paper.

References

- Gootnick, A. (1943) *Archives of Internal Medicine*, **71**, 555-62.
- Clough, C. (1987) *British Medical Journal*, **294**, 262-3.
- Joekes, A. M. (1982) *Journal of the Royal Society of Medicine*, **75**, 546-9.

4. Moss, H. K. and Herrmann, L. G. (1940) *Journal of the American Medical Association*, **115**, 1358-9.
5. Norris, F. H., Gasterger, E. L. and Chatfield, P. O. (1957) *Electroencephalography and Clinical Neurophysiology*, **9**, 139.
6. MacLennan, W. J., Hall, M. R. P., Timothy, J. I. and Robinson, M. (1980) *Age and Ageing*, **9**, 188-92.
7. Aniansson, A., Grimby, G., Hedberg, M. and Krotkiecki, M. (1981) *Clinical Physiology*, **1**, 73-86.
8. Tomlinson, B. E., Walton, J. N. and Rebeiz, I. J. (1969) *Journal of the Neurological Sciences*, **9**, 321-46.
9. Tomonaga, M. (1977) *Journal of the American Geriatric Society*, **25**, 125-31.
10. Grimby, G., Denneskiold, B., Hrid, K. and Sattin, B. (1982) *Acta Physiologica Scandinavica*, **115**, 125-34.
11. Arnold, N. and Harriman, D. G. F. (1970) *Journal of Neurosurgery and Psychiatry*, **33**, 55-61.
12. Jacobs, J. M. and Love, S. (1985) *Brain*, **108**, 897-924.
13. O'Sullivan, D. J. and Swallow, M. (1968) *Journal of Neurology, Neurosurgery and Psychiatry*, **31**, 464-70.
14. Dorfman, L. J. and Bosley, T. M. (1979) *Neurology*, **29**, 38-44.
15. Campbell, M. J., McComas, A. J. and Petito, F. (1973) *Journal of Neurology, Neurosurgery and Psychiatry*, **36**, 174-82.
16. Newrick, P. G. and Langton-Hewer, R. (1984) *British Medical Journal*, **289**, 539-42.
17. Jones, K. and Castleden, C. M. (1983) *Age and Ageing*, **12**, 155-8.
18. Stout, R. W. (1987) *Age and Ageing*, **16**, 65-73.
19. Sumner, D. S. (1977) In *Vascular surgery* (ed. R. B. Rutherford), p25. Philadelphia: Saunders.
20. Rivlin, S. (1973) *Lancet*, **i**, 203.
21. Morgan-Hughes, J. A. (1979) *British Journal of Hospital Medicine*, **22**, 361-5.
22. Lane, R. J. M. and Mastaglia, F. L. (1978) *Lancet*, **ii**, 562-5.
23. Keidar, S., Binenboim, C. and Palant, A. (1982) *British Medical Journal*, **285**, 1241-2.
24. Fitzgerald, M. X. and Brennan, N. J. (1976) *British Medical Journal*, **i**, 1381-2.
25. Palmer, K. N. V. (1968) *British Medical Journal*, **ii**, 833.
26. Scharf, S. M. (1984) In *Sleep and breathing* (ed. N. A. Saunders and C. E. Sullivan), pp221-5. New York: Dekker.
27. Miller, J. C. and Helander, M. (1979) *Aviation, Space and Environmental Medicine*, November, 1139-44.
28. Parmeggiani, P. L. (1985) *Annals of Clinical Research*, **17**, 185-9.
29. Weller, M. (1973) *Lancet*, **i**, 203.
30. Reid, H. E. (1972) *Lancet*, **ii**, 1312.
31. Daniel, N. W. (1979) *New England Journal of Medicine*, **30**, 216.
32. Editorial (1987) *Lancet*, **ii**, 1123.
33. Fowler, A. W. (1973) *Lancet*, **i**, 99.
34. Graham, G. (1965) *Lancet*, **ii**, 537.
35. Anon, (1982) *Drugs and Therapeutics Bulletin*, **20**, 97-8.
36. Kennedy, F. and Wolf, A. (1937) *Archives of Neurology and Psychiatry*, **37**, 68-74.
37. Kaji, D. M., Nottage, W. G., Ackad, A. and Stein, R. M. (1976) *Lancet*, **i**, 66-7.
38. Stewart, W. K., Fleming, L. W. and Manuel, M. A. (1972) *Lancet*, **i**, 1049.
39. Catto, G. R. D., Smith, F. W. and MacLeod, M. (1973) *British Medical Journal*, **iii**, 389.
40. Chillar, R. R. and Desforges, J. F. (1972) *Lancet*, **ii**, 285.
41. Henry, J. (1985) *British Medical Journal*, **291**, 3.
42. Bolland, M. E., Brenand Roper, S. M. and Henry, J. A. (1985) *Lancet*, **i**, 384-5.