Velocity dependent passive muscle stiffness

We refer to the interesting study by Lee et al concerning quantification of velocity dependent properties of the elbow flexors in patients with spasticity and rigidity.1 Their main finding was a velocity dependent increase in reactive torque in both groups, although this was only related to muscle length in subjects with spasticity.

However, the authors’ conclusion that their observations reflect stretch reflex hyperexcitability under lying spasticity is difficult to understand, as is their assumption that the contribution of passive mechanical change, if any, should be minor and uniform during stretch. The investigators used online monitoring of muscle activity in biceps and triceps to ensure that the limb was relaxed before and during the test. The effect of a voluntary contraction or an “unrelaxed limb” on baseline torques derived from slow stretches (fig 3, p 601) clearly demonstrates the distortion of the torque angle curves that would have been evident if a reflex contraction of the biceps muscle had resulted from the faster stretches.

Thilmann et al have examined biceps stretch reflex responses in patients with spasticity secondary to stroke.2 Stretches of similar magnitude to those used by Lee et al invoked bursts of biceps activity with shorter latencies and longer duration than those in age matched controls. Lee et al have avoided inducing activity in the biceps muscle during elbow extension.3 Therefore their findings are consistent with passive muscle elements, not stretch reflex responses, being responsible for the observed velocity dependent increase in reactive torque. This phenomenon has been reported previously in the calf muscles of normal subjects and of individuals with spasticity and spinonal cord injury.4

The data reported by Lee et al have demonstrated a velocity dependent stretch related behaviour of the elbow flexor muscles which is viscoelastic. The velocity dependent increase in reactive torque in both spastic and rigid muscles was significantly greater than in normal muscles, with the relation between reactive torque and limb position (muscle length) in spasticity differing from rigidity or normal tone. The most likely source of this velocity and length dependent resistance is alteration of the contractile component of the muscle, although connective tissue and noncontractile proteins within the sarcomeric cytoskeletons also contribute to passive muscle tension. An increase in the number of weakly attached actin–myosin crossbridges in relaxed muscle, and/or a slower than normal detachment rate during passive stretch, may contribute to hyperton, especially in chronic cases.5 As crossbridge detachment occurs at a steady rate during stretch, such mechanical adaptations could account for some of the velocity dependent resistance observed in this study.

The importance of distinguishing between reflex and non-reflex sources of resistance to stretch lies in the need to target treatment interventions appropriately. An overemphasis on the contribution of stretch reflex excitability to motor disability in patients with spasticity may lead to an inappropriate treatment focus.

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Transoesophageal echocardiography: which stroke patients benefit most from this investigation?

In a recent article in this journal, Strandberg et al reported 441 patients who were investigated by transoesophageal echocardiography (TOE) after ischaemic stroke or transient ischaemic attacks.1 The authors looked for cardiac sources of embolism. They found that in patients who were in sinus rhythm and without any cardiac disease, 8% have been given anticoagulation drugs on the basis of TOE data.

Although we agree with their conclusions that TOE should be used in patients with stroke, we have, however, concerns about the following points:

- No definition is given of what is meant by “anticoagulation drugs.” Did the authors mean only oral anticoagulation with acenocoumarol or phenprocoumon, or did they also mean other antithrombotic drugs like heparin, acetylsalicylic acid, or clopidogrel?
- What contraindications to anticoagulant drugs did they consider?
- How long after the stroke or transient ischaemic attack was anticoagulant treatment started?
- The decision about anticoagulation was based on “clinical assessment.” They report that “if a minor risk factor for a cardiac source of embolism was detected, anticoagulation treatment was started after clinical consideration of the probability of cardiac embolism and treatment hazards,” but the decision criteria for “assessment” and “considerations” are not reported, and thus these decisions are not reproducible. Did the number of risk factors influence the decision for anticoagulation?
- It would be especially interesting to know which patients in whom TOE had detected major and minor risk factors did not receive anticoagulation drugs. Did the type of risk factor or the TOE findings influence the decision about anticoagulation?
- The detailed TOE findings of the 8% of patients in sinus rhythm in whom TOE prompted anticoagulant treatment are not given, thus making the impact of TOE in therapeutic decisions difficult to determine.

We suspect that a patent foramen ovale was a frequent finding in this group of patients and are interested in the authors’ diagnostic and therapeutic approach in these patients with suspected paradoxical embolism. Our special interest in stroke patients with patent foramen ovale results not only from own experience but also from the recently published large prospective study in patients with patent foramen ovale.1 This has not shown any differences in the recurrence rate of stroke in patients with patent foramen ovale between those treated with warfarin and those treated with acetylsalicylic acid.

In routine clinical practice, echocardiography is not only relevant to decisions about anticoagulant treatment in stroke patients, but also to other forms of medical treatment—such as ACE inhibitors or β blockers. If in left ventricular dysfunction accompanying dilated cardiomyopathy or left ventricular aneurysm, or antibiotic treatment in infective endocarditis. Furthermore, echocardiographic findings may prompt cardiac surgery, for example in patients with left atrial myxoma, aortic aneurysm, mitral or aortic valvotomy, or infective endocarditis. Thus some of the major and minor risk factors for cardiac embolism are disorders which might require treatment other than anticoagulant drugs. The authors do not indicate how often such therapeutic decisions, “beyond” anticoagulation, were prompted by echocardiographic findings.

In conclusion, we agree that TOE is a useful tool in stroke patients to clarify diagnostic and therapeutic issues. The important question, however, as to which stroke patients should undergo TOE remains unresolved.

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Author’s reply

We would like to thank Drs Stöllberger and Finsterer for their interest and noteworthy opinions concerning our paper.

By “anticoagulation drug,” we meant only oral anticoagulation with warfarin. The contraindications to this treatment were allergy to warfarin, a bleeding tendency, hepatic or renal insufficiency, serious hypertension, endocarditis, alcohol abuse, pregnancy, and dementia.

The decision to start or continue anticoagulation after a stroke with a clear cardiac source of embolism is a difficult one and must be made after careful thought. When a major risk factor for a cardiac source of embolism was detected using transoesophageal echocardiography (TOE) and none of the contraindications mentioned above was present, the patient was given anticoagulant drugs. In cases with a minor risk factor for a cardiac source of embolism the decision to start anticoagulant treatment was based on the number and type of risk factors. In the study population, anticoagulant treatment was usually started within one to three days from the event, after haemorrhagic transformation had been excluded by brain imaging.

When patients who were in sinus rhythm and had no history of cardiac disease were evaluated as an independent group, anticoagulant treatment appeared to be based on the PFO in Cryptogenic Stroke Study (PICSS) Investigators. Circulation 2002;105:2625–31.

“Sentinel” or “early warning” bleed

I am writing to take issue with Richard Davenport's prejorative objection to the term “sentinel” or “early warning” bleed in patients found subsequently to have had a subarachnoid haemorrhage. His dismissive comment that the term was “coined by an Edinburgh neurosurgeon” is particularly offensive. The neurosurgeon in question was no journeyman but in fact was Professor F John Gillingham, an internationally esteemed vascular neurosurgeon, the protege and successor of the legendary Mr Norman Dott.

In a 1993 address to the Harvey Cushing Society (predecessor of the American Association of Neurological Surgeons) Professor Gillingham reported that over half of the patients who were admitted in coma or stupor—or were otherwise severely disabled from a ruptured intracranial aneurysm—had a clear cut history of a minor haemorrhage a few days or weeks before the major episode. Admittedly this was a retrospective survey in an era that pre-dated modern diagnostic imaging modalities and a time when there was less awareness on the part of general practitioners and emergency room personnel of the importance of new onset acute headache.

Professor Gillingham’s observation was supported by Dr W M Lougheed, a highly regarded Canadian vascular neurosurgeon. My own experience, and that of many of my neurological colleagues, is consonant with the above viewpoint. Finally, I suspect that some of the 37 subjects presenting with subarachnoid haemorrhage in the prospective study alluded to may, in fact, have been experiencing a minor (“sentinel”) bleed that was correctly diagnosed rather than being overlooked.

N H Horwitz

Reference


Author’s reply

I am perplexed that Dr Horwitz should have concluded that my comments regarding “sentinel” bleeds were “offensive”. Having worked in Edinburgh for over a decade, I am well aware of Professor Gillingham’s reputation. Although I doubt many other readers will have similarly misinterpreted my words, I am happy to reassure Dr Horwitz that no disrespect was remotely intended, to Professor Gillingham or anyone else.

However, I stand by my message that the term “warning leak” should be abandoned. The prospective evidence now available, and quoted in my paper, indicates that most such “warning leaks” are probably the result of recall bias from previous retrospective studies. In some cases there may indeed have been a true subarachnoid haemorrhage that was unrecognised at the time (by either the patient or their medical attendants), although the study by Linn et al suggests these account for a minority—in which case let us be honest and call them “missed subarachnoid haemorrhage”, rather than “warning leaks”. The key educational point is that all doctors should appreciate the potential importance of a true sudden onset headache, and refer them accordingly to the appropriate unit for further investigations.

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Reference

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*J Neurol Neurosurg Psychiatry* 2003 74: 283-284
doi: 10.1136/jnnp.74.2.283-a

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