Letter to the Editor

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I was very interested to read the clinical review, Neurogenic heterotopic ossification in spinal cord injury, by van Kuijk *et al.*¹ The authors quote my two papers on the primary prevention and on the relation of movement to the causation of the condition.

The background of how I came to this conclusion will be of interest to your readers although the initial work was done nearly 40 years ago. Despite my provocative paper in 1969 my views were not refuted.

In 1965, I was appointed single-handed Consultant to the Liverpool Regional Paraplegic Centre. There were no full time physiotherapy staff. Patients did not receive as much passive movements as was considered desirable and, coming from Stoke Mandeville, I introduced a vigorous programme of physiotherapy.

My attention was first drawn to the problem by the case of Mrs J, a 20-year-old woman, sustained a complete cord lesion at D6 as the result of a car accident on 4 December 1966. She had no other injuries and was admitted to the Centre the following day. Some 10 days after injury, she complained of chest pain and the left leg was observed to be swollen and hot. A presumptive diagnosis of pulmonary embolism was made, secondary to deep vein thrombosis in the left leg. She was placed on anticoagulant therapy (phenindione) and movements of the left leg were discontinued. Movements of the right leg were continued and she made an uneventful rehabilitation. She got out of bed on 18 January 1967 but X-rays on 8 March 1967 showed slight ossification around the right hip. At no stage had she complained of any disability in the right leg and by 22 May 1967 she was standing, cooking, dressing and undressing herself and was virtually independent. She was discharged home on 23 June 1967 to look after her young children and husband. When she returned for a check-up on 27 November 1968, although there was no disability in her right hip the ossification was much more extensive.

A further patient developed calcification around his knee which I related to the use of a Stryker frame.

A detailed analysis was carried out.² With the regimen of treatment in 1963–64 (early mobilisation and few passive movements with no anticoagulant therapy) only one patient out of 42 developed heterotopic calcification. With the regimen followed during 1966, 1967 and 1968 (of vigorous passive movements), four patients out of 43 developed heterotopic ossification.

It would appear that the change in treatment comprising increased passive movements, anticoagulants and turning beds had been associated with an increased incidence of calcification. I postulated then

that there was a relation with trauma by either passive movements or from a stryker bed causing heterotopic calcification, a view suggested to me Damanski,³ the previous consultant at Liverpool.

This was contrary to the accepted thinking at the time and when I was invited to participate in the Sir Ludwig Guttmann birthday volume of Paraplegia (1969) I asked an impertinent question: could passive movements be causing the heterotopic calcifications? I hoped that my ideas would be tested by further observations and would either be disproved or confirmed.

On my return to Stoke Mandeville in 1970 the accepted treatment at that time was, if calcification formed, to try and break it down by increasing the passive movements. As a result of my researches I did not subscribe to this view and if a patient developed calcification discontinued violent passive movements just maintained the existing movement of the joint.

Despite my provocative paper in 1969 my views were not refuted.

Stimulated by the observation that some patients developed heterotopic calcification while under my care, further research was instituted between 1983 and 1989. Heterotopic calcification appeared to be more frequent in patients who were admitted late to the Centre and statistical analysis confirmed this.⁴ Heterotopic calcification occurred only in those patients where the start of passive movements was delayed until 7 or more days after injury to the spinal cord since it was known that no passive movements were given at the receiving hospital.

This was backed by the experimental work of Izumi,⁵ who showed that there was no difference in the incidence of heterotopic bone between paralysed and nonparalysed animals, under the same conditions of immobilisation and passive movement. However, it was demonstrated that when a paralysed rabbit was immobilised for an interval and then passive movements were commenced, heterotopic bone was formed, thus showing it was the movement following the period of immobilisation that caused the heterotopic bone. This experimental work provides the key to my findings.

The paralysed patient is an ideal model for this situation, which follows the rabbit model closely. If the paralysed patient is admitted immediately to a spinal injuries unit and passive movements are commenced immediately, heterotopic bone does not occur, but if he/she is paralysed and receives a period of immobilisation in the receiving hospital, then heterotopic bone will appear when passive movements commence at the spinal centre. The worst cases of heterotopic bone are seen in

patients who are treated on plaster beds or private patients who come to the spinal unit after a considerable interval.

The results suggest that the time when passive movements are begun plays a vital role in the development of heterotopic ossification. If passive movements are started from the day of injury then the joint capsule is kept supple, muscles do not shorten, and contractures do not develop in the ligaments and fascia. On the other hand, if the paralysed limbs are not moved from the very beginning, exactly the reverse occurs. In the joint capsules and muscles, ligaments and fascia shrink and adhesions develop between various soft tissue compartments. Once these changes have occurred, subsequent passive movements result in shear and tear of the soft tissue and if this is a large enough, it triggers off an abnormal process of repair. Further studies will be required to show whether muscle and joint contractures and restriction of movement are related to the future development of heterotopic ossification in spinal cord injured patients.

Wharton and Morgan⁶ advocated vigorous movement in the acute stage to reduce bone formation and to prevent future ankylosis of the joint. My findings, however, demonstrated that vigorous movement in this acute stage, in the majority of patients, aggravated the clinical picture and increased the amount of heterotopic ossification. It also diminished joint mobility in the long term and prolonged the period of rehabilitation. It has therefore been the practice in the National Spinal Injuries Centre to treat patients with heterotopic ossification with bed rest and very gentle passive movement. This is continued until the acute effects have settled.

The further analysis that has been carried out by van Kuijk *et al*¹ suggests that patients who are spastic are more likely to develop heterotopic bone formation than

those who are flaccid. This would be in keeping with the fact that a spastic patient when the lower limbs are in spasm and adducted there would be much more trauma involved in stretching the paralysed muscles than in patients who are flaccid.

This shows that very simple clinical observations can provide worthwhile answers leading to useful treatment. While biochemical and biomechanical research is vital if we are to make further progress, there is still room for clinical observations. I am delighted that after 40 years my own observations, which were against the grain, have now been accepted as mainstream treatment and are incorporated in van Kuijk *et al*'s final conclusions.

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