pighian carcinoma invading the thyroid and the right wall of the trachea. He was treated with ablation of the right thyroid lobe, tracheotomy, and three courses of chemotherapy (CCDP, 5FU) followed by radiation therapy on the larynx (70 Gy) and upper mediastinum (45 Gy) with a 20 Gy dose on the sub-glottic area. The calculated total radiation dose received on the spinal cord was 48 Gy at the C6 level (maximal dose at 2 cm high). Other parts of the spinal cord received 42 Gy at the cervical region (including C7) and 41 Gy for T1. There was no development of the mass on regular CT scans. The only treatment was thyroid hormones after surgery.

Three years after finishing radiation therapy and after transient parasthesia of the hands and a Lhermitte sign (lasting less than two weeks), the patient developed progressive wasting and weakness of both hands and forearm extensors. He had no symptoms in the legs nor sphincter disturbance. We found no sensory abnormality (except a questionable reduction of tactile perception on the inner side of the right hand) nor autonomic dysfunction, and tendon reflexes were present except supinator and triceps jerks on both sides. Neurological examination elsewhere was unremarkable.

Electromyographic examination showed fibrillation, giant potentials and some fasciculations in right and left hand muscles, and reduced recruitment in forearm extensors. The more proximal muscles of the arms and muscles of the face and legs were normal. Motor conduction velocities, distal motor latencies, and amplitude of sensory potentials were normal in all limbs. Values of somatosensory evoked potentials were in the normal range after median and posterior tibial stimulation. Haematological, biochemical, and hepatic routine tests, thyroid hormones, lipidogram, syphilitic serology, viral serologies, serum lead and urine lead concentrations were normal. Cerebrospinal fluid was normal for cellular count, biochemistry and protein electrophoresis.

Cervical MRI was performed five months after the first neurological symptoms on a 1,5 Tesla superconducting magnet (Magnetom Siemens). Two excitations and a display matrix of 256 × 256 were used; the section thickness was 4 mm. Sagittal and axial studies showed a cystic lesion with well defined borders and a signal isointense with respect to CSF on T1 weighted images (TR = 500 ms, TE = 15 ms) and T2 weighted images (FIS P a = 10°, TR = 100 ms, TE = 27 ms) within the spinal cord from C4 to C6 and a smaller cavity behind the body of the atlas (figure). There was no enhancement of this image after IV injection of gadolinium and the cerebellar tonsils were not in an ectopic position.[2] The fatty replacement of spinal bone marrow due to radiation therapy accounted for the high signal intensity of the cervical spine on the same level.[3] The thoracic MRI was normal. There was no change in neurological condition after eight months.

This case fulfils the strict criteria established for an accurate diagnosis of radiation myelopathy[4]: spinal cord included in the area of radiation; principal neurological manifestations compatible with the irradiated portion of the cord; other possible aetiologies such as compression due to metastases excluded by neurological investigations; and a latent period of at least nine months before the development of symptoms. Radiation myelopathies may be divided into five main

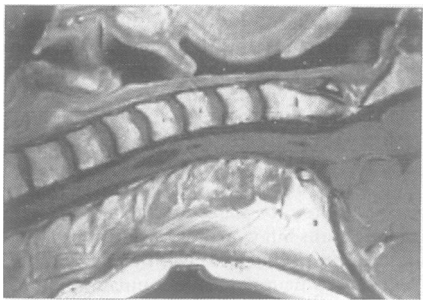

Figure Sagittal TI weighted SE (500 ms/15 ms) image showing cystic hypointense cavity affecting spinal cord from C4 to C6 without ectopic tonsils. Note fatty replacement of cervical spine bone marrow from C2 to C6, responsible for hypersignal on TI weighted images.

types:[1 4] transient myelopathy (the more common and generally marked only by a Lhermitte sign); chronic progressive myelopathy; arrested radiation myelopathy; selective anterior horn cell injury or amyotrophy; and disseminated demyelination of the central nervous system. Our patient was in the fourth category as there was weakness and amyotrophy without significant sensory change, sphincter disturbance, or clinical involvement of corticospinal tract. There was electrophysiological evidence of an involvement of the anterior horn: giant potentials and normal motor and sensory conduction velocities. MRI data agree with this location. A coincidence, however, cannot be excluded but the only alternative diagnosis would be idiopathic syringomyelia for which the clinical picture, without a considerable sensory problem, the rapid progression of symptoms, the absence of Chiari or kyphoscoliosis would be atypical. There was no evidence for a syrinx secondary to an intraspinal tumour (thoracic MRI normal). Furthermore, before the wasting of the hands, a Lhermitte sign occurred, which is a common symptom of radiation myelopathies.

Nevertheless, to our knowledge such a clinical presentation has not previously been described. All reports of delayed lower motor neuron syndrome after radiation concern the legs, generally after irradiation for lymphomas or testicular malignant tumours.[7-11] These motor neuron syndromes usually present as a progressive weakness of the legs without sensory or sphincter disturbance. Development stops after some months or years and so far the prognosis is better than in other forms of myelopathy. Signs suggestive of a cervical anterior horn involvement (muscle wasting, fasciculations, tendon reflex loss) have been mentioned in some cases[1 5] but they were associated with corticospinal and long sensory tract lesions (mainly Brown-Sequard syndrome).

The pathogenesis of radiation myelopathies remains controversial, but vascular changes are almost constant and are considered as the primary damage rather than neuronal or immunological disorders. As the grey matter is classically considered less vulnerable to radiation than white matter, the anterior horn is presumed to be less or not involved, although in several anatomical cases of radiation myelopathies an extension of lesions towards the anterior horns has been reported.[5 6] There are no well documented anatomical reports of motor neuron syndromes after radiation and some controversy exists as to the site of lesions (anterior horn, motor anterior roots, or plexus), but clinical and electrophysiological data from most cases suggest an involvement of the anterior horns.[7] We know of only one report of MRI data from a patient with motor neuron syndrome of the legs and the thoracolumbar spinal cord images were normal.[8] In a case reported by Lechevalier *et al* a patient had Brown-Sequard syndrome after radiation with proximal wasting and weakness of the left arm. Histological examination of the cervical spinal cord showed a centrospinal necrosis with complete neuronal loss of the left anterior horn. Perhaps in our case the spinal cavity on MRI was secondary to necrosis involving the cervical anterior horns.

D MALAPERT
P BRUGIERES
JD DEGOS
Departement de Neurosciences, Hopital Henri Mondor, Créteil-94010 France

Correspondence to: Dr Malapert

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The incidence of muscle cramp

In true muscle cramp there is sudden involuntary and painful shortening of muscle and visible or palpable knotting of muscle often with abnormal posture of the affected joint which is relieved by stretching or massage.[1] Epidemiological data on muscle cramp are sparse and based upon empiricism. Wilder in 1940 found that 19% of young soldiers, 33% of workers in a silk mill, and 67% of medical staff questioned had experienced muscle cramp. The incidence in a group of internal, neurological, and psychiatric patients was 40%.[2] In a study in 1947 of 100 men and 100 women aged 15–80 years, Hall found that 56% had suffered from cramp; of those aged 50 years or more, 70% had suffered.[3] Of 121 college students questioned in 1956, Norris found that 115 had experienced spontaneous muscle cramp at least once, 18

being woken from sleep more than twice a month, usually by cramp in the calf muscles.[4] We studied the incidence of muscle cramp in adults in The Netherlands.

Telephone ownership in The Netherlands is universal. After a pilot study to estimate the incidence of muscle cramp and indicate the extent of the sample, 780 numbers were randomly selected from the telephone directory by a computer (95% confidence interval 17·5% to 22·5% for an estimated frequency of 20%). These numbers were dialled by a single interviewer. Numbers answered by people under 18 years or not Dutch were disregarded and a new randomly drawn telephone number was dialled. Telephone numbers of businesses, shops, institutions, foundations, corporations, societies, and unions were excluded and new numbers were dialled. The interviewer continued to call numbers at different times of the day and week until all could be contacted. When co-operation was refused by a subject, another number was dialled. No record was kept of the refusal rate but we estimated it to be less than 5%. Non-respondents may have been selectively less likely to have symptoms. Thus minimum estimates of incidence were calculated. Interviews were made on weekdays from 0800 hours until 2100 hours, from 12 November until 5 December 1988. In total 240 men and 540 women were interviewed (median age 44 years). Sampling was inherently biased because of an under-representation of the working population, who are mostly not at home during office hours. It probably over-represents women, non-working, ill, and disabled people. Data were therefore corrected for the sex and age distribution of The Netherlands in 1988, though this would not correct for illness and occupation.

Subjects who agreed to fill out a questionnaire and admitted suffering from cramp more often than 20 times in 1988 received a form. An equal number of subjects who agreed to fill out a questionnaire but did not suffer from cramp received a questionnaire as an internal standard. A random sample of subjects who agreed to fill out a questionnaire but suffered from muscle cramp less often than 20 times a year also received a form. The overall response rate for the postal survey was 82% (90 men, 245 women). Seventeen questionnaires were filled out by persons other than the subjects interviewed by telephone, and seven were not filled out correctly. A total of 311 questionnaires were analysed. In the telephone interviews and questionnaire, cramp was defined as a sudden involuntary painful muscle contraction accompanied by hardening of muscle but lasting no longer than 10 minutes. The telephone interviewer excluded other types of muscle or limb pain by repeatedly emphasising this definition. Writer's cramp and other occupational cramps probably represent task-specific dystonias and were excluded from the study.

The incidence calculated from telephone interviews and questionnaires was adjusted for age and sex distribution (I_{int}). Sensitivity and specificity of data collection by telephone interview were 86% and 71% respectively, compared with data collection from the questionnaires. The real incidence of muscle cramp in the general population (I_{real}) was computed from equations, $I_{int} = I_{real} \times \text{sensitivity} + (1 - I_{real}) \times (1 - \text{specificity})$ $I_{real} = (I_{int} - 0{\cdot}29)/0{\cdot}57$.

The table shows the real incidence of muscle cramp calculated from the telephone interview study.

Reported incidence of at least one muscle cramp in Dutch adults in 1988

	Men	*Women*
18–39 years	26%	48%
40–59 years	31%	32%
⩾60 years	26%	52%
Total	28%	44%

Muscle cramp is difficult to diagnose in a community survey. The criteria we used, however, were fairly stringent.[1] The proportion of the adult population of The Netherlands that had at least a single muscle cramp in 1988 was estimated at 36%. This high incidence agrees with empirical data from previous studies.[3] Occurrence of cramp may thus be considered as nothing unusual in otherwise healthy adults. Age adjusted incidence ratios showed a 3:2 female preponderance, which was not accounted for by pregnancy associated muscle cramp, as women beyond childbearing age were particularly affected (table). Age had little effect on the proportion of the population suffering. Nevertheless, older people suffering from cramp had attacks more often than younger people (data not shown). At any age, muscles in calves (84%) and feet (39%) were most often affected. Men showed a slight tendency for cramp in calves (88% *v* 78%) and arms and hands (25% *v* 11%) whereas women more often suffered from cramp in feet and toes (53% *v* 25%). Wearing high heeled shoes may in part account for this. Pregnancy was the most important risk factor predisposing to muscle cramp (odds ratio 6·3; 95% confidence interval 1·0 to 38·6). Musculoskeletal pain and stiffness also correlated with muscle cramp (2·8; 1·1 to 7·2). Irritation from diseased, overloaded, or overburdened joints, tendons, or muscles may provoke cramp.[5] Subjects with generalised muscle twitching and fasciculations are prone to developing cramp.[6] In our survey, however, the correlation between fasciculations and muscle cramp was insignificant (1·6; 0·9 to 2·8).

PHP JANSEN
EMG JOOSTEN
Institute of Neurology, Division of Neuromuscular Diseases, Catholic University of Nijmegen, The Netherlands
JAAM VAN DIJCK
ALM VERBEEK
Department of Social Medicine, Epidermiology Unit, Catholic University of Nijmegen, The Netherlands
FW DURIAN
ACF Farma, Medical Research Department, Maarssen, The Netherlands

Correspondence to: Dr Joosten, Institute of Neurology, St Radboud University Hospital, PO Box 9101, 6500 HB Nijmegen, The Netherlands.

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BOOK REVIEWS

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Yearbook of Neurology and Neurosurgery 1991. Edited by RD CURRIER AND RM CROMWELL. (Pp 407; Price: £41.00.) 1991. London: Wolfe Publishing Ltd. ISBN 0 8151 2466 X.

This well known annual round-up of neuroscience topics has continued since 1902. An invaluable collection, it retains its customary format, but the style of Currier's comments shows even more informality than before, containing pertinent remarks and many personal chatty asides which considerably enliven the text.

Any attempt to capsulise the literature in Neurology and Neurosurgery can be seen as tempting a neurological Armageddon. There are now available many publications of abstracts, advances and trends; the individual reader's preference rests with his assessment of the articles selected, the adequacy of the abstract, and the presentation. Once again the Year Book seems to have forgotten nothing of significance. The skill required to produce such intelligible, readable abstracts and commentaries is self-evident.

The preface records the sad passing of Russell DeJong on August 21, 1990:

> "In northern Michigan there was a most unusually beautiful shimmering display of northern lights . . . it was not hard to believe, as probably the Indians did, . . . that there was a message intended, or perhaps a signal of some happening, such as the death of a great chief."

With such touches, it is irresistible.

JMS PEARCE

Neurobehavioural Aspects of Cerebrovascular Disease. Edited by R A BORNSTEIN AND G BROWN. (Pp 367; Price £40.00.) Oxford, Oxford University Press, 1991. ISBN 0 19 505431 8.

As the editors of this multi-author text rightly point out much of the early work on cerebral localisation was based on study of patients with cerebrovascular lesions. The title of this volume might lead one to expect an update on this but, in fact, it covers a much wider field. There is a good section on cerebrovascular pathophysiology, blood flow, metabolism and imaging. There follows an overview of the clinical situation including epidemiology, the relationship with cardiac disease and a summary review of current management.