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Anatomical and functional perspectives of the cervical spine: Part II: the "hypermobile" cervical spine †

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This is the second of a three part series describing the clinical issues surrounding the radiographic assessment of the working definition of hypermobility. Described are the two major forms of hypermobility, namely generalized and segmental. Each form is reviewed and supported with available documentation. A case report is presented which highlights the clinical aspects of segmented hypermobility. (JCCA 1989; 33(4): 177–183)

KEY WORDS: cervical spine, hypermobility, chiropractic, manipulation.

Voici le deuxième d'une série de trois articles décrivant les questions cliniques qui entourent l'évaluation radiographique de la définition de travail d'hypermobilité. On y décrit les deux formes les plus importantes d'hypermobilité, c'est-à-dire la généralisée et la segmentaire. Chaque forme est étudiée et appuyée de la documentation disponible. On y présente un rapport de cas, lequel met en lumière les aspects cliniques de l'hypermobilité segmentaire. (JCCA 1989; 33(4): 177–183)

MOTS CLÉS: colonne cervicale, hypermobilité, chiropratique, manipulation.

Introduction

In Part I we reviewed the parameters defining stability in the cervical spine. In particular, the two components of stability – normal structure and normal function – were discussed. As well, differences in structure and function between the upper and lower cervical spine were evaluated and summarized.

In Part II, we begin exploration of aberrant, excessive intersegmental motion by defining the parameters that comprise the clinical interpretation of the term "cervical hypermobility".

With hypermobility, as with "stability", definitions are subject to the bias of authors and the effect of circumstance. At this time, two major uses of the term "hypermobile" are reported in the literature. The first can be referred to as "generalized hypermobility", and the second will be called "segmental hypermobility".

Generalized hypermobility

Generalized hypermobility has been defined as the upper extreme in the normal range of joint motion, for the human body[1,2]. When accompanied by symptoms, this extreme range has been described as "hypermobility syndrome"[1,3].

Hypermobility as a syndrome can arise either from a distinct pathology such as Marfan's and Ehlers-Danlos syndromes or naturally in the general population[1,2,4]. There is some indication that it may be genetically determined; however, this issue is controversial. Amidst this controversy, Carter and Wilkinson in 1964[5] stated their belief that persistent generalized joint laxity was a familial occurrence, and Beighton and Horan in 1970[4] were very clear in stating that a genetic cause could be determined. Beighton and Horan reviewed two cases as well as the family pedigrees involved, and suggested that these entities represented two separate autosomal dominant trait disorders. They noted that even in cases where conditions such as Marfan's did not exist, a genetic link could be found. Grahame *et al.*[6] in reviewing 87 rheumatology clinic patients (none of whom had Ehlers-Danlos syndrome type I) agreed with this observation, since their data showed cardiovascular, skin and osseous abnormalities indicative of an hereditary disorder. Jessee *et al.*[7] on the other hand, surveyed 637 healthy blood donors, and found a 5% prevalence of hypermobility, with no increase in cardiovascular abnormalities. They concluded, as did Wood[2], that it is simply one extreme of the normal range of joint motion. The discrepancies in these authors' viewpoints can probably be attributed to the differences in the respective samples studied. That is, patients from a rheumatology clinic may be more likely to suffer from the additional complications that Grahame and his colleagues associated strictly with hypermobility.

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In addition to the controversy surrounding the etiology of this syndrome, authors have also been concerned with the degree that hypermobility can vary with sex, age, ethnicity and physical activity[1,4,8]. With respect to gender differences, Carter and Wilkinson[5] pursued the theory of "temporary hormonal laxity", long thought to be due to the actions of the hormone "relaxin" in women. However, in their survey of 145 school age boys and 140 school age girls, they found no significant difference in the presence of hypermobility. Beighton, Solomon and Soskolne[9] on the other hand, in assessing 1081 members of a Tswana community in Africa from all age groups, concluded that a persistently greater amount of joint laxity was observed uniformly among women. They further concluded that flexibility tended to decrease with age[9].

Many authors have explored ethnic differences in the degree of joint motion and have come to varying conclusions. Schweitzer in 1970[8] observed that in their inter-racial study in Africa, Indians had the highest degree of joint mobility, black Africans (Hlubi and Xhosas) were next, while whites had a lesser range of motion than both of these groups. Phillip Wood, however, refuted this claim in his 1971 essay[2]. In this work, he reviewed McKusik's impression of increased clinical mobility in the negro population and concluded that there was, in fact, no basis for this claim. Wood presents a table consisting of information from 81 Caucasians and 45 Negroes where he notes the mean proportions of hypermobility in various joints in each group. Of the five joints measured, (in the fingers and elbows) only two were more mobile in the negro population. Wood critisized McKusick's lack of formal hypothesis and small sample size in drawing any conclusions at all.

Aside from the demographic variations, a common held view is that generalized hypermobility varies with physical activity. Athletes, specifically those in gymnastics and dance, train their appropriate joints from early years on, to achieve the ranges of motion necessary for the aesthetics of their art. In a study of joint mobility, Grahame and Jenkins[10] found an increase in generalized hypermobility in ballet dancers as compared to the controls, even in joints other than those typically used in training. They concluded that their sample of ballet students may have been favoured for training, in part due to this natural mobility. Klemp, Stevens and Isaacs[11], however, using individual mobility scores rather than the combined scores of Grahame and Jenkins, found that hypermobility was not more common in their sample of dancers, and that injuries were observed more frequently in ballerinas with generalized joint laxity. They attributed the increased risk of injury to postural deficits created by hyperextension of such joints as the elbows and knees.

Assessment methods

The controversy apparent in much of the literature involving generalized hypermobility, reflects in part the different criteria used to evaluate this condition. The most common criteria for assessment was initially set by Carter and Wilkinson[5] (1964) and then modified by Beighton, Solomon and Soskolne[9] in 1973. The measure is scored using a 9 point system evaluating the extensibility of various joints (Table I).

Table I

The following 9 tests are used to assess the degree of generalized hypermobility. A score of 3–4 or more, is taken to indicate hypermobility. (After Beighton *et al.*, 1973)

Test	Score
1. Passive dorsiflexion of left 5th digit beyond 90 degrees	1
2. Passive dorsiflexion of right 5th digit beyond 90 degrees	1
3. Passive apposition of left thumb to flexor aspect of forearm ..	1
4. Passive apposition of right thumb to flexor aspect of forearm	1
5. Hyperextension of left elbow beyond 10 degrees	1
6. Hyperextension of right elbow beyond 10 degrees	1
7. Hyperextension of left knee beyond 10 degrees	1
8. Hyperextension of right knee beyond 10 degrees	1
9. Flexion of trunk with knees straight, palms contact floor	1
TOTAL	9

In addition to this 9 point system, measures of global joint laxity have been derived from a "hyperextensometer[12,13] torque measurement of the metacarpophalangeal joint of the index finger. Wood, however, questioned the value of this particular measure finding both stiff and excessively mobile joints in different areas of the same finger[2]. Bird, Brodie and Wright[14], on the other hand, correlated the hypermobility results from the finger hyperextensometer, Carter and Wilkinson's scale as modified by Beighton, and a "global index" of joint laxity. They reported that the systems correlated well after trying all three on a group of 96 "non-physical education students" and a group of 54 "physical education students" who worked at a physical activity at least three times per week. These researchers calculated r (the precise correlation used was not specified in the paper) values between 0.56 and 0.81 when comparing methods. All comparisons were calculated by the authors (again with the method unspecified) to reach significance with p less than 0.001.

Clinically, the significance of increased overall mobility scores, from any mode of assessment, is relevant due to the variety of associated musculo-skeletal complaints[3,13]. For example, Beighton and his associates[9], found a consistently positive relationship between musculo-skeletal problems and an increased mobility score. Hull, in 1985[15] described three patients whose hypermobility was associated with various neck and back problems following aerobic exercise. Most commonly, authors cite recurrent dislocations of the patella and elbows, congenital dislocation of the hips[2,4,10] and the tendency for injury to various joints with and without trauma[10,16]. Hypermobility simulating chronic rheumatic diseases in children have

been cited by several authors[17,18,19]. In this regard, Biro *et al.*[20] concluded that the syndrome is not sufficiently well recognized as a source of musculo-skeletal complaints in the United States.

There has also been great interest in the association between this form of hypermobility and the possibility of an increased prevalence in osteoarthrosis. Scott, Bird and Wright[12] in 1979, attempted to evaluate the relationship between joint laxity and osteoarthrosis. They observed two groups of subjects – those with symptomatic osteoarthrosis, and age-matched controls without, and found hypermobility to be significantly more common in patients with symptomatic disease. Given the method of evaluation, it is impossible to know whether hypermobility caused the inflammatory process. The result is interesting, however, given the lack of correlation commonly found between a patient's symptom state and radiologic evidence of osteoarthritis. Wright[13] noted higher mobility scores in patients with osteoarthrosis. Unfortunately, he did not produce the figures upon which this observation was based. Lewkonia[21] found osteoarthritis only in restricted areas in patients with generalized hypermobility, and concluded that hypermobility was associated with an increased risk of degeneration in a limited number of joints.

Early osteoarthritic changes in various joints of patients with generalized hypermobility have been seen by several authors[1,4,22]. It would seem that a relationship does indeed exist, and most clinicians presume that the tendency for a person to have benign generalized hypermobility, regardless of the cause, will tend to result in additional risk of early joint symptomatology and degenerative changes. As suggested by Grahame[16], however, a real understanding of premature osteoarthrosis as it relates to joint hypermobility, will require a long-term prospective study.

Segmental hypermobility

Segmental hypermobility can be defined in two ways. In its purest sense, we can refer to it as:

> "The mobility of a given motion unit which is excessive but not so extreme as to be life-threatening or require surgery."

Clinically, and for the purpose of this paper, this definition is restricted to state:

> ***"The mobility of a given motion unit in the cervical spine which is excessive and is accompanied by local and/or peripheral symptoms, but not so extreme as to be life-threatening or require surgery."***

Very little work has been done to quantitatively evaluate the intervertebral motion of patients in this category. Authors have suggested that segmental hypermobility is a distinct category along a continuum from normal joint motion to pathological movement requiring intervention[23,24].

Appropriate guidelines of ranges of motion in x-ray analysis in flexion and extension of the cervical spine are available for "stable" and "unstable" spines, but values for this level of joint pathology remain hypothetical. Henderson and Dorman[25] provided the most recent hypothetical boundaries for these ranges of motion. Using x-rays, they assessed the extremes of motion of 16 males and 14 females ranging in age from 18 to 35 years. Using templates to study the excursion of one vertebrae over another, they listed normal and abnormal motion as a percentage of the sagittal body diameter. Further, they defined ratio ranges from "relative hypermobility" to "absolute hypermobility" in the cervical spine. The percent body diameter method of study is popular and has been used by others[26] because it avoids the difficulties of magnification and distortion encountered by most assessments.

"Absolute" hypermobility was defined by Henderson and Dorman as the figure for one standard deviation above what they found to be the mean value for the total average body diameter excursion. This was determined by averaging the excursion for all normal subjects in their sample at all of the levels of the spine (C2–C7). The authors did not take into account the differences in intersegmental motion at each level; and that it is possible that relative hypermobility may occur at a greater range of excursion for areas that are already known to be more mobile, such as C4–C5 and C5–C6. Neither did they present values expressly for flexion or extension. They did, however, acknowledge that differences exist in generalized hypermobility for men and women, and gave the range from relative hypermobility to absolute hypermobility as 0.57–0.68 in men, and 0.63–0.74 in women.

Interesting data was presented by Friedenberg and Miller in 1963[22]. The authors matched symptomatic and asymptomatic patients and evaluated the cervical spines for evidence of segmental degenerative disc disease. There was a statistically significant difference (p less than 0.005) according to a chi square test, for the level C5–C6. That is, at this level, there were significantly greater numbers of subjects in the symptomatic group who exhibited degenerative changes in the disc. This finding is intriguing, since this level is also known as an area of increased physiological mobility in the "stable" cervical spine. This paper gives some credence to the possibility that areas of increased joint motion may eventually result in symptomatic degenerative change. Farfan[23] speaks to this in the lumbar spine, stating that motor abnormalities exhibiting clinical symptoms are usually the result of repeated torsional injury and gradual loss of nuclear integrity. Thabe[27] argued that hypermobility causes stress both at the segment involved and above and below the affected joint, to the extent that they may be considered pre-arthrotic factors. Some support for Thabe's theory is supplied by an in-vitro study of movement in the normal, injured, and stabilized cervical spine. Goel *et al.*[28] found that capsular ligament injury to C5–C6 produced significantly more relative motion at C4–C5. This effect persisted even after stabilization of C5–C6.

In a retrospective survey of children with articular hypermobility simulating chronic rheumatic disease, Lewkonia and

Ansell[18] suggested that the syndrome was rarely properly diagnosed because the hypermobility in these cases was segmental rather than general. These authors recognized segmental hypermobility as a process initially causing irritation to the joint and found that a childhood history of this condition predisposed patients to osteoarthritis particularly in the thumb, knee and midcervical spine.

Thus, along the continuum from normal intersegemntal cervical joint motion to pathology and symptoms, hypermobility is the "pre-surgical" state. The spine is not "stable" and thus does not conform with the patterns of motion described by studies of normal function, but it is also not so unstable that it poses a serious threat to the patient's central and/or peripheral nervous system. It is the clinical state where the patient does not require stabilization through such means as bracing (halo apparatus, and/or traction) or invasive techniques.

In a paper put forward by Barnes and Saunders[29] the effect of cervical mobility on the natural history of cervical spondylotic myelopathy was discussed. In a retrospective analysis of 45 patients with the condition, the authors concluded that there were possible predictive factors of patients more likely to deteriorate and require surgical intervention. They noted that females with significantly more cervical mobility were more likely to develop further disabilities. Figures are not available from the paper to calculate the actual relative risk of this statement, and it was not provided by the authors.

Much more work has been done to describe this continuum in intersegmental mobility states for the lumbar spine[24] but again authors vary in their descriptions and definitions. Nachemson[30] in reviewing a submitted questionnaire to 30 surgeons in the Society for the Study of the Lumbar Spine asking for the "typical" history and clinical findings of such spinal problems as "instability", received *30 different responses*. This discrepancy in definitions for the lower back is magnified in the cervical spine, where little literature is available and hypermobility as a distinct entity is a relatively new concept. Because of this, ten clinicians at the Canadian Memorial Chiropractic College with experience ranging from 2 to 10 years were polled in an unpublished in-house study in order to determine those attributes which can generally be applied to the patient with cervical hypermobility at a segmental level. The following is a compilation of the results of this survey and the presentation of a "typical" case history of cervical intersegmental hypermobility:

History

The patient typically presents with the onset of symptoms in one of three ways:

1. he/she may have a history of repeated minor traumas (as in high-jumping) or of work related factors (such as being a painter),
2. the patient may have a history of one or more acute traumas in the neck (typically "whiplash" – a hyperflexion-hyperextension injury) but not considered serious by the patient at the time of injury, or
3. some patients recall no history of trauma at all and relate an insidious onset of unknown origin.

Generally, the disorder is slowly progressive and the symptoms build over a period of years.

The patient complains of recurrent episodes of neck pain which may include spasm. If he/she is under 30, they may have a history of recurrent, simple "wry" neck as a child. There may also be a complaint of "clicking" or "grinding" in the neck at the extremes of motion.

The pain at presentation may be unilateral or bilateral, and may occur at any level – although it is more typically related to the C5–C6 region postero-laterally. It is usually described as "dull" or "aching", although in some areas it may be "sharp"; and "focal". The patient often complains of a "tight, tired, stressed" feeling throughout the neck.

Associated symptoms may include a chronic recurrent stiffness, often associated with increased muscle tone in the neck and shoulders.

Patients may complain of headaches (usually tension-type) which may be associated with the pain at the C2–C3 region, extending to the forehead or perhaps behind the eyes. There may also be occasional arm pain of short duration and radicular or scleratogenous symptoms. The symptoms are aggravated by stress and postural fatigue, especially sustained forward flexion, and relieved by rest. The patient may state that they "crack" their own neck for relief.

Physical examination

Examination findings would be expected to indicate a decreased global range of active motion in the cervical spine and a full passive range. Kemp's and Jackson's orthopaedic tests would be expected to be positive and there may be a "catching" on extending from the flexed position, or a flexion "klunk" on bending forward. Moderate muscle hypertonicity is expected at the area of involvement. In fact, there may be signs of spasm locally and in the upper thoracic and cervical muscles. Muscular triggers are palpated in the hypertonic muscles (usually the suboccipitals, trapezius and levator scapula). Point tenderness is noted over the pillars of the involved segments. Motion palpation usually reveals decreased movement above and/or below the level of symptoms (although the area itself may appear to be moving normally). Transient decreases in motion may be noted directly at the level involved. Crepitus may be felt during palpation. Reflexes and muscle strength are normal and some paresthesias may develop later in the course of the problem.

Radiographic examination

X-rays indicate normal structure or mild spondylosis (mild degenerative changes) in the joints of Luschka, on the anterior/posterior view. The neutral-lateral may also be normal or exhibit mild spondylosis with decreased disc height at the involved level. There is usually no evidence of posterior joint arthrosis during the active stage of hypermobility. In the late stage, bony

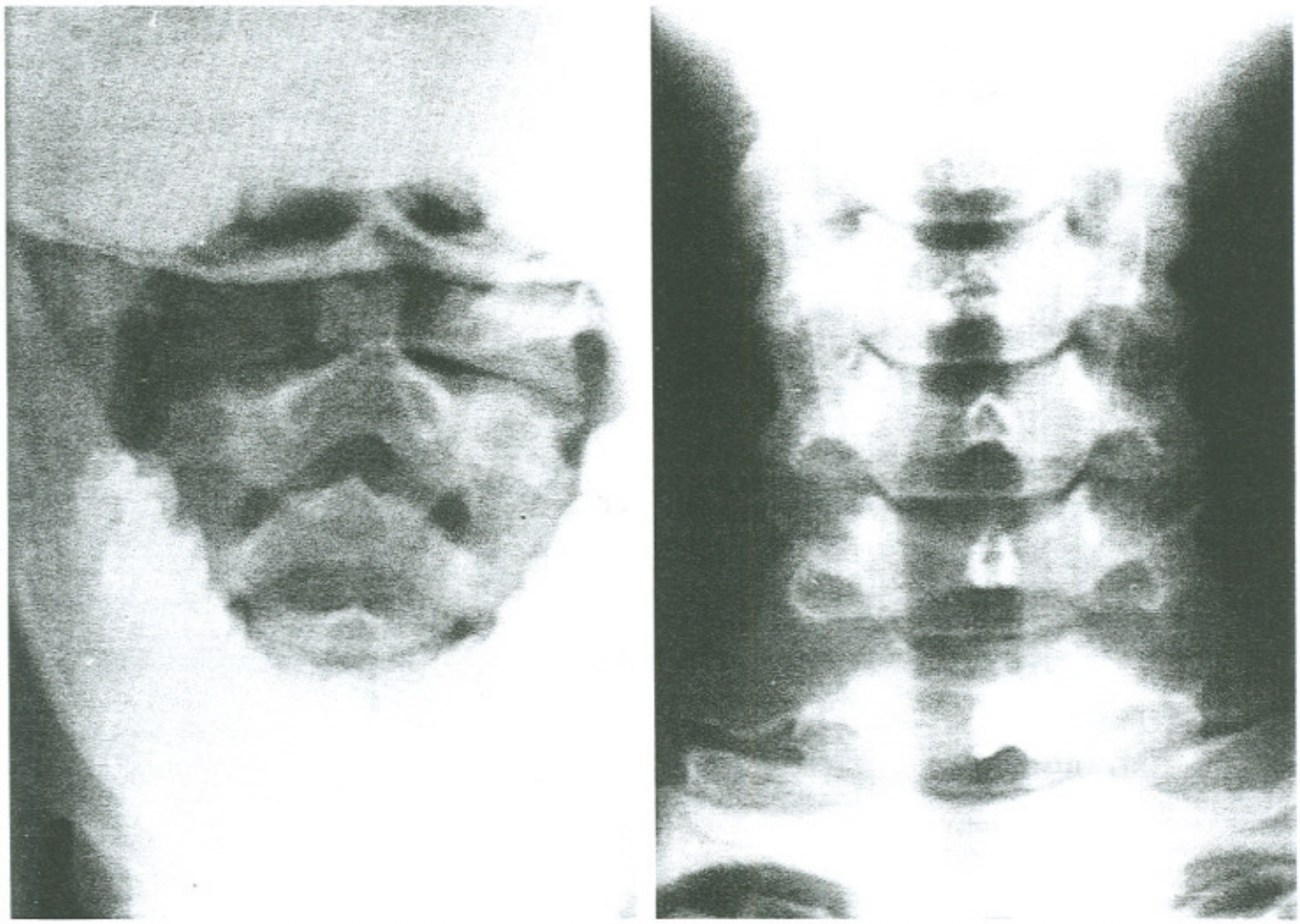

Figure 1. Antero posterior radiographs of the cervical spine of the 32-year-old female presented. Minimal degenerative changes are noted at the intervertebral spaces of C5–C6 and C7–T1.

proliferation is suggestive of impending stabilization. There may a break in George's line (the line formed by posterior vertebral bodies) and a flexion malposition may be noticed. In an acute stage, there may be a decrease or reversal of the lordosis, widening of the interspinous space and evidence of soft-tissue swelling. Flexion-extension films show increased excursion at the level involved (figure 2), and decreased movement above or below. The increased translation will be localized and in line with Henderson and Dormon's findings.

Case report

A 32-year-old female presented to the CMCC Outpatient Clinic, complaining of a stiff neck and upper back pain. She stated that the problem originally began 8 years ago when she was hit in the head with a canoe. She had a further history of being hit in the head with a discus. The neck stiffness and upper back complaint has recurred 3 times over the past 8 years. One year prior to presentation, her complaint was initiated by carrying a child on her shoulders. Four days before the onset of her current pain, she stated that she had also been lifting her child in this manner. She related waking up with the pain, and feeling somewhat "dizzy" and short-tempered, feeling the sensation of "pressure" at the back of the occiput. She described the pain as a "burning" feeling across the left shoulder which was "constant" in nature. On movement she revealed that she felt a "grabbing" in her neck and that it had been "crunching" lately. She further described a "catching" sensation on extending her neck from the fully flexed positon. Her problem was aggravated by stress and relieved by stretching and ice. She saw a chiropractor 8 years ago after the original injury and felt that the treatments had helped her.

Physical examination findings revealed a mild flattening of

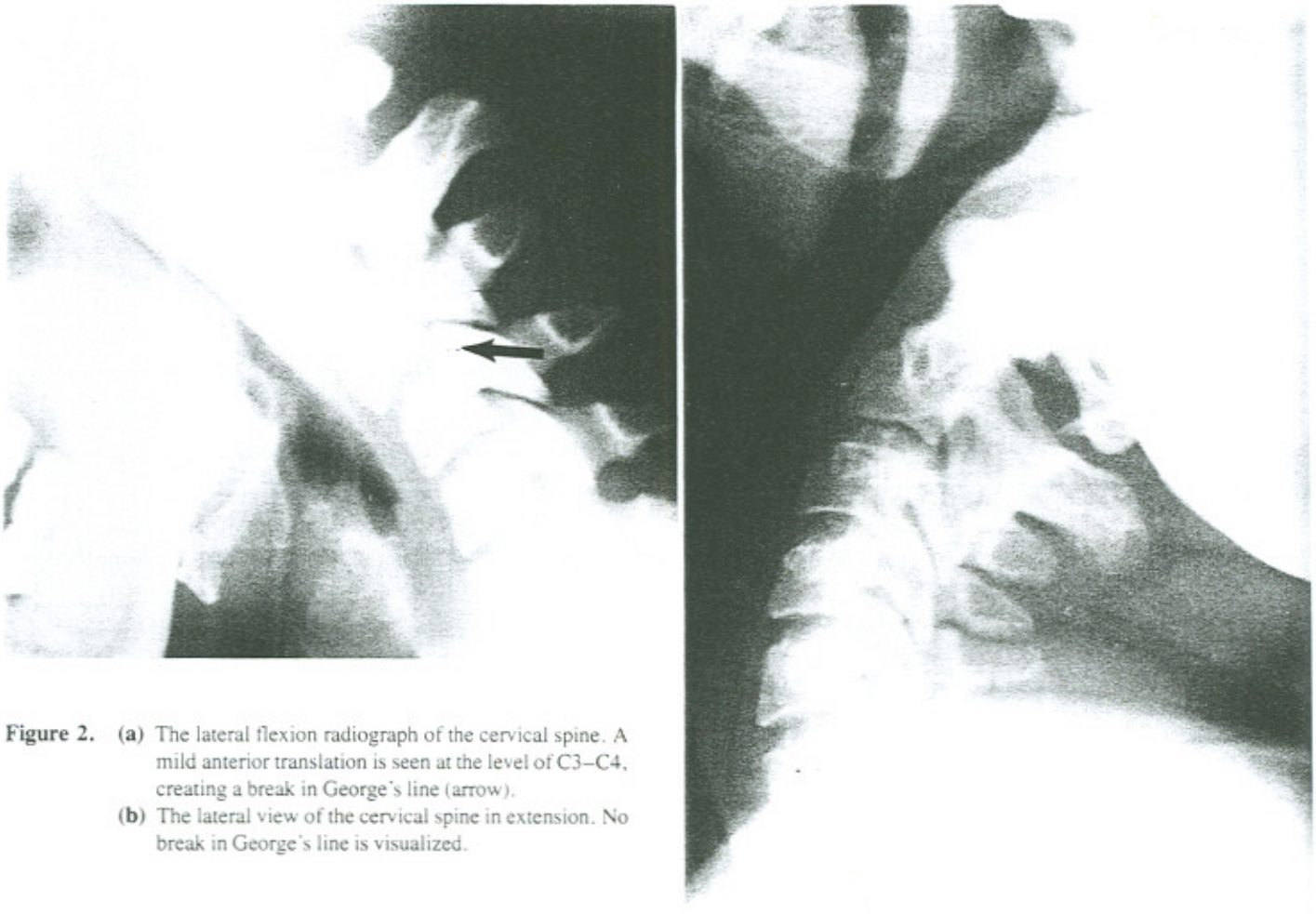

Figure 2. **(a)** The lateral flexion radiograph of the cervical spine. A mild anterior translation is seen at the level of C3–C4, creating a break in George's line (arrow).
(b) The lateral view of the cervical spine in extension. No break in George's line is visualized.

the cervical lordosis, and anterior head carriage. All tests for generalized hypermobility were negative. Range of motion was full and painfree in all directions, except for a pulling sensation created by forward flexion, and right and left lateral flexion. The patient described mild pain on full extension. Neurological examination was negative. Slight subjective weakness was noted when the left deltoid and left arm internal rotators were tested; this was attributed to right dominance. Kemp's test was mildly positive, and Jackson's test was negative. Hypertonicity was apparent in the left trapezius, scalenes and sternocleidomastoid muscles bilaterally. Fixations were noted at C1, C5, C6 and C7, as well at T2 to T5.

X-rays indicated adequate bone density. The cervical lordosis was flattened and a mild anterior shift was noted. Minimal discopathy was suggested at C5–C6, and C7–T1 (figure 1). On forward flexion, a break in George's line is apparent at the C3–C4 level (see figure 2a).

She was given a treatment regime of manipulations concentrating in the upper and lower cervical area, and a gently stretching exercise programme. Her symptoms resolved within one month after initiating treatment. However, she returned 3 times over the course of one year for recurrence of her neck complaints, in each case, related to minor traumatic incidents.

Discussion

This case typifies what has been clinically described as cervical intersegmental hypermobility, in this case at the C3–C4 level. Its importance lies in the conservative management of neck pain patients. It is suggested that intersegmental hypermobility would impact on the patient's prognosis, resulting in poorer response to conservative care, and that greater complexity may be involved in the application of spinal manipulative therapy. Unfortunately, the clinical manifestations of intersegmental hypermobility, and/or generalized hypermobility have not been objectively substantiated with flexion-extension x-ray results.

Summary

As discussed above, the difficulty in clearly defining the boundaries of abnormally excessive intervertebral movement in the cervical spine is as complex as outlined in Part I. This abnormality may be complicated by conditions affecting the body as a whole, as seen in generalized hypermobility. It is nonetheless, an issue that must be taken into account prior to manipulating the patient. It is reasonable to conclude, that unnecessary manipulation would not be well tolerated by a motion segment already affected by soft tissue and bony changes, hence predisposing it to increased movement. Although, empirically this is a rational assumption based on biomechanical logic, no known published study has attempted to integrate the clinical and x-ray result to this end.

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References

1 Bird HA, Tribe CR, Bacon PA. Joint hypermobility leading to osteoarthrosis and chondrocalcinosis. Ann Rheum Dis 1978; 37: 203–211.

2 Wood PHN. Is hypermobility a discrete entity? Proc Roy Soc Med 1971; 64: 690–692.

3 Finsterbush A, Pogrund H. The hypermobility syndrome: musculoskeletal complaints in 100 consecutive cases of generalized joint hypermobility. Clin Orthop 1982; 168: 124–127.

4 Beighton PH, Horan FT. Dominant inheritance in familial generalized articular hypermobility. J Bone Joint Surg (Br) 1970; 52: 145–147.

5 Carter C, Wilkinson J. Persistent joint laxity and congenital dislocation of the hip. J Bone Joint Surg (Br) 1964; 46: 40–45.

6 Grahame R, Edwards JC, Pitcher D, Gabell A, Harvey W. A clinical and echocardiographic study of patients with the hypermobility syndrome. Ann Rheum Dis 1981; 40: 541–546.

7 Jessee EF, Owen DS, Sagar KB. The benign hypermobile joint syndrome. Arthr Rheum 1980; 23: 1053–1056.

8 Schweitzer G. Laxity of metacarpophalangeal joints of fingers and interphalangeal joint of the thumb: a comparative interracial study. S Afr Med J 1970; 44: 246–249.

9 Beighton P, Solomon L, Soskolne CL. Articular mobility in an African population. Ann Rheum Dis 1973; 32: 413–418.

10 Grahame R, Jenkins JM. Joint hypermobility – asset or liability? Ann Rheum Dis 1972; 31: 109–111.

11 Klemp P, Stevens JE, Isaacs S. A hypermobility study in ballet dancers. J Rheumatol 1984; 11: 692–696.

12 Scott D, Bird H, Wright V. Joint laxity leading to osteoarthrosis. Rheumatol Rehabil 1979; 18: 167–169.

13 Wright V. Hypermobile states. Manuelle Medizin 1981; 19: 78–80.

14 Bird HA, Brodie DA, Wright V. Quantification of joint laxity. Rheumatol Rehabil 1979; 18: 161–166.

15 Hull RG. Articular hypermobility presenting after "aerobic" exercise. Clin Exp Rheumatol 1985; 3: 359–364.

16 Grahame R. Joint hypermobility – clinical aspects. Proc Roy Soc Med 1971; 64: 692–694.

17 Bird HA, Wright V. Joint hypermobility mimicking pauciarticular juvenile polyarthritis. Br Med J 1978; ii: 402–403.

18 Lewkonia RM, Ansell BM. Articular hypermobility simulating chronic rheumatic disease. Arch Dis Child 1983; 58: 988–992.

19 Scarf Y, Nahir AM. Hypermobility syndrome mimicking juvenile chronic arthritis. Rheumatol Rehabil 1982; 21: 78–80.

20 Biro F, Gewanter HL, Baum J. The hypermobility syndrome. Pediatrics 1983; 72: 701–706.

21 Lewkonia RM. Does generalized articular hypermobility predispose to generalized osteoarthritis? Clin Exp Rheumatol 1986; 4: 115–119.

22 Friedenberg ZB, Miller WT. Degenerative disc disease of the cervical spine: a comparative study of asymptomatic and symptomatic patients. J Bone Joint Surg (Am) 1963; 45: 1171–1178.

23 Farfan HF, Gracovetsky S. The nature of instability. Spine 1984; 9: 714–719.

24 Grieve GP. Lumbar instability. Physiotherapy 1982; 68: 2–9.

25 Henderson DJ, Dormon TM. Functional roentgenometric evaluation of the cervical spine in the sagittal plane. J Manip Physiol Ther 1985; 8: 219–227.

26 Dohler JR, Kahn MRH, Hughes SPF. Instability of the cervical spine after anterior body fusion – a study of its incidence and clinical significance in 21 patients. Arch Orthop Trauma Surg 1985; 104: 247–250.

27 Thabe H. Die Bewegungsstorung des Wirbelogengelenkes als praearthrotischer Faktor. Manuelle Medizin 1984; 22: 136–138.

28 Goel VK, Clark CR, McGowan D, Goyal S. An in-vitro study of the kinematics of the normal, injured and stabilized cervical spine. J Biomech 1984; 17: 363–376.

29 Barnes MP, Saunders M. The effect of cervical mobility on the natural history of cervical spondylotic myelopathy. J Neurol Neurosurg Psychiatry 1984; 47: 17–20.

30 Nachemson A. The role of spine fusion, question 8: how do we define instability? how is this diagnosed and what surgical treatment policy do you follow? Spine 1981; 6: 306–307.