Benign joint hypermobility – developing clinical significance

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ABSTRACT

The aim of this paper is to examine the clinical significance of joint hypermobility, and to suggest some diagnostic and management protocols which might be used in a chiropractic practice. Joint hypermobility is a largely unrecognized condition that is little understood, little talked about and often misdiagnosed. Clinicians may encounter patients with joint hypermobility but fail to appreciate the significance in terms of overall morbidity. The clinical significance of joint hypermobility is examined from many aspects. Considerations include the effect of joint hypermobility on different body structures as well as during pregnancy, on newborn, school-aged and adolescent conditions and the effect of different sports on the hypermobile child. Finally, the effects of joint hypermobility on spinal adjusting, and the modifications thereof, are discussed.

Key words: joint hypermobility, newborn, infant, child, pregnancy, sport, chiropractic, spinal adjusting, motor development, disc degeneration, scoliosis, attention deficit hyperactivity disorder.

History of Joint Hypermobility

An early clinical description of hypermobility was attributed to Hippocrates in the fourth century B.C., wherein he described the Scythians, a race of nomadic equestrian warriors who inhabited a region which is now the Ukraine.[1] One of the Scythians' main problems noted was the hyperlaxity of their elbow and shoulder joints which made it difficult for them to draw their bows or launch their javelins effectively. The clinical significance of hypermobility was not further reported until the late nineteenth century, when physicians were energetically describing and naming medical syndromes. During this period, the hypermobile character of joints became an important feature of conditions, notably in the Ehlers-Danlos and Marfan syndromes.

Definition and Characteristics of Joint Hypermobility

Hypermobile joints are defined as those that typically move beyond the normally accepted ranges of motion, taking into consideration age, sex, and ethnic background. The maximal range of movement that a joint is capable of is determined by the degree of tightness of the restraining ligaments. Thus, it has been determined that the primary cause of hypermobility is ligament laxity. Epidemiological studies have determined that hypermobility is seen in up to 10% of individuals in Western populations and as high as 25% in other populations.[2] The incidence of joint hypermobility within individual families suggest genetic inheritance, while the incidence difference between genders would imply a hormonal contribution. Joint hypermobility seems to be transmitted by an autosomal pattern, and first-degree relatives with the disorder can be identified in many cases.

Hypermobility may occur in a few joints (pauciarticular) or in multiple joints throughout the body (polyarticular). All joints have mobility; it is when joints demonstrate the ability to move excessively that issues occur. Joint mobility can be considered as a sliding scale, with some patients falling at the stiff jointed end of the scale, while others fall at the other end, i.e. the hypermobile end of the scale. The remaining patients who fall somewhere in the middle of the range, with joint hypermobility but without demonstrable clinical symptoms may go unnoticed clinically, leading to a frustrating life of undiagnosed pain and disability. Hypermobility (provided it is looked for) is seen commonly in clinical practice. Measurement scales for joint hypermobility have been devised, which allow an individual to be assigned a hypermobility rating. More will be discussed on rating systems in a later section of this paper.

Most of the research publications in the literature describe syndrome manifestations and associated management protocols. For many years, discussions in the literature have emphasized and reported on patients with increased levels of joint hypermobility, such as in Ehlers-Danlos or Marfan syndrome. Fewer studies have discussed the type of hypermobility that has no associated syndrome, but which affects a larger percentage of the population. It is the author's contention that chiropractors more frequently encounter this latter type of patient, and it is on this group that greater emphasis is placed in this paper.

Benign Joint Hypermobility Syndrome

Joint hypermobility is a term used to describe excess joint movement. However, when joint hypermobility leads to

symptoms in joints or other areas of the body, it is called Benign Joint Hypermobility Syndrome (BJHS). The characteristics of BJHS involve proprioception impairment, increased frequency of pain within joints and the tendency to injure soft tissues while performing physical activities. Most papers in the literature relating to joint hypermobility discuss this form known as BJHS.

Hypermobility of the joints is a common clinical finding in children, although not symptomatic in the majority. In general, girls have greater joint mobility than boys of the same age, with ranges usually being greater on the non-dominant side of the body. In studies which included race, Asians have been found to be more mobile than Caucasians.[3]

Causes of Joint Hypermobility
Joint strength is dependent upon the supporting ligament structure that crosses the joint space. Ligaments are composed mainly of collagen, so it is important that we discuss the collagen factors that contribute to joint hypermobility. There are 28 known types of collagen in the body, identified as types I through XXVIII.

Collagen is the main component of connective tissue and is the most abundant protein in the body, making up between 25% and 35% of the whole-body protein content. It is mostly found in fibrous tissues such as tendons, ligaments and skin. Collagen tissues may be rigid, as in bones, compliant as in tendons, or have a gradient from stiff to flexible as in cartilage. Collagen is also abundant in the tissues of blood vessels, the digestive tract, intervertebral discs and viscera. In muscle tissue, collagen makes up about 6 percent of the tissue serving as a major component of the endomysium, the tissue that sheaths each individual muscle fiber. The fibroblast is the most common cell that creates collagen and plays a critical role in tissue repair and wound healing.[4]

Collagen Variants
Different types of collagen serve different purposes in the body.

Type I collagen is the most abundant type of collagen in the human body. It is present in scar tissue, tendons, ligaments, muscles, bone, skin and viscera.

Type II collagen forms articular cartilage and hyaline cartilage. It makes up 85 to 90% of collagen found in articular cartilage.

Type III collagen is an essential component of ligaments, vascular structures, arterial walls and veins, skin and the digestive tract. Some studies have suggested the possibility that type III collagen deficiency may be implicated in congenital heart disease.[5]

The collagen that exists within the ligaments and joints of the skeleton is mainly composed of collagen type I and type III. Types I and III are the major constituents of ligament tissue, with type I collagen accounting for approximately 90% and type III for the remainder.[6]

Genetic Inheritance
A study of joint hypermobility by Bridges, demonstrated that up to 65% of patients with joint hypermobility had first-degree family members with a history of joint hypermobility.[7]

In a large chiropractic practice, it is not uncommon to find 3 to 4 generations of family members demonstrating various symptoms associated with joint hypermobility.

Assessment of Joint Hypermobility
The most widely used method of joint hypermobility assessment is to test whether a patient can perform a standard set of maneuvers, providing a numerical score, known as the Beighton score. Unfortunately, many clinicians omit these tests from their examination; as a result, joint hypermobility is often overlooked and its importance passes undetected.

Beighton Score
The Beighton score for assessing joint hypermobility is considered the gold standard for diagnosis, because it is quick, it is easy to use, and it has high intra-rater reliability.[8]

The 9-point scale is based on the following assessments:

1. passive apposition of the thumbs to touch the flexor aspect of the forearm,
2. passive dorsiflexion of the 5th fingers beyond 90°,
3. hyperextension of the elbows beyond 10°,
4. hyperextension of the knees beyond 10°, and
5. ability to place the palms of both hands flat on the floor, with knees in extension.

By this method a score can be assigned, with a maximum of nine points, one point for each thumb, one point for each 5th finger, one point for each elbow, one point for each knee, and one point for the ability to place the hands flat on the floor (spinal hypermobility). The maximum score is 9. A score of four or greater, on the 9-point scale, confirms the classification of hypermobility.

The Beighton score is a useful starting point, but it has a few shortcomings. For instance, it gives no indication of the severity of the hypermobility throughout the body. It merely indicates how widely that hypermobility is distributed throughout the musculoskeletal system. Because collagen is ubiquitous throughout the body, it became increasingly apparent that organ systems may also become involved and

should be considered as part of any evaluation. Further, certain individuals in different ethnic groups can demonstrate striking hypermobility, without any apparent symptomatology.

Brighton Criteria
The British Society of Rheumatology addressed the issue in 1999 and developed an updated evaluation system which became known as the Brighton Criteria.[9]

The advantage seen with the Brighton Criteria is that it incorporates symptomatology, thereby increasing the specificity for the diagnosis of benign joint hypermobility syndrome (BJHS).

The Brighton Criteria requirements are classified into major and minor criteria. According to the Brighton Criteria, benign joint hypermobility syndrome (BJHS) is diagnosed in the presence of 2 major and 2 minor criteria or 4 minor criteria. Two minor criteria will suffice if there are first degree relatives with a diagnosis of BJHS.

Major Criteria
- A Beighton score of 4 or greater (either currently or historically)
- Arthralgia for longer than 3 months in 4 or more joints

Minor Criteria
- Beighton score of less than 4
- Arthralgia in 1 to 3 joints for more than 3 months
- Back pain for more than 3 months
- Spondylosis, spondylolysis or spondylolisthesis
- Dislocation/subluxation in more than 1 joint, or in 1 joint on more than one occasion
- Soft tissue inflammation (epicondylitis, tenosynovitis, bursitis) in more than 3 locations
- Marfanoid habitus (tall, slim, arm span/body height ratio >1.03, upper/lower segment ratio <0.89, arachnodactyly)
- Abnormal skin striae, hyperextensibility, thin skin, thin (papyraceous) scars
- Eye signs: drooping eyelids, myopia, antimongoloid (medial to lateral upward) slant
- Varicose veins, hernia or uterine/rectal prolapse

Joint hypermobility is regularly identified in clinical practice. Use of the Beighton scoring system provides a quick and simple method of identifying those patients whose joint hypermobility may require further assessment.

Hypermobile patients often create the need for variation in delivery of spinal adjusting techniques. Close assessment of extremity joint hypermobility is important since it may be a factor contributing to other symptoms in the body. Joint hypermobility can have different effects in each age group and gender. In this regard, we will further examine specific problems of each age group and gender with appropriate recommendations for care.

Joint Hypermobility in Infants
The onset of hypermobility can be recognized at birth and, because it can significantly affect the newborn, diagnosis becomes important for the pediatric population. The recognition of potential problems is important when examining the infant as is the importance of the recently recognized effects linked with slowed motor development. Infants who test positive for joint hypermobility typically can bend further than typical. As a result, the trunk and extremity joints can appear weak and floppy. This increased flexibility also affects the muscles causing them to appear similarly floppy and weak.

Delayed Motor Development
Joint hypermobility is associated with an increased incidence of delayed motor development in infants.[10] Muscle weakness leads to difficulty sitting upright. Normal developmental milestones suggest that an infant should be able to sit unsupported in the upright, seated position by 6 months of age. Infants with joint hypermobility may not only be late sitting, but when they eventually sit, the spine characteristically flexes forward into kyphosis.

Three joints have been found to be significantly associated with motor delay. Joint hypermobility associated with motor delay is seen significantly with hip abduction, elbow hyperextension and foot dorsiflexion.[10] It is recommended that particular attention should be given to evaluation of these three joints when examining infants. Flexibility of the hip joints can affect an infant's ability to get up into a kneeling position or on to all fours. A hypermobile infant, on their tummy, will typically position their legs wider apart than usual, making it more difficult for them to flex the hips and come up to a kneeling position. One study identified that infants, from the ages of 8 to 14 months, who had hypermobile joints, also demonstrated a significantly increased incidence of motor delay.[11] This study further reported that within the following six months, normal motor development was achieved in the majority of subjects.

Congenital Hip Dislocation
Infants at high risk of developing congenital hip dislocation (CHD) are females, firstborn, and breech presentation deliveries. Many studies have demonstrated the association between congenital hip dysplasia and joint hypermobility. Breech births are two to eight times more common in females, which might explain why females account for 80 percent of all cases of hip dysplasia.[12] Salter described the likely etiology for CHD as involving marked congenital laxity of the hip joint capsule, being possibly hormonal and

possibly genetically determined.[13] Carter and Wilkinson showed that children who have congenital hip dysplasia, and their first-degree relatives, tend toward generalized joint hypermobility.[14] Wynne-Davies reported on the association between joint hypermobility and congenital hip dislocation as far back as the early 1970s.[15] A study by Carr demonstrated that children with congenitally dislocated hips had significantly more joint laxity than did controls.[16]

Developmental Dysplasia of the Hip

The term developmental dysplasia of the hip (DDH) which is now more commonly used, describes the whole range of deformities involving the growing hip, including frank dislocation, subluxation and instability, and dysplasia of the femoral head and acetabulum. Early diagnosis and treatment for DDH are critical. Screening for this condition is of utmost importance and traditionally has involved orthopedic testing after delivery (Ortolani test, Barlow test, and others). An awareness of a family history of generalized joint hypermobility can be of importance in early identification and management.

Joint Hypermobility in Childhood

In a British study by Adib, a group of 125 children (64 females) with joint pain were evaluated to help determine the etiology of their symptoms. Examination for hypermobility revealed that 94% scored more than 4/9 on the Beighton scale for generalized hypermobility. The joints most frequently involved were knees (92%), elbows (87%), wrists (82%), hand metacarpophalangeal joints (79%), and ankles (75%). The major presenting complaint was arthralgia in 74%, abnormal gait in 10%, apparent joint deformity in 10% and back pain in 6%. The mean age at first walking was 15 months; 48% were considered 'clumsy' and 36% as having poor coordination in early childhood. Twelve percent had 'clicky' hips at birth and 4% had congenitally dislocatable hips. Urinary tract infections were present in 13 and 6% of the female and male cases, respectively. Thirteen and 14%, respectively, had speech and learning difficulties diagnosed. A history of recurrent joint sprains was seen in 20% and actual subluxation/dislocation of joints in 10%. Forty per cent had experienced problems with handwriting tasks, 48% had major limitations of school-based physical education activities, 67% other physical activities and 41% had missed significant periods of schooling because of symptoms. Forty-three per cent described a history of easy bruising.[17]

Effect of Joint Hypermobility During Pregnancy

Questions one might consider, relating to joint hypermobility during pregnancy, include 1) does benign joint hypermobility during pregnancy cause an increase in low back or pelvic pain; 2) does benign joint hypermobility change birth outcomes; and 3) does benign joint hypermobility cause an increase in the time taken post-partum for the maternal pelvis to regain stability?

Concerning low back and pelvic pain, Mogren identified that women with joint hypermobility had more persistent low back and pelvic pain after pregnancy and had significantly earlier onset of pain during pregnancy.[18]

Knoepp identified that benign joint hypermobility syndrome may facilitate spontaneous vaginal birth but does not appear to be a risk factor for pelvic floor disorders in the first decade after childbirth.[19]

Calguneri conducted a study of changes in peripheral joint laxity occurring during pregnancy in 68 females. A significant increase in joint laxity measured during the last trimester of pregnancy was greater than measurements from the same individuals after parturition. When primigravida and multigravida were compared, a highly significant increase in laxity was found in women having their second baby over those having their first though no further increase in laxity occurred in subsequent pregnancies.[20]

A French study of obstetric outcomes in women with joint hypermobility indicated no significant increase in the incidence of deliveries by cesarean section or premature births: the incidence of both multiple and singular spontaneous abortion however, was significantly higher.[21]

Sandoz conducted a radiographic study of pubic symphysis stability post-partum.[22] With subjects standing, weight-bearing on one leg, it was determined that significant unilateral height deviation on the weight-bearing side of the pubic symphysis could be measured, indicating hypermobility of the joint supporting structure. Serial radiography determined that this hypermobility persisted for up to 6 months post-partum. No information was provided however relating to the subjects' joint hypermobility status or family history of joint hypermobility.

Joint Hypermobility and Musculoskeletal Pain

Children and adolescents with increased joint laxity have been found to frequently suffer from chronic musculoskeletal pain complaints.[23, 24]

One study found that 81% of Israeli school-children with fibromyalgia had joint hypermobility,[25] and another study based in the United States reported that 40% of adolescents with fibromyalgia also had joint hypermobility.[26]

An increasing number of studies have demonstrated a significantly increased incidence of back pain in subjects with BJHS. Morris (2017) conducted an extensive literature review on the topic of hypermobility and musculoskeletal

pain in adolescents. [27]

Joint Hypermobility and Intervertebral Disc Degeneration
A controlled study was carried out on male subjects aged between 20 — 30 years with lumbar disc herniation diagnosed by MRI. Joint hypermobility scores were evaluated based on the Beighton scale. The prevalence of joint hypermobility equal to or greater than 4/9 was significantly greater in the study group (13.2%) than in controls (5.1%).[26] An increasing number of studies have been conducted to determine the incidence of disc herniation in adolescents.[29-33] A controlled MRI study of 39 students at 15 years-of-age identified disc degeneration was present in 15 (38%) of the children with LBP and in 10 (26%) of the control subjects.[29] Although an increasing number of studies have identified disc degeneration as a cause of low back pain in children, no studies could be located that considered collagen gene mutations to be a potential cause of early deterioration of the intervertebral disc. This is an area of research that may help refine the etiology of intervertebral disc degeneration in the pediatric population.

Joint Hypermobility and Osteoarthritis
Joint hypermobility is common, familial and associated with joint pain and osteoarthritis. A U.S. study of 130 adult patients demonstrated a statistically significant association between joint hypermobility and the premature development of osteoarthritis.[7] There is increasing evidence that joint hypermobility is an important, yet largely unacknowledged, risk factor in the pathogenesis of osteoarthritis (OA). Hypermobility might be considered to place additional stress on the cartilage supporting and insulating the joint capsule, resulting in premature degeneration. Remember, we have already identified type II collagen as being responsible for the formation of most of the articular cartilage in joints. Genes have been identified as the strongest risk factor for OA in the general population and mutations in the genes for Collagen I, II, IX and X1 have been implicated in osteoarthritis.[34] A further study by Mustafa suggests that female hip OA is linked to a defect in the type IX collagen gene.[35]

Association between Joint Hypermobility and Adolescent Idiopathic Scoliosis
The prevalence of BJHS in a group of 1584 adolescents, 14 years of age, was 60.6% in girls and 36.7% in boys, when using the standard Beighton cut-off score of ≥4.[36]

Controlled studies to assess the incidence of joint hypermobility in adolescents with idiopathic scoliosis demonstrated that joint hypermobility occurred in 51% of the study group, whereas in the control group, joint hypermobility was identified in only 19% of cases.[37] No significant relationship was found between the prevalence for joint hypermobility and the Cobb's angle, degree of apical vertebral rotation, the number of vertebrae within the curve or the age of the subjects. Joint hypermobility prevalence however, was found to be higher in children with single curve scoliosis than in children with double curve scoliosis. The prevalence of generalized joint hypermobility in girls with idiopathic scoliosis varied by age. The younger the subject, the more likely she was to have joint hypermobility. The prevalence for each group was determined to be 9—12 years (34.2%), 13—15 years (25.6%), and 16—18 years (5.6%).[37] These findings suggest that children with a higher risk of developing adolescent scoliosis can be identified earlier by performing assessments for joint hypermobility. The prevalence and severity of scoliosis is higher in girls than in boys. For mild curves (10° to 20°) the ratio has been reported to be 1.4 to 1, whereas for more severe curves the ratio is reported as 7.2 to 1.

A study of 2600 female junior high school students demonstrated that classical ballet training was most common in adolescent girls with idiopathic scoliosis. The odds of adolescent idiopathic scoliosis developing increased as the frequency of training, the number of years of experience, and duration of training in ballet increased.[38]

Children with joint hypermobility have increased joint flexibility and are more likely to develop adolescent idiopathic scoliosis. Joint hypermobility is a physical characteristic that provides a distinct advantage for participation in classical ballet. Subjects with joint hypermobility may be drawn to ballet as a sport because their body is better able to perform the required movements. This fact may help to explain the predominance of ballet dancers with scoliosis. Dancers who are naturally predisposed to their sport may proceed to higher levels of performance, being reflected in their frequency of training and duration and years of experience. They perform at a higher level because their body affords them this ability.

Effect of Joint Hypermobility on Muscles
A clinically consistent finding in patients with BJHS is tight paraspinal muscles. Affected patients seek out exercise routines and home care to help with this affliction but the effects appear to be only short term. Involuntary muscle contraction in these patients is likely associated with nerve compression or irritation of the nerve supply to the involved muscles. The increased motion of the spinal joint capsules, being a cause of mechanical stimulation, could be a result of irritation of the free nerve endings (nociceptors) in the intervertebral facet joint capsules and associated ligaments. Peripheral nociceptive fibers transmit sensory impulses to the spinal cord, performing a "loop", similar to the neurology of deep tendon reflexes, before transmitting a motor stimulus out to skeletal muscle fibers. The brain

is simultaneously informed of the painful stimuli but can significantly suppress the pain signals by secreting endogenous opioids. The result of this muscle stimulation activity likely produces the common finding of tight paraspinal muscles in patients with FLL.

Patients with tight muscles associated with BJHS will frequently find their way into yoga classes where the exercise routines stretch and loosen the tight spinal muscles. Because such patients naturally have increased joint flexibility they find yoga movements quite easy to perform. "Pilates" exercise routines also seem to have similar effects on patients with BJHS. Massage also would appear to help, but the effect is only short term. Because these patients' muscles are chronically tight, possibly due to the build-up of lactate due to anaerobic metabolism, patients with FLL will often complain of intense muscle soreness when tissues are pressed upon as in a deep soft tissue massage. It is not uncommon for such patients to have been previously diagnosed with fibromyalgia. An increasing number of studies, previously discussed, have shown a significant link between fibromyalgia and BJHS.[26, 39]

Infants and toddlers with joint hypermobility tend to have tight hip muscles affecting their ability to crawl, walk and balance. This may contribute to a delay in their ability to sit independently, often sitting with a very rounded back or sitting cross-legged in the "W" position (flexed, internally rotated hips, with flexed knees). These infants often skip crawling completely, being inclined to bottom shuffle instead. Children with joint hypermobility tend to develop co-ordination and attention problems as they get older.[17] This characteristic may be attributed to their having tight muscles and, since movement helps to loosen and stretch out tight muscles, it may be that their more frequent mobility provides them with some level of comfort. The question arises: Could such children be labeled as "hyperactive" simply due to the difficulty they have in sitting still in class?

Effect of Joint Hypermobility on Vascular Tissues

Joint hypermobility and Ehlers-Danlos syndrome (EDS) has been well documented. The increased flexibility and fragility of the soft connective tissues in such patients results in a wide range of changes in the skin, ligaments, joints, blood vessels and internal organs. Ehlers-Danlos syndrome has been sub-classified into six types based on the associated clinical manifestations. The type IV classification of EDS has increased vascular findings.[50] These findings include easy bruising, early onset of varicose veins, fragile arteries, intestinal symptoms and uterine fragility or rupture. Abnormalities with the expression of collagen type III have been identified as being associated with EDS.

Patients with a similar problem in the expression of type III collagen, but who are not as far along the hypermobility scale as those with EDS, may well have some of these vascular signs and symptoms as seemingly unrelated findings. Patients with varicose veins are commonly seen to have joint hypermobility. Along with a high frequency of occurrence of varicose veins, hemorrhoids and uterine prolapse have also been identified.[40] The development of varicose veins during pregnancy is recognized as being due to hormonal mechanisms which create venous dilatation. The contributing factors are mechanical obstruction of the venous outflow in the pelvis due to the increasing size of the baby, increase in the circulating blood volume and hormonal effects causing smooth muscle dilatation with an inhibition of normal contractility.

Pulmonary Embolism

A pulmonary embolism occurs when an embolus, usually a blood clot, blocks the blood flowing through an artery that feeds the lung. The most common cause of these emboli is deep vein thrombosis (DVT) in an extremity. The cause of DVT can be an injury to the muscles, soft tissues or blood vessels causing clots to form which travel to the lungs. An inherited weakness of the tissues forming the walls of the blood vessels could be a precipitating factor in pulmonary embolism in which type III collagen rich systemic arteries may be predisposed to undergo dissection, aneurysm, or rupture. A study to evaluate the hypermobility of patients diagnosed with pulmonary embolism may well add to the knowledge base of BJHS.

Effect of Joint Hypermobility on Bones

When treating a large cohort of patients, it may often be noticed that patients who present with joint hypermobility also have a history of one or more fractures. The question arises: is there a link between joint hypermobility and fractures?

Joint hypermobility syndrome is a characteristic feature of osteogenic imperfecta (OI), a disorder caused by genetic defects that affect the body's ability to make strong bones. The affected individual has too little type I collagen or a poor quality of type I collagen due to a mutation in one of the type I collagen genes. Collagen is the major protein of the body's connective tissue and genetic mutations that interfere with collagen production result in fragile bones that break easily. Type I collagen defect is also associated with ligament laxity, so it is likely that the possible link between JHS and OI is noticeable.

OI has various subtypes, some of which are lethal. OI Type I is the most common and mildest form of OI and the two conditions may therefore be linked clinically. The following list describes the clinical features of the mildest form of OI.

- Most common and mildest type of OI.

- Bones fracture easily; most fractures occurring before puberty.
- Normal or near-normal stature.
- Loose joints and muscle weakness.
- Sclera (whites of the eyes) usually have a blue, purple, or gray tint.
- Triangular face.
- Tendency toward spinal curvature.
- Bone deformity absent or minimal.
- Brittle teeth possible.
- Hearing loss possible, often beginning in early 20s or 30s.
- Collagen structure is normal, but the amount is less than normal.

Osteoporosis
Studies have shown that femoral and trochanteric bone mineral density scores were significantly lower in hypermobile patients compared to controls. Low bone mass was more frequently found among subjects with hypermobility (p=0.03) and hypermobility was found to increase the risk for low bone mass by 1.8 times (95% confidence interval).[41]

Effect of Joint Hypermobility in Sports
Individuals possessing joint hypermobility seem to have an advantage in certain sports and artistic occupations, gymnastics and ballet being most noticeable. Individuals with an increased degree of hypermobility may self-select into sports for which joint hypermobility provides a distinct advantage. Sports coaches too may well be aware of such advantages and be on the look-out for novices who at an early stage of training are already demonstrating the flexibility characteristics required for success.

Gymnastics
Gymnastics is a sport for which joint hypermobility is required to become competitive at a high level of the sport. A young gymnast who at an early age is able to do a backbend and place her head down behind her heels may quickly catch the attention of a coach. Increased joint range in many a hypermobile child is a familial trait that helps with their performance ability. So, can joint range be increased into the hypermobile range by sheer hard work and training? Of course, many world class gymnasts demonstrate the advantages of hard work and long hours of training, but what about the young child who wants to become a gymnast and does not have the joint hypermobility endowed upon so many of her fellow competitors? As one patient (low on the Beighton scale) complained to me, "I was a gymnast as a child and I worked longer and harder than most of the others that I trained with, but I was never able to perform the moves that just seemed to come naturally to the others in my team." Some of the characteristic traits that one sees frequently in world class gymnasts are hypermobility of the lumbar spine and hyperextension of the elbows demonstrating that they would likely score high on Beighton's scoring system.

Ballet
Ballet dancers who are not inherently lax jointed need to acquire hypermobility in certain joints to perform their art. The question we should ponder here is "are ballet dancers born or are they made"? How much of the joint laxity is the result of regular training and how much is due to inherited joint hypermobility? Certainly, joint flexibility is a great advantage to ballet dancers in performing the impressive movements required of their craft. Controlled studies have demonstrated that ballet dancers show a significantly higher Beighton score than non-dancers.[40] A study conducted of the most prestigious ballet companies in the U.K. noted that joint hypermobility was prevalent in 95% of female and 82% of male dancers, which suggests that to some extent, inherited JH was a selection factor in recruiting.[41] The downside of increased flexibility in dancers is that joints such as the elbows tend to hyperextend past the neutral position "spoiling" the graceful lines that professional dancers strive so hard to achieve. This problem however, can be compensated for to a degree by developing the supporting musculature of the hypermobile joints or by voluntary muscular control, thus helping to correct the less acceptable appearance of such alignment. A question one might ponder is "does the flexibility that is so common in ballet dancers cause long term damage to their joints?"

Studies of dancers would suggest that they are more likely to develop premature osteoarthritis of the hip.[44] Radiographic studies of early joint degeneration in professional ballet dancers show thinning and irregularity of the medial knee joint with bone marrow changes in the lateral femoral condyle, loss of joint space and degenerative changes in the hip. Miller conducted a study of injuries to classical ballet dancers and identified a range of problems that included osteochondral fractures, fatigue fractures, sprains, chronic ligamentous instability of the knee, degenerative arthritis of multiple joints and low back pain.[43] One might ponder if the high incidence of stress fractures in the legs and feet of ballet dancers could be contributed to by defective collagen in the effected bones associated with their joint hypermobility.

Swimming
Swimming is a sport that requires muscular strength, good aerobic capacity and endurance. Elite swimmers however are often endowed with an additional quality: hypermobility, which in swimming is an undeniable asset. Flexibility in the shoulder joints makes it possible not only to swim faster but also to swim less slowly (a subtle difference). Increased shoulder mobility helps the swimmer to lift their shoulders out of the water more easily, increasing the am-

plitude of their stroke. In some swimming strokes, such as butterfly, shoulder hypermobility allows the joint to subluxate the humerus from the glenoid fossa, permitting a wide range of shoulder movement and resulting in a greater power stroke. Finally, ankle flexibility is a key asset when swimming. A striking example can be seen watching US Olympic champion, Michael Phelps swim. In addition to his size 14 feet, his ankles can bend 15 degrees further into dorsiflexion than typical. Swimming may be a sport chosen by people with joint hypermobility because of the distinct advantages it provides. Recognizing this, clinicians should be in a better position to advise their hypermobile patients regarding appropriate sports participation.

Athletics

In athletic competition, individuals with small physiques are more suited to long distance running whereas taller individuals have an advantage in long jumping. Hurdlers require a wide range of hip joint flexibility to allow for increased hip abduction, permitting smooth transition over the obstacles in their path. Joint flexibility is an advantage for high jumpers, while javelin throwers need shoulder flexibility. These are just some examples to indicate the advantages conferred by joint flexibility. This is by no means an extensive list of sports but awareness in the mind of the clinician helps identify the causes of some sport-based patient complaints. However, hypermobility is not necessarily an advantage in every sport. In sports such as soccer, football, wrestling, basketball and volleyball, hypermobility is not seen as an advantage, but it may influence the pattern of sporting injuries sustained. Hypermobility studies have demonstrated that joint proprioception in the lower limb is reduced.[46] This may be a contributing factor to ankle sprains in basketball players.

Link Between Joint Hypermobility and Attention Deficit Hyperactivity Disorder

Attention-deficit/hyperactivity disorder (ADHD) and BJHS are two separated conditions, assessed and managed by different specialists without overlapping interests. Recently, some research has highlighted an unexpected association between these two clinical entities: A controlled study by Shiari identified joint hypermobility in 74.4% of children with attention deficit hyperactivity disorder compared with 12.8% of healthy controls.[49] A study of the research literature by Baeza-Velasco highlighted potential symptomatic links between ADHD and joint hypermobility as involving impaired coordination, impaired proprioception, fatigue, chronic pain, and dysautonomia.[47]

Hypothesis: Link Between ADHD and Joint Hypermobility

Here is an untested hypothesis derived from many years of clinical observation by the author. After specializing in pediatrics for many years, the incidence of children presenting with joint hypermobility and ADHD became an expected finding. As we have previously discussed, patients with joint hypermobility frequently suffer with tight paraspinal muscles. Movement and stretching of the body joints and limbs has been shown to help, with adult patients reporting improvement after stretching classes, such as yoga. In school, children often are required to sit still for extended periods of time. Children with joint hypermobility may have difficulty with this request, because they innately feel that movement helps their condition. As a result, they may jump up or squirm and fidget around in their seat, perhaps because moving their muscles and joints around feels good. Is this then why they are labeled hyperactive?

Spinal Hypermobility - Patient Characteristics

A frequently encountered characteristic in any chiropractor's patient population is the patient who can "self-adjust." One example is the school-aged child who, while sitting in class, twists the torso around in both directions to make the spinal joints "pop." Another is the patient who twists the head in both directions far enough to induce cavitation. Both these patient types report deriving some "ease" of the tightness they feel in the spine and associated tissues.

Clinical Considerations for Spinal Adjusting and the Hypermobile Patient

Infant joints are naturally more mobile than older children and adults. Evaluating an infant for hypermobility should include the family history, particularly as it relates to hypermobility. Testing the mother and father, according to the Beighton criteria, may identify a familial propensity to hypermobility which can affect the infant. Additionally, evaluating an infant's motor skills may identify developmental delay.

Manual adjustment of the spine requires a technique to move the intervertebral joints to the point of ligament tension, which is just short of the point at which joint cavitation will occur. This is followed by a light thrust to release the vacuum within the joint, which increases the facet joint space dimension, thus reducing pressure on the intracapsular structures. The difficulty encountered in adjusting hypermobile patients is associated with their increased range of motion. To reach the point of ligament tension a greater range of movement is required.

This requirement creates difficulty in successfully performing manual spinal adjustments on this patient type. To compensate for this excess joint mobility, some doctors will utilize an Activator-type adjusting instrument that delivers the impulse thrust with the spine in the neutral position. Others may use a "drop-technique," "cervical stair-step" or "press and hold" type of adjustment, using an appropriate line of drive. Manual adjusting techniques for the spine are

most commonly the Diversified or Gonstead type adjustments with Activator Methods being the most frequently used instrument adjusting technique.[48] It is the author's opinion that for patients identified with joint hypermobility, the Gonstead seated-type adjustment for the cervical spine is more appropriate than the Diversified adjustment, because it uses a reduced rotational component to reach the point of joint pre-stress. Doctors may find that the hypermobile patient whose cervical spine is difficult to adjust in the supine position responds better to a Gonstead-type seated adjustment as an alternative.

Considering the greater recognition that is being given to the clinical effects of joint hypermobility and the significant advances that have been delineated, chiropractors may well want to modify their standard approach to spinal adjusting. Studying each patient to identify their level of joint hypermobility may help achieve the successful clinical outcomes that all patients deserve.

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