Progressive Spinal Kyphosis in the Aging Population

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Thoracic kyphosis tends to increase with age. Hyperkyphosis is defined as excessive curvature of the thoracic spine and may be associated with adverse health effects. Hyperkyphosis in isolation or as a component of degenerative kyphoscoliosis has important implications for the surgical management of adult spinal deformity. Our objective was to review the literature on the epidemiology, etiology, natural history, management, and outcomes of thoracic hyperkyphosis. We performed a narrative review of literature on thoracic hyperkyphosis and its implications for adult spinal deformity surgery. Hyperkyphosis has a prevalence of 20% to 40% and is more common in the geriatric population. The cause is multifactorial and involves an interaction between degenerative changes, vertebral compression fractures, muscular weakness, and altered biomechanics. It may be associated with adverse health consequences including impaired physical function, pain and disability, impaired pulmonary function, and increased mortality. Nonoperative management may slow the progression of kyphosis and improve function. Surgery is rarely performed for isolated hyperkyphosis in the elderly due to the associated risk, but is an option when kyphosis occurs in the context of significant deformity. In this scenario, increased thoracic kyphosis influences selection of fusion levels and overall surgical planning. Kyphosis is common in older individuals and is associated with adverse health effects and increased mortality. Current evidence suggests a role for nonoperative therapies in reducing kyphosis and delaying its progression. Isolated hyperkyphosis in the elderly is rarely treated surgically; however, increased thoracic kyphosis as a component of global spinal deformity has important implications for patient selection and operative planning.

KEY WORDS: Adult spinal deformity, Dowager's hump, Proximal junctional kyphosis, Senile kyphosis, Thoracic hyperkyphosis

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Degenerative changes affecting the spine accumulate and contribute to an overall trend of kyphosis with increased age.[1] Hyperkyphosis, defined as excessive curvature of the thoracic spine in the sagittal plane, is commonly referred to as "dowager's hump." The prevalence of hyperkyphosis in older individuals is not precisely known but has been reported to be between 20% and 40%.[2,3] In general, a thoracic kyphosis angle greater than 40 degrees, which represents the 95th percentile of normal in young adults, is defined as hyperkyphosis.[4,5] Mean thoracic kyphosis (TK) increases as we age: from 20 to 29 degrees in individuals under 40 years of age[4] to 53 degrees in 60 to 74 year olds, and 66 degrees in those older than 75 years of age.[6] The kyphosis angle increases more rapidly with age in women than in men.[7] This progression makes it difficult to establish uniform thresholds that distinguish pathological hyperkyphosis from "normal" changes associated with aging.

ABBREVIATIONS: ASD, adult spinal deformity; **BMD,** bone mineral density; **DDD,** degenerative disc disease; **LL,** lumbar lordosis; **OR,** odds ratio; **PI,** pelvic incidence; **PJK,** proximal junctional kyphosis; **PT,** pelvic tilt; **SSV,** sagittal stable vertebra; **TK,** thoracic kyphosis; **UIV,** upper-most instrumented vertebra

Hyperkyphosis may negatively impact several aspects of an afflicted individual's health including physical function, pulmonary function, pain and disability, and even mortality. Although evidence is still limited, certain medical strategies seem to be effective in delaying progression and mitigating the adverse consequences of hyperkyphosis. Surgery is not commonly performed in elderly persons with isolated hyperkyphosis due to their typically high-risk profile, which often includes multiple medical comorbidities and poor bone quality. Operative intervention is therefore reserved for patients with neurological compromise, documented progressive disease impacting function, or intractable and refractory pain.[8] In the broader context of the management of adult spinal deformity (ASD), hyperkyphosis has important implications for surgical planning, as its presence often necessitates long segment fusions, including the upper thoracic spine, to reduce the risk of proximal junctional kyphosis (PJK).

The present review will discuss the pathogenesis, evaluation, and management of thoracic hyperkyphosis. The surgical management of degenerative spinal deformity, including isolated sagittal plane deformity, the category in which hyperkyphosis falls, is discussed in a separate article within this supplement entitled "Degenerative Spinal Deformity." The medical and surgical management of osteoporosis and osteoporotic vertebral fractures, a major contributing factor to hyperkyphosis in the elderly, will be discussed in articles within this supplement entitled "Perioperative Medical Management of Spine Surgery Patients With Osteoporosis" and "Management of the Elderly With Vertebral Compression Fractures," respectively.

PATHOGENESIS

The pathogenesis of hyperkyphosis is multifactorial. The main anatomic structures that contribute to the sagittal curvature of the spine are the vertebral bodies and intervertebral discs. As such, any process that results in anterior wedging of the vertebral bodies or asymmetric collapse of the disc will increase kyphosis. Vertebral compression fractures are widely thought to be a major contributing factor in the development of age-related hyperkyphosis. A greater degree of thoracic kyphosis is seen in patients with one or more vertebral fractures than those without such fractures.[7,9,10] Furthermore, observational studies have demonstrated an increase of 3.8 degree in overall kyphosis for each vertebral compression fracture.[11,12] However, the minority of elderly individuals with the worst kyphosis (less than 40%) have underlying fractures of the vertebrae.[9,13] These data suggest that other factors contribute to the development of increased kyphosis in older persons.

There is a strong association between osteoporosis, vertebral fractures, and hyperkyphosis. The failure load of a vertebral body is determined by the density and architecture of trabecular bone and the shape and size of the vertebral body. Fractures occur when forces on the vertebral body exceed its failure load, which can occur in the setting of weak bone or large forces.[14] Biomechanical models of the spine that simulate stress loading indicate that forces applied to the osteoporotic spine comparable to those experienced in daily life are sufficient to cause vertebral wedging and compression fractures.[15,16] In women, a greater degree of kyphosis is correlated with increased likelihood of subsequent vertebral fractures, even in the absence of prior fractures.[17] Additionally, as bone mineral density (BMD) decreases, the severity of wedging associated with compression fractures increases; this perpetuates further vertebral compression fractures due to increased load on the anterior column and progression of kyphosis in a cascading fashion.[1,18,19]

Degenerative disc disease (DDD) likely plays an important role in both loss of lumbar lordosis and progression of thoracic kyphosis angle. With age, discs desiccate with resultant height loss, commonly in an asymmetric fashion with greater loss anteriorly. This may lead to anterior wedging and thus progression of kyphosis.[9,12,18] For example, in a study of healthy women aged 39 to 91, a significant correlation between kyphotic angle and anterior disc height was identified ($r = -0.34$).[20] DDD exists in the majority of older adults with hyperkyphosis, including those with no evidence of vertebral fractures or osteoporosis.[9] Nevertheless, strong associations between vertebral wedging and kyphosis angle[1,18] suggest that it is the combination of DDD and vertebral compression deformity that contribute to development and progression of hyperkyphosis.

One important mechanism through which DDD may contribute to the development of osteoporotic vertebral fractures emerges in the concept of "stress-shielding." According to this principle, disc degeneration is associated with reduced loading of the anterior vertebral body in the erect posture. As a result of decreased loading ("stress-shielding"), there is local reduction in BMD and inferior trabecular architecture. Flexion concentrates forces on the weakened vertebral body leading to failure and fracture at a decreased load.[21,22] Cadaveric studies have demonstrated that, with upright posture, advanced disc degeneration transfers compressive load bearing from the anterior vertebral body to the posterior neural arch. Vertebral bodies between such severely degenerated discs had 20% lower trabecular volume fraction, 16% fewer trabeculae, and greater intertrabecular spacing in the anterior third compared with the posterior third. During flexion, more than 50% of the load bearing is transferred to the anterior vertebral body where compressive strength is proportional to BMD.[21] Thus, intervertebral disc degeneration transmits load bearing from the anterior and middle columns to the posterior column, weakening the anterior vertebral body and predisposing it to fracture during flexion.

In addition to structural changes in the vertebral column, functional changes in posture and muscle strength are associated with degree of kyphosis. Several studies have demonstrated an inverse correlation between spinal extensor muscle strength and hyperkyphosis.[23,24] In the lumbar spine, fatty degeneration and volume loss of the paraspinal muscles has been demonstrated in association with degenerative kyphosis.[25] Weak grip and ankle strength are associated with age-related hyperkyphosis, suggesting that general deconditioning is an important contributing factor in the geriatric population.[19,26] Several spinopelvic parameters have been shown to be strongly correlated with maximum TK including, pelvic incidence (PI), pelvic tilt (PT), and maximum lumbar lordosis (LL).[27] Similarly, cervical lordosis is correlated with thoracic kyphotic angle.[6] These associations suggest that regional alignment of the pelvis and other spine regions influence and are impacted by thoracic kyphosis.

Thoracic hyperkyphosis may also result from a variety of idiopathic, genetic, and metabolic conditions. The most common idiopathic cause of hyperkyphosis is Scheuermann disease, which has an estimated prevalence of 8.3%[24] and likely has a genetic component.[28] Scheuermann disease is characterized by a kyphotic deformity of the thoracic or lumbar spine that typically manifests in early adolescence and results from developmental anterior wedging of the vertebrae. Genetic conditions associated with early onset hyperkyphosis include Marfan syndrome, Ehlers Danlos syndrome, osteogenesis imperfecta, and mucopolysaccharidoses.[3] Hyperkyphosis is also more likely in older individuals who report a family history of the same, even after adjusting for family history of osteoporosis or vertebral fractures.[3]

ADVERSE HEALTH EFFECTS

The adverse health consequences of thoracic hyperkyphosis are varied and include impairment in physical function, diminished pulmonary function, increased vertebral fractures, increased falls, and an increase in mortality. Physical function impairment in association with hyperkyphosis has been demonstrated in several observational studies.[24,29-35] Patients with hyperkyphosis perform poorer on formal tests of physical function including the timed get up and go test,[35] walking speed,[29,33] and standing from a chair test.[34] Self-reported impairment in activities of daily living has also been reported in such patients.[32-34] Women with hyperkyphosis have worse balance, wider stance, and slower gait velocity[26]; importantly, these factors have all been associated with an increased risk of falls.[19]

Hyperkyphosis is associated with back pain, disability,[7,32,36] and decreased quality of life.[37,38] There are multiple potential mechanisms through which hyperkyphosis could cause pain including underlying degenerative changes, vertebral fractures (especially when acute), increased positive sagittal malalignment, sternal insufficiency fractures, and reflux esophagitis. Patients with osteoporosis report increased physical difficulty, more modifications to daily life, and greater fear regarding the future.[37] They also report poorer satisfaction with their health status and with their lives in general.[38]

As discussed above, hyperkyphosis is associated with vertebral fractures. Although traditionally it has been presumed that fractures perpetuate kyphosis, the reverse is likely also true.[17,39] Hyperkyphosis may result in altered mechanical forces on the thoracic vertebrae, which could increase the risk of osteoporotic fractures. Huang et al[17] found an increased risk of fracture in older women with hyperkyphosis compared to those without (odds ratio [OR] 1.7 [1.0-3.0]) even after adjusting for age, BMD, and prior vertebral fracture. This suggests that hyperkyphosis is an independent risk factor for future vertebral fractures. The risk of fracture increases with greater degree of thoracic kyphosis.

Modest impairment in pulmonary function has been consistently demonstrated in studies examining the impact of hyperkyphosis.[40,41] Patients with hyperkyphosis are more likely to exhibit obstructive and restrictive ventilatory dysfunction,[42] dyspnea,[42] and decreased exercise tolerance[43] than those with $<$40 degrees of kyphosis. An increase in kyphosis, as measured by Cobb angle, is associated with decreased forced vital capacity.[44]

Thoracic hyperkyphosis is associated with increased all-cause mortality and this association is stronger with more severe kyphosis. In particular, pulmonary death is more common in the presence of severe hyperkyphosis.[13] Even after adjusting for vertebral fractures and osteoporosis, hyperkyphotic posture is a predictor of increased all-cause mortality.[2,45] Furthermore, Kado et al[45] also showed that older women with hyperkyphosis and vertebral fractures had a higher mortality rate than those with either condition alone.

EVALUATION

Hyperkyphotic posture is readily identified on physical examination. History should focus on when the postural abnormality developed, associated pain and disability, and alleviating and aggravating factors. Risk factors for hyperkyphosis, including osteoporosis, previous vertebral fractures, and family history, should be evaluated. The presence of any neurological deficits should be ascertained, although these are uncommon in isolated thoracic hyperkyphosis. Medical comorbidities, common in this demographic, should be ascertained, as these may guide treatment selection. As with the evaluation of any spinal deformity, physical examination begins with postural assessment in the supine, standing, and walking position. Comparison between upright and supine position gives the examiner a sense of the rigidity of the kyphotic deformity. A thorough neurological examination, including motor power, sensation, and reflexes (including pathological), should be conducted, as the presence of deficits may mandate more urgent investigation and treatment.

The gold standard measurement of kyphosis is the Cobb angle from the cranial endplate of T2 to the caudal endplate of T12 on standing lateral long cassette radiographs. When T2 cannot be visualized due to overlying bone or soft tissue structures, T4 or T5 are used as a substitute. In the context of assessing a patient with ASD, the TK is measured along with standard radiographic parameters including the PI, PT, sagittal vertical axis, C7 plumb line, and Cobb angle of any coronal curves. For discussion of these measurements, please see the article "Degenerative Spinal Deformity" in this supplement. These measurements allow for a comprehensive assessment of spinal alignment to aid in selection of operative vs nonoperative care and, in the case of the former, surgical planning.

Several clinical methods are available to measure kyphosis and are typically used by clinicians who follow patients with hyperkyphosis and are involved in their nonoperative management (eg, physiatrists, geriatricians, rheumatologists). These include the Debrunner kyphometer, flexicurve ruler, inclinometer, and goniometer.[3] The Debrunner kyphometer and flexicurve ruler in particular have similar reliability and validity to standard radiographic measurements in the assessment of TK.[11]

IMPACT OF HYPERKYPHOSIS ON SPINAL ALIGNMENT

Global spinal alignment is dependent on regional alignment of the various spine regions and the pelvis.[46] All else being equal, thoracic hyperkyphosis results in increased positive sagittal malalignment. Multiple studies have demonstrated that sagittal imbalance is the strongest driver of pain and disability in patients with ASD.[46-54] As such, the degree of thoracic kyphosis is an important consideration in the overall management of patients with ASD. When considering operative intervention, the presence of increased thoracic kyphosis has important implications. Selection of the upper-most instrumented vertebra (UIV) in a planned thoracolumbar fusion depends in part on whether thoracic hyperkyphosis is present. The physiological apex of thoracic kyphosis, T5-8, should be avoided,[55,56] and hence the UIV is typically at or below T10 (distal thoracic) or between T2 and T5 (proximal thoracic). Selection of a distal thoracic UIV in the presence of thoracic hyperkyphosis is undesirable, as there is an increased risk of suboptimal realignment and the development of postoperative PJK (Figure 1). Furthermore, patients with increased preoperative TK and distal thoracic, as compared to proximal thoracic UIV, have greater postoperative loss of correction.[57,58] Selection of proximal thoracic UIV is not benign, however, as it has been associated with longer operative time, greater blood loss, and longer hospital stay.[59]

Hyperkyphosis has a significant yet incompletely defined relationship with PJK. Glattes et al[60] have defined PJK as an increase in proximal junctional kyphotic angle by 10 degrees or more as measured from the caudal endplate of the UIV to the cephalad endplate of the vertebra 2 segments cranial to the UIV. According to this definition, PJK generally does not significantly impact clinical outcome or quality of life[60] and rarely leads to revision surgery.[61] There are a subset of patients, however, with more severe PJK which is associated with pain, increased deformity, increased need for revision surgery and, occasionally, neurological deficit.[62-65] The term proximal junctional failure (PJF) has been proposed to encompass this subgroup. Hart et al[66] have defined PJF as PJK that is associated with structural failure and associated with clinical deterioration (Figure 2).

Several studies have shown that thoracic hyperkyphosis is associated with an increased risk of PJK after long segment instrumentation for spinal deformity.[65] In particular, increased kyphosis from T2 to the UIV,[67] preoperative TK greater than 30 degrees[68] and greater than 40 degrees,[69] and greater preoperative and postoperative TK[70] have all been associated with increased risk of PJK. The impact of number of instrumented levels and location of UIV (proximal vs distal thoracic) on PJK rate is less well defined. Studies have demonstrated conflicting results: both greater and lesser number of instrumented levels has been associated with higher risk of PJK.[71,72] Similarly, upper and lower thoracic UIV have been reported as risk factors for PJK.[59,72-74] Conversely, Ha et al[75] found no association between UIV location (proximal vs distal thoracic) and risk of PJK. Interestingly, they showed that the mechanism of failure differed by UIV location. Compression fracture was the most common mechanism for distal thoracic failure, whereas subluxation was more frequent in the upper thoracic spine. Given the increased risk of PJK associated with preoperative thoracic hyperkyphosis and the propensity for surgeons to select a more proximal UIV in the presence of hyperkyphosis, studies evaluating the impact of UIV location on PJK rate are likely confounded. Indeed, the aforementioned studies did not adequately control for preoperative TK. Nevertheless, assessment of preoperative TK is an important consideration in surgical planning in the treatment of ASD.

MANAGEMENT

Nonoperative Treatment

Clinical guidelines for the nonoperative management of age-related hyperkyphosis do not currently exist. Potential modalities include exercise-based interventions, spinal orthosis, postural taping, and pharmacological therapy. Prospective cohort studies have demonstrated a modest improvement in thoracic hyperkyphosis with exercise-based interventions.[76-78] However, randomized trials on the efficacy of such interventions have yielded conflicting results. Although some have demonstrated improved physical functioning[79] and spine extensor muscle strength,[80] others have not shown any difference in back strength[23] or kyphosis angle.[23,81] It has been suggested that flexion exercises increase the risk of fractures in those with underlying osteoporosis and, hence, extension exercises should be used instead.[82]

A single randomized controlled trial evaluating the effect of bracing on kyphosis was conducted in 62 older women with osteoporosis and TK of 60 degrees or greater. Wearing the brace (Spinomed; Medi, Whitsett, North Carolina) 2 hours a day for 6 months resulted in a decrease in kyphosis by 11% and was associated with increased spinal extensor muscle strength and improved standing height.[83] Despite the apparent efficacy of this orthosis, the benefit and potential consequences of passive bracing over the long term are unclear. Similarly, postural taping has been shown to reduce kyphosis in the immediate term,[84] but its long-term efficacy has not been investigated.

Elderly individuals with hyperkyphosis are commonly treated for concomitant osteoporosis with medications to improve bone density. These medications, which include antiresorptive (bisphosphonate) and bone-building agents, are effective in preventing vertebral compression fractures, but have not been demonstrated to improve hyperkyphosis. In a trial of 4432 postmenopausal women with osteopenia or osteoporosis treated with alendronate, there was no decrease in the progression of kyphosis over 4 years compared to placebo.[85]

FIGURE 1. *Standing long-cassette radiographs of a 72-year-old female with severe back pain and inability to stand upright show degenerative kyphoscoliosis.* **A**, *preoperative lateral view demonstrates severe sagittal malalignment with sagittal vertical axis of 18 cm. Thoracic kyphosis of 66 degrees was present, necessitating a proximal thoracic upper instrumented vertebra.* **B**, *postoperative lateral radiographs following T4 to ilium instrumented fusion with multiple thoracic and lumbar Smith-Petersen osteotomies demonstrates improved global alignment with reduction of thoracic hyperkyphosis.*

Operative Treatment

Surgery to correct isolated hyperkyphosis in the elderly is not typically recommended due to the high prevalence of osteoporosis and other medical comorbidities that result in an unfavorable risk-benefit ratio. Based on 2 randomized controlled trials and a meta-analysis thereof, less invasive interventions including vertebroplasty and kyphoplasty do not seem to provide significant improvement in pain relief as compared to sham procedure in the treatment of vertebral fractures.[86-88] Please see "Management of the Elderly With Vertebral Compression Fractures" in this special issue for further discussion.

Surgery may be considered for hyperkyphosis when there is intractable pain, severe disability, significant pulmonary function impairment, or progressive neurological deficits. This is typically performed through a posterior-only approach, which carries the advantage of decreased blood loss and shorter surgical times as compared to anterior or combined approaches. Pedicle screw fixation combined with multiple Smith-Petersen osteotomies

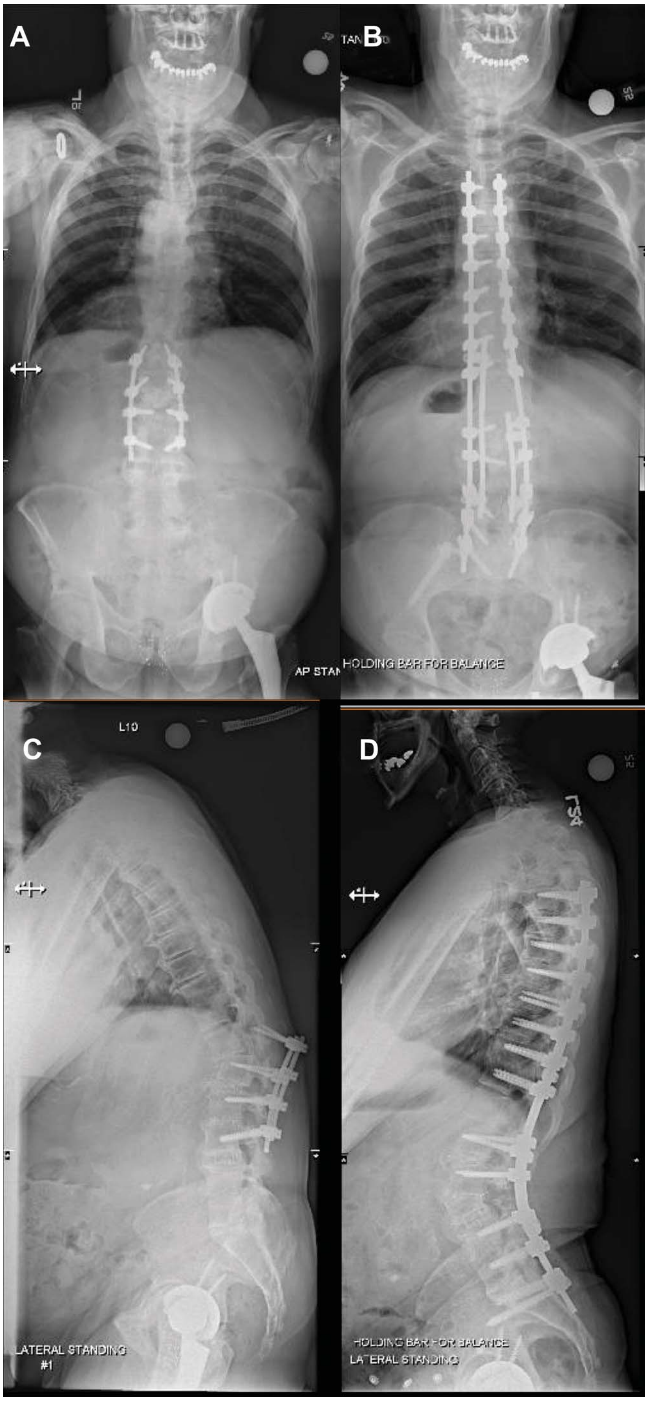

FIGURE 2. *Case of a 75-year-old man with multiple previous spine surgeries who presented with severe pain, inability to stand, and prominent instrumentation. The patient was neurologically intact on presentation. Preoperative radiographs (***A** *and* **C***) demonstrate proximal junctional kyphosis and instrumentation failure (screw pull-out) at the upper instrumented vertebra (T12). The patient underwent T4-ilium posterior instrumented fusion, L3 extended pedicle subtraction osteotomy, and multiple Smith-Petersen osteotomies to achieve the correction shown in postoperative radiographs* **B** *and* **D**.

yields satisfactory curve correction with a low incidence of loss of correction.[89,90] Focal, severe angular deformity may necessitate 3 column osteotomy, typically performed at the apex of kyphosis, to successfully restore alignment.[46,91]

Selection of the proximal fusion level should adhere to the principles outlined above regarding proximal vs distal thoracic UIV. In treating isolated thoracic hyperkyphosis, the UIV must include all kyphotic segments and should be symmetric with the construct below the apex. As stated above, one should avoid stopping at the physiological apex of thoracic kyphosis (T5-8), as this carries an increased risk of PJK. To guide selection of distal fusion level in the operative management of thoracic hyperkyphosis, Cho et al[92] introduced the concept of the sagittal stable vertebra (SSV). They defined the SSV as the most proximal lumbar vertebral body that is contacted by a vertical line drawn from the posterior-superior corner of the sacrum. In 31 patients with thoracic hyperkyphosis who underwent long segment posterior instrumentation and fusion, the authors identified a significantly higher rate of distal junctional disease in patients in whom fusion stopped at the first lordotic vertebra as compared to the SSV. They concluded that the fusion should extend to the SSV to prevent distal junctional kyphosis.[92] Surgical management of thoracic hyperkyphosis that occurs as a component of spinal deformity is discussed in "Degenerative Spinal Deformity."

Recommendations

Hyperkyphosis may be associated with adverse health conditions, pain, and decreased function. As such, it should be recognized and treatment initiated to minimize its progression and mitigate the accompanying negative consequences. Nonoperative management should be considered first line. Patients should have a comprehensive assessment for osteoporosis and, if indicated, appropriate therapy instituted to optimize bone mineral density and reduce fracture risk. Exercise-based treatments focusing on postural alignment, strengthening back extensor muscles, and maintaining flexibility should be initiated. There are limited data showing reliable benefit of other nonoperative options (spinal orthosis, postural taping). Vertebroplasty and kyphoplasty are options for management of severe pain associated with vertebral fractures in select patients; however, data supporting their efficacy is lacking. Surgery may be considered for patients with severe pain and disability associated with hyperkyphosis and should adhere to principles of deformity correction outlined above. Such patients should undergo a thorough preoperative evaluation to characterize their surgical risk in the context of their medical comorbidities, bone quality, and overall health status.

CONCLUSION

Kyphosis is common in older individuals and, with the changing demographics in North America, its prevalence is expected to increase. Hyperkyphosis is associated with vertebral fractures (both as a cause and effect), impaired physical function, decreased quality of life, and increased mortality. Current evidence suggests a role for nonoperative therapies in reducing kyphosis and delaying its progression. Isolated hyperkyphosis in the elderly is rarely treated surgically; however, increased thoracic kyphosis as a component of global spinal deformity has important implications for surgical patient selection and operative planning.

Disclosures

Dr Shaffrey is a consultant or stockholder, receives royalties, or has patents with Biomet, Nuvasive, Stockholder K2M, and Stryker. Dr Lenke has received grant monies from Axial Biotech and DePuy Synthes Spine. Dr Lenke shares numerous patents with Medtronic (unpaid). He is a consultant for Medtronic (monies donated to a charitable foundation). He was a consultant for DePuy Synthes Spine, K2M during the past 3 years. He receives substantial royalties from Medtronic and modest royalties from Quality Medical Publishing. Dr Lenke also receives or has received reimbursement related to meetings or courses from AOSpine, Broad-Water, DePuy Synthes Spine, K2M, Medtronic, the Scoliosis Research Society, the Seattle Science Foundation, and the Spinal Research Foundation. He is a past president of the Scoliosis Research Society. He is an Orthopaedic Research & Education Fund (OREF) board member, on the Associate Editorial Board of *Spine*, on the Editorial Board of the *Journal of Spinal Disorders and Techniques and Scoliosis*, on the Professional Advisory Board of Backtalk and the Scoliosis Association, on the Associate Board of the *Journal of Neurosurgery: Spine*, *The Spine Journal*, an Associate Editor for iscoliosis.com and spineuniverse.com and Deputy Editor of *Spine Deformity*, all of which are unpaid positions. Dr Harrop receives financial support from Asterias, Bioventus, and Tejjin; has stock in Axiomed; is a consultant for Depuy Synthes and Geron; is an unpaid consultant for Ascubio; is on the executive board of *Spine Universe*, *CNS* quarterly, and Congress of Neurological Surgeons (CNS); is a board or committee member for cervical Spine Research Society (CSRS), Peripheral Nerve Society (PNS), Jefferson University Physicians, and Lumbar Spine Research Society (LSRS). Dr Smith is a consultant or receives honorarium or royalties from Biomet; is a consultant, honorarium, with Nuvasive; is a consultant with Cerapedics; receives honorarium from K2M; receives research grants from DePuy Synthes and Arbeitsgemeinschaft für Osteosynthesefragen Spine North America (AOSNA); and receives research support from Arbeitsgemeinschaft für Osteosynthesefragen Spine North America (AOSNA). The other author has no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

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