

# Spinal Disorders as a Cause of Locomotive Syndrome: The Influence on Functional Mobility and Activities of Daily Living

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**Abstract** Spinal disorders are one of the most common causes of locomotive syndrome because the spine plays an important role as the locomotive organs which provide the three functions: body frames, junction sites, and motors/regulators. Previous reports have shown that low back pain or leg pain and/or intermittent claudication due to spinal stenosis in lumbar spine disorders, gait abnormality due to degenerative cervical myelopathy in cervical spine disorders, and trunk imbalance due to adult spinal deformity have negative effects on walking speed, walking distance, and movement ability, thereby increasing the risk of falling. Patients for whom conservative managements have failed are considered for surgical treatment. However, degenerative changes on radiographical examinations are sometimes observed in asymptomatic elderly people, which suggests that degenerative changes are not always the cause of pain or disability in the elderly, particularly non-specific low back pain and neck pain. Therefore, the evidence is insufficient regarding how effective surgical treatments can be for improving walking and movement ability and social participation of elderly patients. In addition to better evidence for the efficacy of various treatments, more attention concerning checkups and prevention of locomotive syndrome are urgently needed in aging populations.

**Keywords** Locomotive syndrome · Lumbar spine · Cervical spine · Adult spinal deformity · Activities of daily living

## Introduction

The proportions of older people in the populations of many countries have been increasing [1]. One of the physical problems of the elderly is musculoskeletal disorders. When older persons cannot perform activities of daily living (ADL) by themselves because of musculoskeletal disorders, they require assistance or nursing care. Therefore, increases in the number of elderly people in a population increases the human and social burdens on society, which must be met in a timely manner. Therefore, to increase awareness in the general public about musculoskeletal disorders in the elderly and the importance of measures to address the associated problems, the Japanese Orthopaedic Association has issued a guidance titled “Locomotive Syndrome” in 2007 and defined the syndrome as “conditions under which the elderly have been receiving care services or high-risk conditions under which they may soon require care services due to problems of the locomotive organs” [2–4]. In a survey in Japan, it was reported that the prevalence of locomotive syndrome remained <10 % until the 1960s but rapidly increased to 16.3 % in the 1970s [5].

The dysfunction of locomotive organs that act as body frames, junction sites, and motors/regulators can cause locomotive syndrome [4]. The spine is one of the most important organs involved in locomotive syndrome because it provides the functionality described above. Spinal malalignment due to osteoporotic vertebral fracture or degenerative (kypho-)scoliosis can impair the body frame functionality of the spine, degeneration of facet

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joints or intervertebral discs can impair junction site functionality, and spinal canal stenosis associated with degeneration can cause neurological deficit, which can impair the motor/regulatory functionality of the spine (Table 1). A decrease in walking ability can result from these spinal dysfunctions in combination or alone. Talaga et al. [6] reported that 56 % of patients with degenerative spinal disorders have experienced difficulty in performing social activities and had psychological problems, such as a sense of inferiority (54 %) and feeling lonely (24 %). Thus, degenerative spinal disorders can affect not only ADL but also social participation of the elderly.

This review discusses spinal disorders as a cause of locomotive syndrome and focuses on the relationship between spinal disorders and ADL, particularly with respect to walking or movement ability.

## Lumbar Disorders

### Low Back Pain (Degenerative Lumbar Spondylosis, Including Non-specific Low Back Pain)

#### Etiology

Many people worldwide suffer from low back pain (LBP). In Japan, LBP is the most frequent subjective symptom in Japanese males and the second most frequent in females, with a prevalence of 92.2 persons per 1000 in males and 118.2 persons per 1000 in females [7]. Hoy et al. [8, 9] showed that the point prevalence and 1-month prevalence of LBP were 11.9 and 23.2 %, respectively; the incidence of LBP increased with age up to 60–65 years of age, and the highest prevalence was seen among female individuals and those aged 40–80 years. Dionne et al. [10] reported

that the prevalence of severe forms of LBP increased with age. Muraki et al. [11] reported in their nationwide cohort study (2288 participants) that the prevalence of LBP was 28.8 % in people  $\geq 60$  years old. In Swedish multicenter prospective observational study of 3009 male participants between 69 and 81 years of age, Ghanei et al. [12] reported that the 1-year prevalence of LBP was approximately 50 % in older males. Although degenerative findings are common in imaging studies in the elderly, it is also well known that these degenerative findings are not necessarily associated with LBP [13–23]. Dayo et al. [24] reported that the cause of chronic LBP could not be identified by various imaging studies in 85 % of cases, and LBP without any evident cause is commonly referred to as non-specific LBP. Muraki et al. [11] investigated the relationships between lumbar spine degenerative X-ray findings and LBP, but only disc space narrowing in females was associated with LBP. Teraguchi et al. [25] reported that intervertebral disc degeneration on magnetic resonance imaging (MRI) was seen in 90 % of people  $\geq 50$  years old in a community-dwelling cohort with 975 participants. In addition, they showed that intervertebral disc degeneration by itself was not associated with LBP, but the combination of intervertebral disc degeneration and end plate signal change on MRI was highly associated with LBP [26].

#### Association with Locomotive Syndrome

Although many researchers have suggested that abnormal findings on imaging examinations of the lumbar spine are not strongly associated with LBP, walking and movement ability of patients is limited by LBP regardless of the cause of the pain. Hirano et al. [27] investigated the association between LBP and patient-based quality of life (QOL) outcomes in 386 community-dwelling people  $\geq 51$  years

**Table 1** Relationships between the roles of locomotive organs and spinal disorders in the elderly

Roles as locomotive organs	Disorders associated with locomotive syndrome
<i>Lumbar spine</i>	
Body frames	Osteoporotic vertebral fracture
Junction sites	Degenerative spondylosis, non-specific low back pain
Motors/regulators	Lumbar spinal stenosis
<i>Cervical spine</i>	
Junction sites	Degenerative spondylosis, non-specific neck pain
Motors/regulators	Degenerative cervical myelopathy (cervical spondylotic myelopathy, ossification of the posterior longitudinal ligament or ligament flavum, calcification of the ligament flavum)
<i>Thoracic spine</i>	
Body frames	Osteoporotic vertebral fracture
Motors/regulators	Thoracic spondylotic myelopathy, ossification of the posterior longitudinal ligament or ligament flavum
<i>Whole spine</i>	
Body frames	Adult spinal deformity

old and concluded that the patients with LBP had lower scores for QOL outcomes regarding lumbar function, walking ability, social life function, and mental health. In addition, fear of LBP can lead to restricted ADL. Sions et al. [28] analyzed the association of fear-avoidance beliefs with disability, physical health, and falling in older community-dwelling people. They showed that pain intensity, assistive device use, and fear-avoidance beliefs were associated with LBP-related disability, and fear-avoidance beliefs were also associated with falling. There are several reports about the relationship between LBP and obesity in the elderly. Vincent et al. [29] investigated the relationship between walking ability and kinesiophobia or fear-avoidance beliefs in older obese participants and showed that kinesiophobia was associated with the intensity of LBP and lower Oswestry Disability Index scores but not with walking distance. In addition, they showed that gait base of support increased, double-support time increased, and daily step numbers decreased depending on the severity of obesity in older patients with LBP [30]. Ono et al. [31] reported an association between metabolic syndrome, including obesity, and LBP in their community-based Japanese cohort study among 1395 participants. According to this report, the risk ratio for metabolic syndrome in subjects with LBP compared with that in those without LBP was 1.5 in women, whereas there was no relationship between metabolic syndrome and LBP in men. These results indicate that LBP is also associated with metabolic syndrome as well as locomotive syndrome.

### *Treatments*

For specific LBP, conservative treatments and surgical treatments for the causes of pain are both effective. However, for non-specific LBP, conservative treatment is the first choice, and surgical treatment is rare because abnormal findings in imaging studies are not always associated with LBP [32]. Many researchers have stated that physical therapies and psychosocial approaches, including multidisciplinary approaches, were effective for non-specific LBP [33–36]. However, it is still unknown whether the same approach can be applied to the elderly or should be modified according to age, and the degree to which these approaches affect the walking and movement ability of the elderly with LBP is also unknown [37, 38].

## **Lumbar Spinal Stenosis**

### *Etiology*

Degenerative lumbar spinal stenosis (LSS) is a condition in which the neural elements are compressed in the spinal canal by static factors and/or dynamic factors. The static

factors are bulging of intervertebral discs and hypertrophy of the ligament flavum or facet joints secondary to degenerative changes in the lumbar spine. The dynamic factor is instability of mobile segments due to dysfunction of intervertebral discs and/or facet joints. LSS can cause leg pain and motor weakness of the lower extremities. One of the characteristics of gait disturbance in LSS is intermittent claudication, which deteriorates while standing and walking and improves during lumbar spine flexion. Symptomatic LSS directly affects patients' walking and moving ability; therefore, several large population-based cohort studies have been conducted since locomotive syndrome was noted in Japan. In their cross-sectional study, including 938 participants with a mean age of 66.3 years, Ishimoto et al. [39] found that 77.9 % of participants had more than moderate central stenosis of the lumbar spinal canal and 30.4 % had severe central stenosis on MRI. They also reported that the prevalence of LSS increased with age, but only 17.5 % of the participants with severe central stenosis were symptomatic. Furthermore, in the same cohort in their study, they showed that the overall prevalence of symptomatic LSS was 9.3 %, and the prevalence increased with age, particularly in women [40]. Yabuki et al. [41] estimated the prevalence of LSS using a diagnostic support tool in their population-based study of 2666 subjects with a mean age of 60 years, and they showed that the prevalence of symptomatic LSS was 5.7 %, increased with age, and was particularly high in subjects aged 70–79 years (>10 %), irrespective of sex. In Europe, a large community-dwelling cohort study from Sweden that included 3009 old men found that 22 % of the participants had sciatic pain [12]. The prevalence of LSS is estimated to increase by nearly 60 % by 2025 as a result of the increasing number of older people in the population [42].

### *Association with Locomotive Syndrome*

Regarding locomotive syndrome, several studies have been reported on the relationships between LSS and walking and movement ability or ADL. Sigmundsson et al. [43] examined the relationship between the cross-sectional area of the dura mater on MRI and walking distance for patients who had been scheduled for surgical treatment for LSS, but no correlation was observed. Kim et al. [44] compared the walking ability of patients with LSS and that of patients with osteoarthritis of bilateral knees using functional mobility tests and showed that patients with LSS spent significantly more time performing the 6-m walk test and sit-to-stand test than patients with osteoarthritis of bilateral knees. They concluded that symptomatic LSS had a risk of falling comparable to that of patients who had osteoarthritis of bilateral knees. Winter et al. [45] investigated the

walking status of patients who had been scheduled for surgeries for LSS and osteoarthritis of the knee or hip using a uniaxial accelerometer for a week. They showed that the numbers of gait cycles per day and per minute were significantly lower in patients with LSS and osteoarthritis of the knee or hip than those in healthy control individuals. Moreover, walking dysfunction was more apparent in patients with LSS than in patients with osteoarthritis of the knee or hip. In their study among community-dwelling participants, Ishimoto et al. [40] reported that the results of the 6-m walking test, chair-standing test, and one-leg standing test were worse in patients with symptomatic LSS than those in participants without LSS. Tomkins–Lane et al. [46] analyzed the walking ability using a pedometer in subjects 55–80 years old and found that the patients with LSS had decreased 1-week walking distance, daily step counts, and 15-min walking distance and velocity than asymptomatic subjects, but there were no significant differences in the walking parameters between the patients with LSS and LBP. In contrast, Ghanei et al. [12] reported that patients with both LBP and sciatic pain or neurological deficit had more limitations in ADL (bending down to pick up light objects, lifting a 5-kg object from the floor, putting socks on either foot, and getting in or out of the front seat of a car) than patients with only LBP. Similarly, in their cross-sectional study of 2673 patients, Kongsted et al. [47] reported that patients with leg pain in addition to LBP had a more severe condition, with limitations not only in ADL but also in work participation, than patients with LBP only. Tong et al. [48] compared pain severity using the visual analog scale, 15-min walk test, 7-day walking distance, Quebec Back Pain Disability Scale, and Pain Disability Index among patients with LSS >55 years old, those with LBP, and asymptomatic participants. They showed that all of the abovementioned variables were worse in patients with LSS than in asymptomatic seniors, but there was no significant difference in the 15-min walk test results and 7-day walking distance between the patients with LBP and asymptomatic seniors. The researchers suggested that it was more difficult to compensate for walking disability in patients with LSS than in patients with LBP only.

### Treatments

Good outcomes of patients of LSS with mild or moderate symptoms have been reported in approximately half of the cases; however, in patients with severe symptoms, outcomes have been poor for conservative treatments [49–51]. On the other hand, LSS rarely deteriorates rapidly in the natural course [52]. Surgical treatment is performed if symptoms are resistant to conservative treatments. Ammendolia et al. [53] concluded in their review that there was no clear evidence of non-operative treatment for LSS

with neurogenic claudication, although a meta-analysis of two trials comparing direct decompression, with or without fusion, with multimodal non-operative care found no significant difference in function at 6 months and 1 year; however, a significant difference was found favoring decompression at 24 months. A systematic review for the management of LSS in the elderly (>65 years old) recommended that elderly patients should not be excluded from surgical intervention for symptomatic LSS because the majority of elderly patients exhibit significant symptomatic improvement, with overall benefits observed for pain and disability [54]. Shabat et al. [55] also reported that surgery for patients with LSS who were  $\geq 80$  years old did not show increases in associated morbidity and mortality, and most of the patients benefited from the surgery in terms of reduction in pain and increased ADL and walking ability. A systematic review investigating the efficacy of treatments on walking ability in neurogenic claudication with LSS showed that prostaglandins, gabapentin, or methylcobalamin improved walking distance, epidural injections improved walking distance up to 2 weeks, and decompression surgery was no better than non-operative treatment in improving walking ability; however, current evidence for surgical and non-surgical treatment to improve walking ability is of low and very low quality [56].

## Cervical Disorders

### Neck Pain (Degenerative Cervical Spondylosis, Including Non-specific Neck Pain)

#### Etiology

In their community-dwelling cohort study of 1527 participants, March et al. [57] reported that 36 % of men and 40 % of women  $\geq 65$  years old experienced neck pain. Palazzo et al. [58] reported that the prevalence of neck pain was approximately 12 % in people >60 years old in a cohort study of 29,931 people. Palacios-Ceña et al. [59] reported that the prevalence of neck pain was 8–9 % in a population-based national study of 43,072 people. Some variation in the prevalence of neck pain exists, but many reports have shown that the prevalence increased with age.

Degenerative changes in the cervical spine as well as in the lumbar spine can occur in all people. Gore et al. [60] reported that 95 % of males and 70 % of females who were asymptomatic and 60–65 years old showed degeneration in the cervical spine on plain radiographs. In MRI studies, Nakashima et al. [61] found that 87.6 % of 1211 asymptomatic subjects aged 20–79 years had disc protrusion, and Matsumoto et al. [62] reported that 86–89 % of 497 asymptomatic subjects >60 years old had disc

degeneration. The prevalence of degenerative changes on imaging studies increased with age [60–62] and differed greatly from the prevalence of neck pain. These results suggest that degenerative changes in the cervical spine do not always cause neck pain. Kumagai et al. [63] investigated the association between radiographical cervical spine findings and neck pain in 762 community-dwelling volunteers and showed that there was no association between the sagittal alignment of C2–C7 and neck pain, but there was a significant correlation between the degree of cervical spine degeneration and neck pain in females. Reported correlations between abnormal intensity of vertebral bodies in the cervical spine and neck pain are controversial [64, 65]. Neck pain without any evident cause is referred to as non-specific neck pain [66].

#### *Association with Locomotive Syndrome*

Regarding the relationship between neck pain and physical health-related QOL (HRQOL) in large population-based cohort studies, Nolet et al. [67] reported that neck pain was associated with worse physical HRQOL, and Rezai et al. [68] reported that neck pain was weakly associated with physical HRQOL and that comorbid conditions accounted for most of the association between neck pain and physical HRQOL.

In a study that focused on neck pain and walking ability of older subjects, Poole et al. [69] analyzed the association of neck pain with balance and gait in subjects aged 65–82 years. They showed that older subjects with neck pain had poorer balance, slower gait speed and cadence in the head turn condition, and longer gait cycle duration than healthy controls. Uthaknup et al. [70] also reported that older subjects with neck pain had greater sensory motor disturbances and were afraid of falls more than healthy controls despite no difference in gait speed and step numbers. These results suggest that neck pain can cause restriction of walking or social participation because of poor space perception and balance rather than because of impairment of lower extremities.

#### *Treatments*

For specific neck pain, conservative treatments and surgical treatments for the causes of pain have both been shown to be effective. However, for non-specific neck pain, conservative treatment is the first choice and surgical treatment is rare because abnormal findings in imaging studies have not always been associated with neck pain. As non-specific LBP, medications, physical therapies, and psychosocial approaches have been performed for non-specific neck pain, but current evidence for treatments of non-specific neck pain is still of low quality [71].

## **Cervical Myelopathy**

### *Etiology*

The most common cause of cervical myelopathy in the elderly is compression of the cervical cord because of developmental spinal canal stenosis and degeneration of the cervical spine, such as disc bulging, hypertrophy of the ligament flavum, and/or segmental instability (cervical spondylotic myelopathy, CSM). Ossification of the posterior longitudinal ligament or ossification or calcification of the ligament flavum can also cause compression of the cervical cord; therefore, Nouri et al. [72] proposed a new term, “degenerative cervical myelopathy” to comprise spondylosis as well as ligamentous calcification and ossification. The prevalence of cervical myelopathy due to degenerative conditions varies by race and region [73–77]. It has been estimated that degenerative spinal conditions encompass 59 % of non-traumatic spinal cord injuries in Japan, 54 % in the USA, 31 % in Europe, 22 % in Australia, and 4–30 % in Africa [78]. In North America, the incidence and prevalence of cervical myelopathy due to degenerative compressive pathologies have been estimated to be at least 41 and 605 per million, respectively [72]. Nagata et al. [79] conducted a population-based cohort study with 959 participants and investigated the prevalence of cervical myelopathy and the relationship between cervical myelopathy and spinal canal diameter. They found that cervical spinal canal diameter narrowed with increasing age, and the prevalence of cervical cord compression and increased signal intensity in the spinal cord on MRI and symptomatic myelopathy were higher in participants with narrow spinal canal. The prevalence of cervical myelopathy was 10.1 % in participants with spinal canal diameters of  $\leq 13$  mm. In the elderly, traumatic cervical spinal cord injury without major fracture or dislocation is a common cause of spinal cord injury, with a reported incidence of 10–16 % in North America [80]. In Japan, the annual incidence of traumatic cervical spinal cord injury without major fracture or dislocation has been estimated to be 3000–10,000 persons per year and is increasing dramatically in Japan [80, 81].

### *Association with Locomotive Syndrome*

In a study on the relationship between cervical myelopathy and dysfunction of walking, Nishimura et al. [82] reported that the gait velocity and step length decreased and step angle and step width increased as compensation for unstable gait as myelopathy progressed. Malone et al. [83] also reported that patients with CSM walked significantly more slowly with shorter stride lengths and longer double-support duration than healthy controls and decreased in range of motion at the hip, knee, and ankle joints.

Furthermore, they evaluated the muscle activity of lower extremities by electromyography in patients with CSM and showed that patients with CSM had prolonged duration of activation of the biceps femoris and tibialis anterior in addition to prolonged co-activation of the rectus femoris and biceps femoris compared with healthy controls [84]. Nagata et al. [85] investigated the relationship between cervical cord compression on MRI and locomotive functions in their population-based cohort study with 977 subjects and showed that cervical cord compression was associated with poor results of the grip and release test, 6-m walking time, step length, and chair-stand time.

A recent review stated that the natural course of CSM is highly variable and not well defined [86, 87]. Nakamura et al. [88] reported that the function of lower extremities evaluated by Japanese Orthopaedic Association (JOA) score was improved in 57 % and remained stable in 39 % of patients with CSM who received conservative treatments, although 30 % of them converted to surgical treatment. Yoshimatsu et al. [89] reported that the JOA score deteriorated in 19.6 % of patients with CSM in the course of an average 36 months of conservative treatments. Sumi et al. [90] also showed that deterioration in myelopathy was observed in 25.5 % of patients with CSM, whereas 74.5 % maintained mild extent myelopathy without deterioration through the average follow-up period of 94.3 months. Ohshima et al. [91] recently reported that Kaplan–Meier survival analysis revealed that 82 or 56 % of patients with CSM did not require surgery 5 or 10 years after the initial treatment, respectively. However, few studies have examined in detail the walking and movement ability in the natural course of CSM, and the prognostic factors of CSM determined in clinical manifestations or imaging studies remain unclear.

### Treatments

Surgeries for decompression of the spinal cord are performed in cases in which symptoms are resistant to conservative treatments or progress in patients with CSM. Singh et al. [92] showed that both walking time and the number of steps taken in the 30-m walking test were significantly worse in preoperative patients with CSM than in controls, and these walking parameters improved postoperatively. Furthermore, in another study, they showed that patients with CSM gained a significant recovery of function evaluated by the 30-m walking test, which was maintained after surgery; non-operated patients continued to show decreased walking ability [93]. Moorthy et al. [94] also showed that walking speed, stride length, percentage of single-limb support time, vertical and backward ground reaction forces, and range of motion at the hips improved postoperatively in patients with CSM and indicated that the

stability of gait as well as greater flexibility in the knee improved after decompression of the spinal cord. Yoshida et al. [95] concluded that decompression surgery can improve locomotor ability and decrease nursing care requirements among elderly patients with CSM, even though the perioperative sum of gait and stair items of the functional independence measure (FIM) and JOA score were significantly lower and cerebral infarction and previous lumbar surgery were more frequent as neurological comorbidities in patients with CSM >75 years old than in those <75 years old. Karpova et al. [96] showed that the transverse area of the spinal cord at the site of maximal compression on MRI was negatively correlated with preoperative and 1-year postoperative results of the 30-m walking test. Kadaňka et al. [97] conducted a 10-year prospective randomized study to compare conservative and operative treatments for CSM with mild or moderate symptoms. They showed that there was no difference in walking ability between conservative and operative treatments by the evaluation of video recordings of ADL and 10-m walking test results. Consequently, most authors have reported that surgical treatments for CSM had positive effects on walking ability, at least in the short or middle term. On the other hand, the long-term results of surgical treatments for locomotive function remain unclear, and further studies are needed.

## Thoracic Disorders

A large population-based cohort study showed that degeneration of intervertebral discs on the MRI was very frequently observed and developed with age in the thoracic spine: 79 % of men and 89 % of women  $\geq 80$  years old had degeneration of the intervertebral discs in the thoracic spine [25]. However, compressive myelopathy due to degeneration is rarer in the thoracic spine than in the cervical spine. Therefore, locomotive syndrome caused by compressive myelopathy due to thoracic degeneration is very rare.

On the other hand, locomotive syndrome caused by osteoporotic vertebral fracture in the thoracic spine is common. This topic is discussed further in the other part of this issue.

## Whole Spine Disorders

### Adult Spinal Deformity

#### Etiology

Adult spinal deformity is defined as an abnormality in alignment, formation, or curvature in one or more areas of

the spine in adults [98]. Both scoliotic deformity on a coronal plane and kyphotic deformity on a sagittal plane are important clinically. Many authors recently have suggested that sagittal malalignment can cause back pain, limitation of ADL, and deterioration of QOL [98–101]. Various factors can cause adult spinal deformity, but degenerative spinal scoliosis or kyphoscoliosis with aging is most frequent [98]. The prevalence of adult scoliosis varies by reports, and the prevalence increases with aging. Kebaish et al. [102] reported that the overall prevalence of scoliosis in 3185 participants was 8.85 % in patients  $\geq 40$  years old, 14.76 % in patients in their 70s, and 21.56 % in patients in their 80s. Schwab et al. [103] also reported that the prevalence of scoliosis was 68 % in healthy adult volunteers  $>60$  years old. In contrast, estimates of the prevalence of degenerative kyphosis in the elderly have ranged from 20 to 50 % [104]. Vertebral compression fractures are the most commonly cited cause of kyphotic deformity, but postural changes, degenerative disc disease, muscular weakness, and genetic influences can also cause kyphotic deformity individually or in combination [98].

#### *Association with Locomotive Syndrome*

Dubousset et al. [105] described the theory of the “cone of economy” as the relationship between adult spinal deformity, particularly sagittal deformity, and trunk balance. Deviation from a hypothetical cone that extends upward from the feet results in the increased use of postural muscles and compensatory mechanisms in an attempt to restore global alignment at the cost of increased energy expenditure [98, 105]. These compensatory mechanisms affect the function or range of motion of hip and knee joints, and malalignment of the hip or knee joint can occur with sagittal deformity [106]. Various thresholds of parameters for coronal or sagittal imbalance have been proposed as indices for poor ADL or HRQOL [98]. A mismatch between spinal alignment and pelvic morphology recently has been considered to be critically important for pain and QOL [98, 106]. Further studies on racial differences in pelvic morphology and appropriate spinal alignment are needed.

There are many reports on the relationship between spinal balance and QOL, but there are few on the relationship between spinal balance and locomotive syndrome, such as walking or movement disabilities. In a community-dwelling cohort study, Hirano et al. [107] stated that a decrease in back muscle strength and an increase in spinal inclination angle might be the most important risk factors for locomotive syndrome and that lumbar kyphosis was an important factor related to back muscle strength and spinal inclination angle in elderly people  $>70$  years old.

Takahashi et al. [108] also reported that participants with lumbar kyphosis had poor basic and instrumental ADL relative to those of normal elderly controls, and trunk deformity reduced outdoor activities. Miyazaki et al. [109] reported that a decrease in lumbar lordosis caused deterioration in the results of gait performance tests, such as maximal walking speed, timed up-and-go test, 10-m obstacle walking time, and 6-min walk distance. Imagama et al. [110] showed that worsened spinal sagittal alignment and poor body balance, poor back muscle strength, and slower gait speed were also significantly associated with falling.

#### *Treatments*

In a recent review article, non-operative management was recommended for patients with mild or non-progressive symptoms, but evidence of its efficacy is limited [98]. The main goal of operative treatment for adult spinal deformity is to restore global spinal alignment, and several reports have indicated that restoration of spinal alignment by operative treatment resulted in better outcomes assessed by various measurement tools (ODI, SRS, NRS, SF-36) than outcomes of conservative treatment [98]. However, the surgery for adult spinal deformity is associated with high invasiveness and a high complication rate [98]. Cho et al. [111] reported that the overall complication rate of posterior corrective fusion surgery for patients with adult spinal deformity with a mean age of 66.6 years was 68 %. Smith et al. [112] also showed that the overall complication rate of surgery for patients with adult spinal deformity aged between 65 and 85 years was 71 %. Therefore, it is quite important to evaluate a patient’s background and physical condition with respect to selection of a treatment method.

#### **Conclusions**

In this article, we outlined the relationship between spinal disorders and locomotive syndrome. LBP itself or leg pain and intermittent claudication due to spinal stenosis in lumbar spine disorders, gait abnormality due to degenerative cervical myelopathy in cervical spine disorders, and trunk imbalance due to adult spinal deformity have negative effects on walking speed, walking distance, and movement ability, thereby increasing the risk of falling. Patients in whom conservative managements have failed are considered for surgical treatment; however, there is insufficient evidence for how much surgical treatments can improve the ability of walking, movement, and social participation of elderly patients. More evidence supporting suitable treatments in addition to checkups and prevention of locomotive syndrome are urgently needed in rapidly aging populations.

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### Compliance with Ethical Standards

**Conflict of interest** Takahiro Makino, Takashi Kaito, and Kazuo Yonenobu declare that they have no conflict of interest.

**Human/Animal Studies** This article does not include any studies with human or animal subjects performed by any of the authors.

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