Anatomical Aspects of the Sacroiliac Joint and Their Clinical Applications

3

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3.1 Arthrokinematics and Osteokinematics

Arthrokinematics is the area of kinesiology which focuses on the motion between articular surfaces of synovial joints. *Osteokinematics* is the area of kinesiology which focuses on geometrical bone movements in a space. Measurement of range of motion in orthopedic medicine is based on osteokinematics (Fig. 3.1). In order to understand sacroiliac joint (SIJ) dysfunction, knowledge of "joint position" and "accessory movement" is required.

Regarding "joint position," there is a close-and a loose-packed position. The maximal congruency of the articular surface pair is the close-packed position. In this position, most ligaments and parts of the joint capsule are pulled taut, which provides stability to the joint. All positions other than the close-packed position are loose-packed positions and every joint has the least-packed position, in which the joint capsule is the most relaxed.

For example, when the knee is fully extended, it is the close-packed position of the knee. This position provides stability for weight bearing when standing and walking (Fig. 3.2). The other positions are the loose-packed positions. The slightly flexed position, which indicates the least-packed position, is unstable but relaxes the knee during the swing phase. This position is, however, weak against the external force and the soft tissue around the joint can be easily damaged [1, 2].

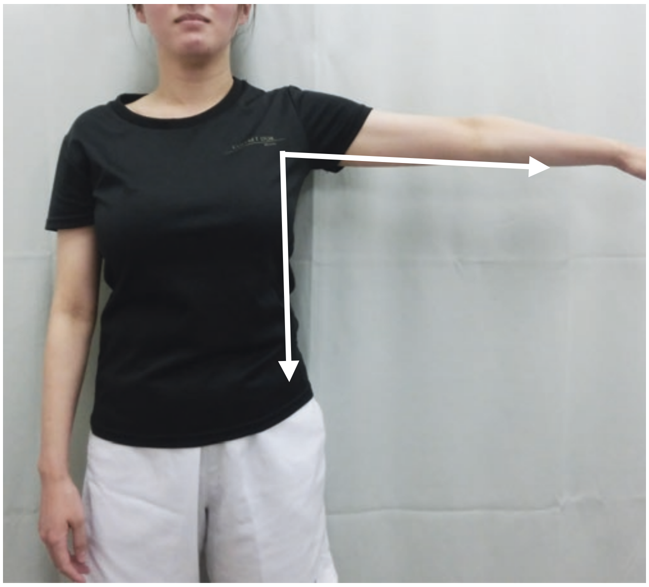

Fig. 3.1 Osteokinematics. Osteokinematics is the area of kinesiology which focuses on geometrical bone movements in a space

These joint positions are determined according to each joint structure in the human body. The collateral ligament of the knee is attached in a posterior femoral condyle manner and becomes tense at the knee extension. The surface of the articular region of the tibia slides backward in a flexed position and generates loosening of the ligament. This position is the loose-packed position [1] (Fig. 3.3).

If humans did not have a close-packed position in a joint position, we would not be able to achieve a stable upright posture when walking, which means that it would require huge amounts of muscle strength and a lot of energy to stand and walk, like a gorilla. In this way, we would not be able to walk for a long distance. Human beings might have started walking out of Africa to the rest of the world when the mechanism of the close-packed position developed. In the standing position, the joint

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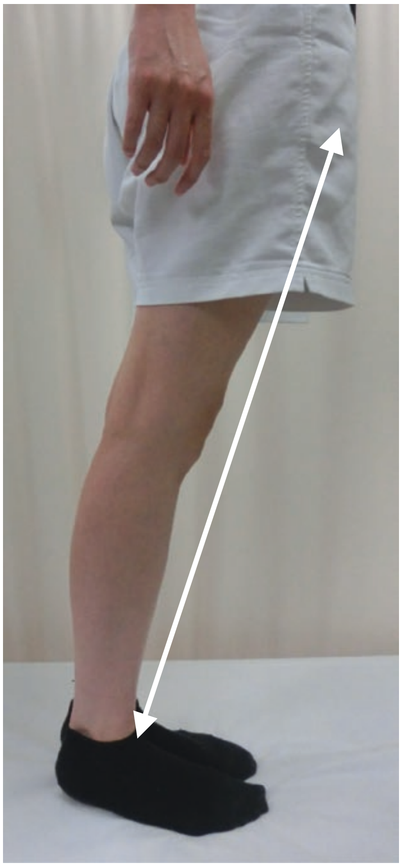

Fig. 3.2 The close- and loose-packed position of a joint. Knee extension and ankle dorsiflexion provide stability for weight bearing when standing and walking

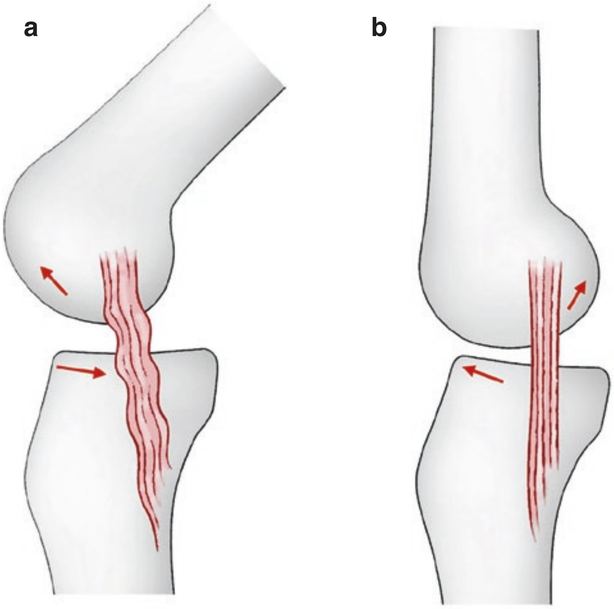

Fig. 3.3 The loose- and close-packed positions of the knee. (**a**) Loose-packed position: the collateral ligament attaches at the back of the medial femoral condyle. The surface of the tibia slides backward in the knee-flexed position, loosening the ligament. (**b**) Close-packed position: the ligament becomes taut with knee extension, securing stability without much muscle strength

position of the knee, hip, SIJ, and spinal facet is close-packed. In the other postures, a joint state is a loose-packed position.

3.2 Bone Movements of the SIJ

3.2.1 Nutation, Counter-Nutation, and Rotation

The SIJ is the synovial joint between the sacrum and the ilium. A part of this joint is the syndesmosis. The joint surface becomes less smooth at age 50 and up [3]. In the standing posture, the sacrum is caught and hanging down between the bilateral iliac bones, and bears the weight of the upper half of the body. For this purpose, the posterior sacroiliac ligaments mainly work for bearing vertical loads.

Kapandji defined nutation as an anterior inferior movement of the sacral promontory. Meanwhile, the iliac bones are approximated, whereas the ischial tuberosities move apart. The movement of counter-nutation involves displacements in the opposite direction. The sacral promontory moves superiorly and posteriorly. Also, the iliac bones move apart and the ischia tuberosities are drawn together [4].

Regarding axial rotation, there are several theories, but there is still disagreement among researchers on the true axis of rotation. Among individuals, there are differences which, in clinical settings, are thought to vary due to posture. SIJ should have the complex movements of rotation accompanying the translation.

The movement of the SIJ cannot be observed visually like other joints in the extremities. Therefore, many researchers have tried to understand complicated SIJ movements. Kissling et al. reported [5] on a three-dimensional stereophotogrammetric method for measuring the mobility of the SIJ. Intraosseous markers were put on for a total of 24 healthy volunteers. It was revealed that the average values for rotation and translation were low, being 1.8°/0.7 mm for males and 1.9°/0.9 mm for females. Individual variability was also found for both the measurements of the rotation and the translation. This study indicated that the opinion “SIJ was an immobile joint” should be rejected.

3.2.2 The SIJ in Standing Posture

The center of gravity line in standing passes through the front of the SIJ’s center of rotation and through the center or posterior of the hip joint [6]. This gravity line and the position of the SIJ and hip joint could generate two rotary movements in the pelvis. The gravity force rotates the sacrum forward at the SIJ, and rotates the pelvis backward

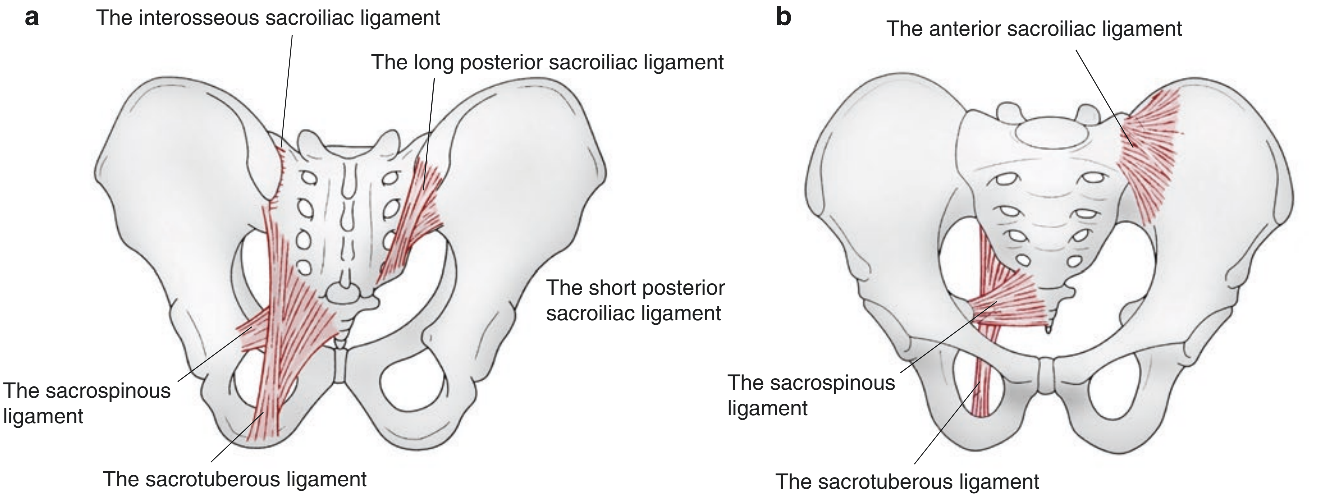

Fig. 3.4 The ligaments around the sacroiliac joint related to the movement of the joint. (**a**) Posterior ligaments. (**b**) Anterior ligaments

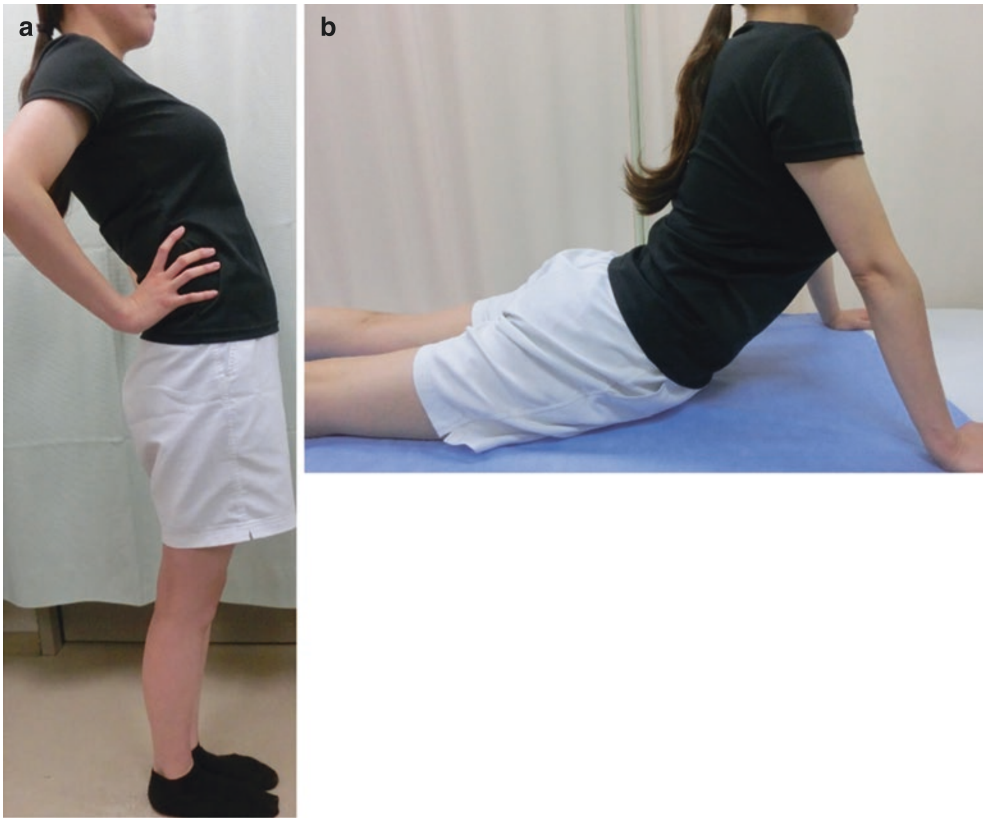

Fig. 3.5 The close-packed position of the sacroiliac joint. (**a**) The position of maximal lumbar extension: This position gives the most stability and is suitable for lifting heavy things. (**b**) Maximal extended position when prone: In this position, the sacroiliac joint cannot move. Pressing on the sacrum in this position can harm the sacroiliac joint

at the hip joint. When the sacrum is rotated forward, the interosseous ligaments are stretched. Caudal tip of the sacrum moves backward and the sacrotuberous ligament and the sacrospinous ligament are stretched [7]. The tension of the sacrotuberous and sacrospinous ligaments stabilizes SIJ (Fig. 3.4). Vleeming reported that the tensile force of the sacrotuberous ligament generated the friction force in the joint surface between the sacrum and the ilium [8]. Standing posture with intensified lumbar lordosis is the most stabilized form for SIJ. This is the close-packed position of the SIJ (Fig. 3.5).

3.2.3 Osteokinematics of SIJ in Walking

In normal walking, pelvic movement is asymmetrical at heel contact. The right coxal bone rotates backward at right heel contact and the left coxal bone rotates forward (Fig. 3.6). The sacrum flexes toward to right lateral direction. The right joint surface approximates at the upper cranial, and the left joint surface approximates at the lower caudal portion. The SIJ dysfunction restricts SIJ movement of the involved side, resulting in claudication. Intermittent claudication is often caused by SIJ dysfunction.

Fig. 3.6 Bone movement of the sacroiliac joint in walking. (**a**) Bone movement of the coxal can be felt by palpating with both hands when walking. (**b**) When walking with maximal lumbar extension, less bone movement can be felt

3.2.4 Osteokinematics of the SIJ in Sitting

The sacrum is in the nutation position when sitting in intensified lumbar lordosis, and is in the counter-nutation position in lumbar kyphosis.

3.3 Articular Neurology and Clinical Applications

Articular neurology [9, 10], which was studied by B. Wyke, is useful when trying to understand synovial joint dysfunction and pain. Four types of joint receptors were identified, and each had a specific role in joint function. Particularly, an understanding of the function of type I and IV receptors is important when applying AKA-H treatments (Table 3.1).

The type I joint receptor, which is similar to the Ruffini corpuscle, is distributed in the peripheral layers of each fibrous capsule of a joint. These kinds of joint receptors are more often distributed in the proximal joints than in the distal joints. In the spine segment, type I receptors are often observed in cervical facet joints. These receptors respond to the mechanical stress on the fibrous capsule with a low threshold, slowly adapting. Type I receptors provide information concerning the static position of a joint by firing constantly. The receptors discharge constantly at frequencies of 10–20 Hz. Wyke described that articular mechanoreceptor reflexogenic effects due to the type I receptors were arthrostatic reflexes. This kind of reflex was expressed as low-grade tonic motor unit activity in muscles in the body in response to the continuous afferent discharge of the type I receptors located in the joint capsules of immobile joints upon tonic fusimotor neurons. However, the function of type I receptors was to increase the tension of not only muscles, but also soft tissues, including the joint capsule and ligaments. This was identified by Dr. Hakata in his clinical settings. These findings showed that the joint dysfunction causes hyper-tension of the soft tissues around the joint. He also discovered that the hypertensive state could be observed in other ipsilateral joints, which were named "*the arthrostatic hyper-reflex chain*" by Dr. Hakata.

The type II joint receptors, which are a Vater-Pacinian-like corpuscle, are distributed in the deep layer of the fibrous capsule. They are located more frequently in the distal joints. They respond with low thresholds, quickly adapting, and fire only on quick changes in joint movements. They do not respond in the static position of a joint at all. This type of joint receptor provides information concerning acceleration and deceleration of joint movement. Arthrokinetic reflexes, which are operated during active or passive joint movement, are produced by the combined effects of the altered discharge of the type I receptors and the brief discharges induced from the type II receptors. Arthrokinetic reflexes involve coordinated facilitatory and inhibitory effects on motor unit activity in the body musculature in response to joint movement.

The type III joint receptors, which are similar to the Golgi end organ, are located in the ligaments of the joints in extremities and in spinal facet joints, and are also located in crucial ligaments in knee joints and ligaments of the head of the femur. They are absent from the longitudinal ligaments and the interspinous ligaments of the vertebrae. They are slowly adapting, and are high threshold mechanoreceptors, which work for monitoring directions of a joint movement. This type of receptor has reflex effects on muscle tone to provide a "braking" mechanism against over movement of the joint.

The type IV joint receptors are the free nerve ending (type IVb) or a plexus (type IVa). These nerve endings are very small myelinated or unmyelinated fibers, which are located in the fibrous joint capsule of the extremities, spinal facet, and temporomandibular. They are also located in the periosteum near a joint, fat pad, and the outer membrane of blood vessels in a joint. They work as nociceptors.

The soft tissues around the joint will become tense due to the responses of the type I receptors. Type I immediately responses against pressure to the bone, ligament, and joint capsules. This type of reaction is called an "arthrostatic reflex." For example, an arthrostatic reflex in an elbow joint, when a physician examines the range of motion (ROM) of passive flexion by using his palm on the distal forearm, the ROM of flexion is larger than when grasping the forearm. When a physician flexes the elbow joint slowly by grasping

Table 3.1 The classification of joint receptors

	Description	Location	Related fiber	Action
Type I	Similar to Ruffini corpuscle	Superficial joint capsule	Small (6–9 μ) myelinated	Slowly adapting low threshold: mechanoreceptor
Type II	Similar to Pacinian corpuscle	Joint capsule (deeper layers) fat pads	Medium (9–12 μ) myelinated	Rapidly adapting low threshold: dynamic mechanoreceptor
Type III	Similar to Golgi end organ	Intrinsic and extrinsic joint ligament	Large (13–17 μ) myelinated	Very slowly adapting. High threshold: dynamic mechanoreceptor
Type IV	Free nerve endings and plexus	Fibrous capsule intrinsic and extrinsic ligaments, fat pads, periosteum	Small (2–5 μ) myelinated and unmyelinated	Non-adapting high threshold: pain receptors

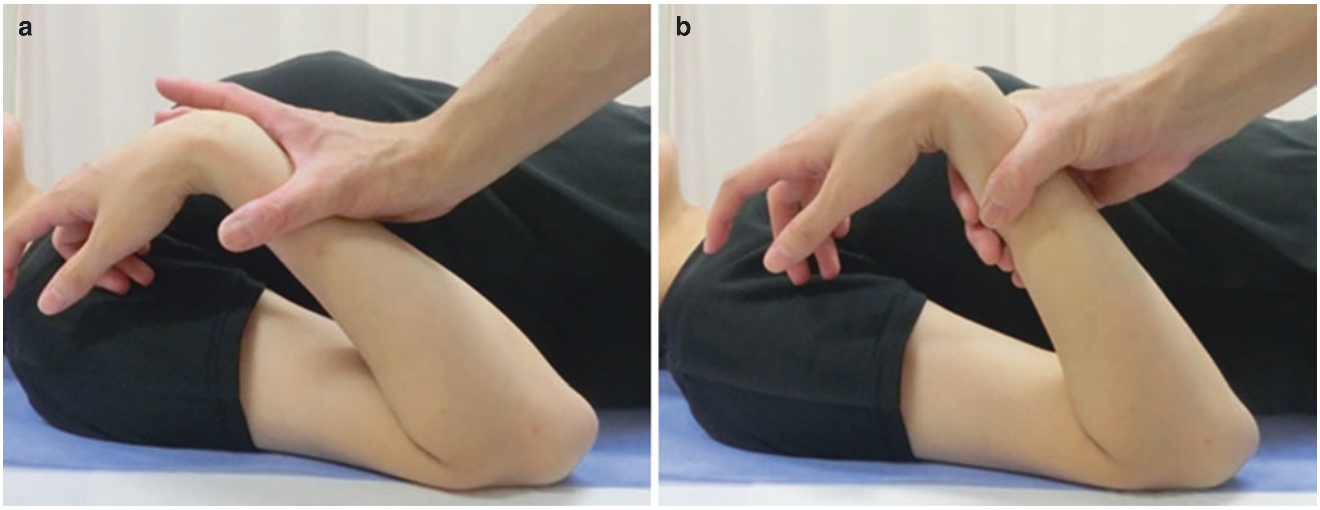

Fig. 3.7 Elbow movement and arthrostatic reflex. (**a**) Passive flexion with the palm on distal forearm can provide 160° flexion of the elbow joint. (**b**) Passive flexion with holding distal forearm elicits the arthrostatic reflex and restricts flexion of the elbow joint

the forearm, end feelings at the elbow joint flexion become stiff, and a resistance force from the joint can be noticed (Fig. 3.7). This experience indicates that the ROM of a joint could decrease when an arthrostatic reflex occurs, because a soft tissue around the joint tenses up. This type of reflex, however, is not elicited when moving the forearm fast.

An arthrostatic reflex is a mechanical reflex, and it could occur with type I responses in every joint. This reflex protects a joint from external forces or stimulations. When doing sports, immediate responses via an arthrostatic reflex are useful. For example, a tennis player's wrist becomes tense at the instant the racket hits the ball, followed by a relaxed swing (Fig. 3.8).

The arthrostatic reflex continues to be active in the case of joint injuries. The reflex influences soft tissues around the joint. Muscle tension increases, and numbness and swelling are also often observed in clinical settings in the remote regions in related ipsilateral upper and/or lower extremities. For example, when an arthrostatic reflex in the SIJ continues for a long time, it could influence ipsilateral knee and/or ankle joints and cause pain and contracture of these joints.

Type IV is the nociceptive receptor which is located in the fibrous capsule, periosteum, fat pad, and outer membrane of blood vessels, but which is absent in articular cartilages. Theoretically, the articular cartilage does not cause joint pain because type IV receptors are not located there. This is why degenerative changes of the joint cartilage of the hip or knee as well as the extent of joint deformity don't correspond to the severity of pain.

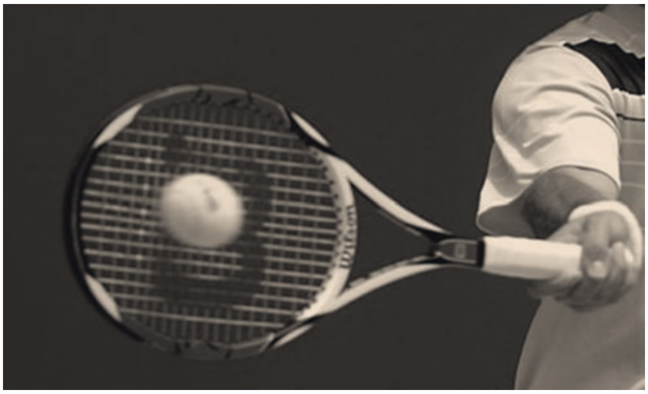

Fig. 3.8 Example of the arthrostatic reflex. Regarding hitting a tennis ball (speed of 200 km/h) with the wrist in dorsiflexed position, when the ball just hits the racket, the joint and soft tissues around the hand tense up by the arthrostatic reflex and the fast ball can be returned

3.4 Accessory Movements of Joints and Clinical Applications

It is important to know joint position to understand joint movement. In the close-packed position, joint surfaces are fully contacted and the joint is stiff and tight. The hip joint is typical of the ball and socket joint, which is maximally congruent in the position of extension and internal rotation. In this position, the high tension of the joint capsule and ligaments causes joint surfaces to come in close proximity to each other. Therefore, elderly people easily suffer from femoral neck fractures when they fall down in this close-packed position. In any other joint position except for the close-packed position, the hip joint can move without restriction because the periarticular soft tissues are loosened. This loosening positions of this joint are useful for swing phases in walking, for instance.

Joint positions such as close and loose are very important for free joint movements. In most kinds of joint structures, curves of the joint surface do not completely resemble each other, even if the joint shape is classified as a convex and concave structure. The fundamental structure of every joint includes loose states in the joint movement. Therefore, in the case of osteoarthrosis, the congruence of the joint surface is lost, and joint movements are restricted. However, a certain joint mobility can be maintained.

As mentioned above, the looseness and tightness of the joint depend on the structure of a joint and ligaments around a joint. In case of the metacarpophalangeal (MP) joint, firm grasping is the close-packed position, and the rest are loose-packed positions (Fig. 3.9). The close-packed position of the MP joint can deliver the “strong punch” to opponents in a boxing match, but are often susceptible to fractures of the metatarsal neck (boxer’s fracture). In the loose-packed position, the MP joint is susceptible to finger sprains and ligament damage (Fig. 3.10).

Intra-articular movements such as distracting, twisting, and shaking in the loose-packed position of the joint are called “joint play,” which is not obtained in voluntary move-

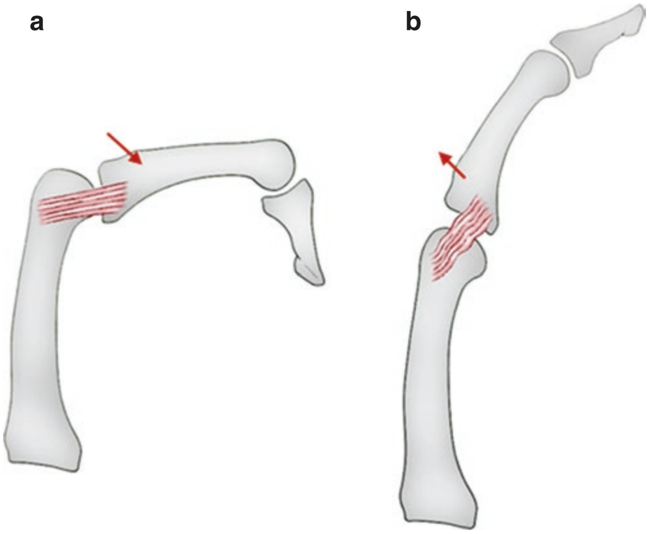

Fig. 3.9 The close- and loose-packed positions of the MP joint (part 1). (**a**) The close-packed position: The joint surface of the proximal phalanx slides ventrally in maximally flexed position. In this position, ligaments are tightened and the joint is stabilized. (**b**) The loose-packed position: The joint becomes loose in extended position giving better mobility but less stability

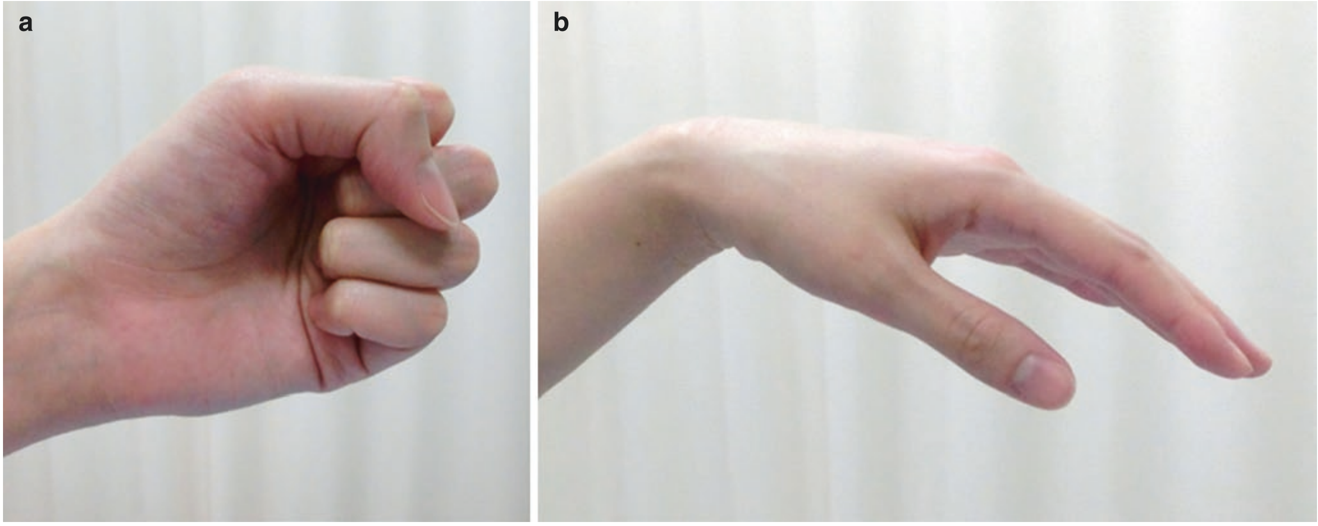

Fig. 3.10 The close- and loose-packed positions of MP joint (part 2). (**a**) The close-packed position: In this position, MP joint can deliver a strong punch, but is often susceptible to fractures of the metatarsal neck (boxer’s fracture). (**b**) The loose-packed position: In this position, the MP joint is susceptible to finger sprains and ligament damage

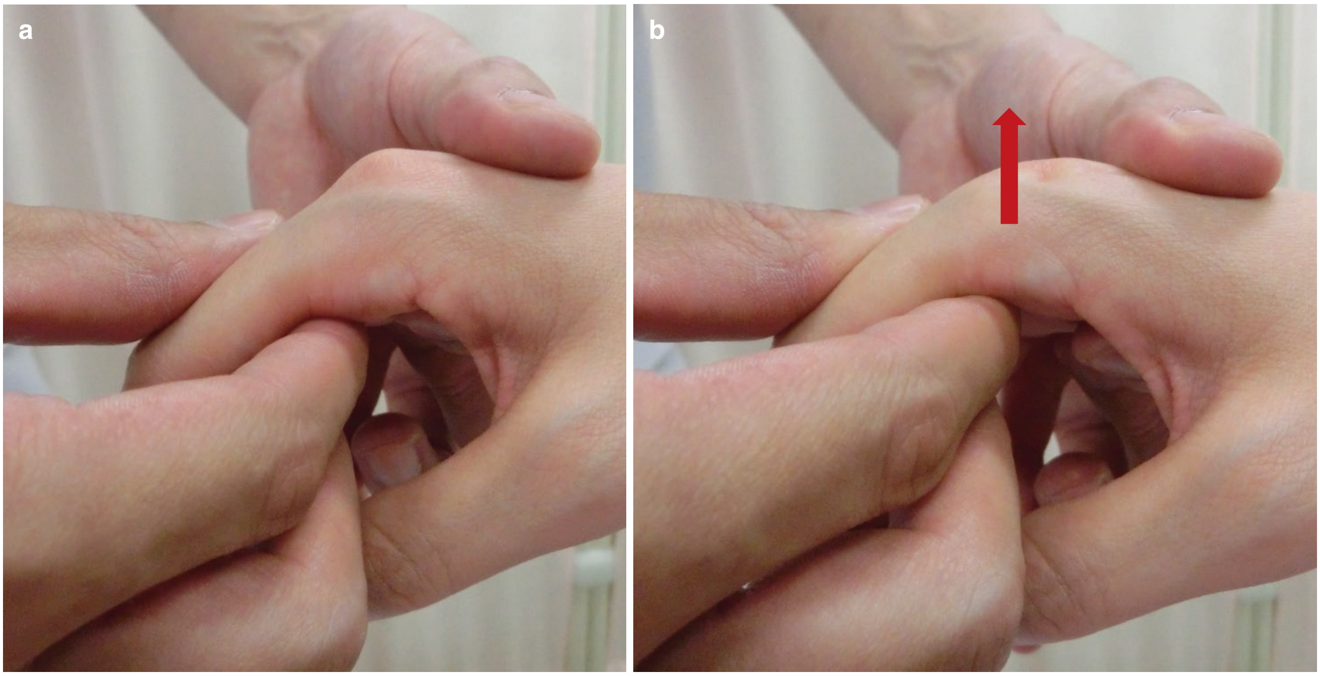

Fig. 3.11 Accessory movement 2nd type in the MP joint. This type of movement can be felt by gliding the bone. (**a**) Gently hold the proximal phalanx. (**b**) Glide dorsally. Pinching the bone decreases the joint play

ments. In the AKA-H theory, this joint play is defined as the "accessory movement 2nd type." There are glides, as well as distraction and axial rotation in this type of joint movement. These joint movements can be observed in the MP joint (Fig. 3.11).

The accessory movement 2nd type (movement in the joint capsule) has a close relation with the joint movement. Initially, the limitation of the accessory movement 2nd type restricts the joint movement. Secondary, accessory movement 2nd type is under the control of the mechanoreceptors. The arthrostatic reflex generated by type I joint receptors restricts the accessory movement of the 2nd type as a result of tensing up of the joint capsule and ligaments. In this state, the joint does not move in the same way as in the close-packed position of the joint (Fig. 3.12).

Another such movement is "the accessory movement 1st type," which is the movement at the terminal phase of a joint motion. This is an available movement at the terminal range. We can take the Japanese sitting position "Seiza" due to the accessory movement 1st type (Fig. 3.13). When falling down on the ground, the femoral joint surface of the knee moves forward to minimize the damage. This is also the work of the accessory movement 1st type. The accessory movement 1st type is also observed in flexion of the MP joints. When grasping a baseball, the rotation range of the MP joint becomes larger than when grasping the hand without a ball. This available range of motion of the MP joint is given by the accessory movement 1st type (Fig. 3.14).

Fig. 3.12 Restriction of the 2nd type of accessory movement due to arthrostatic reflex, which is triggered by pinching the bone

This available movement at the terminal range is sensed by examiners as the "end feel" sensation. The end feel is sensed softly in a normal joint, but tightly in case of joint dysfunction. The accessory movement first type is also influenced by an arthrostatic reflex. In passive flexion of the knee, for instance, the range of motion differs depending on the way of holding the leg. The end feel is not soft and the motion is limited when gripping the leg firmly. Firm gripping triggers an arthrostatic reflex, and periarticular soft tissues

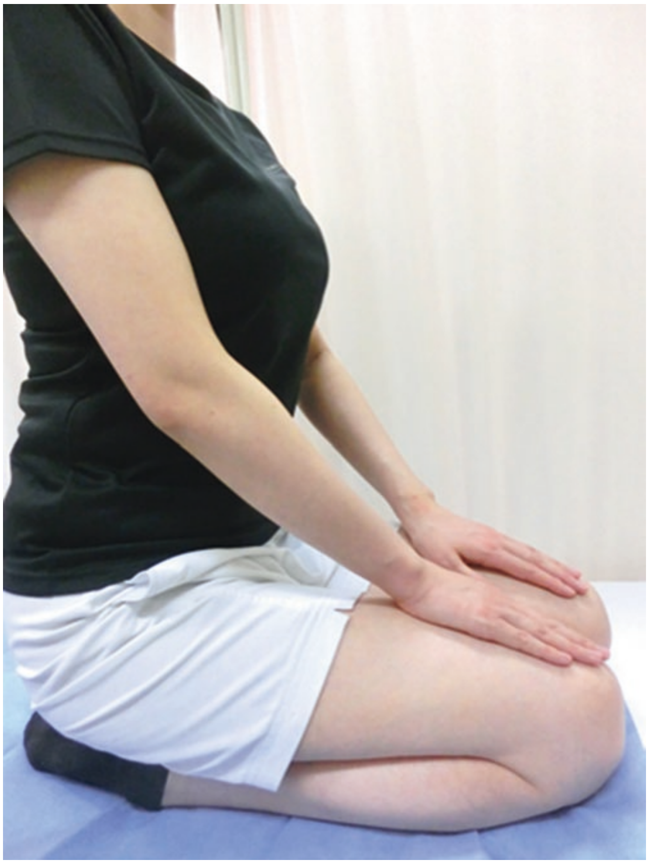

Fig. 3.13 The 1st type of accessory movement in taking the Japanese sitting position “Seiza.” The knee flexion is about 150° with hip flexed position. The 1st type of the accessory movement in the terminal phase of the knee joint flexion enables to take this sitting position

become tense, resulting in restriction of the first type of accessory movement (Fig. 3.15). The end feel is applied for the diagnosis of SIJ dysfunction, such as sensing the end feeling in the straight leg raising (SLR) test. Training for sensing the end feel in the SLR test is very important to evaluate an SIJ dysfunction.

3.5 Accessory Movements of SIJ and Dysfunction

The accessory movement 2nd type of SIJ includes sliding and distraction. The range of the 2nd type is different in each person. Examiners feel the range of the joint motion from 0.5 mm to 3 mm with the sensation of their hand. In the case of normal SIJ or SIJ dysfunction with slight inflammation, the accessory movement type 2nd is felt soft. However, in the case of SIJ dysfunction with severe inflammation, the feeling is restricted. In case of SIJ contractures after repeated inflammation of the joint, the accessory movement 2nd type becomes narrower.

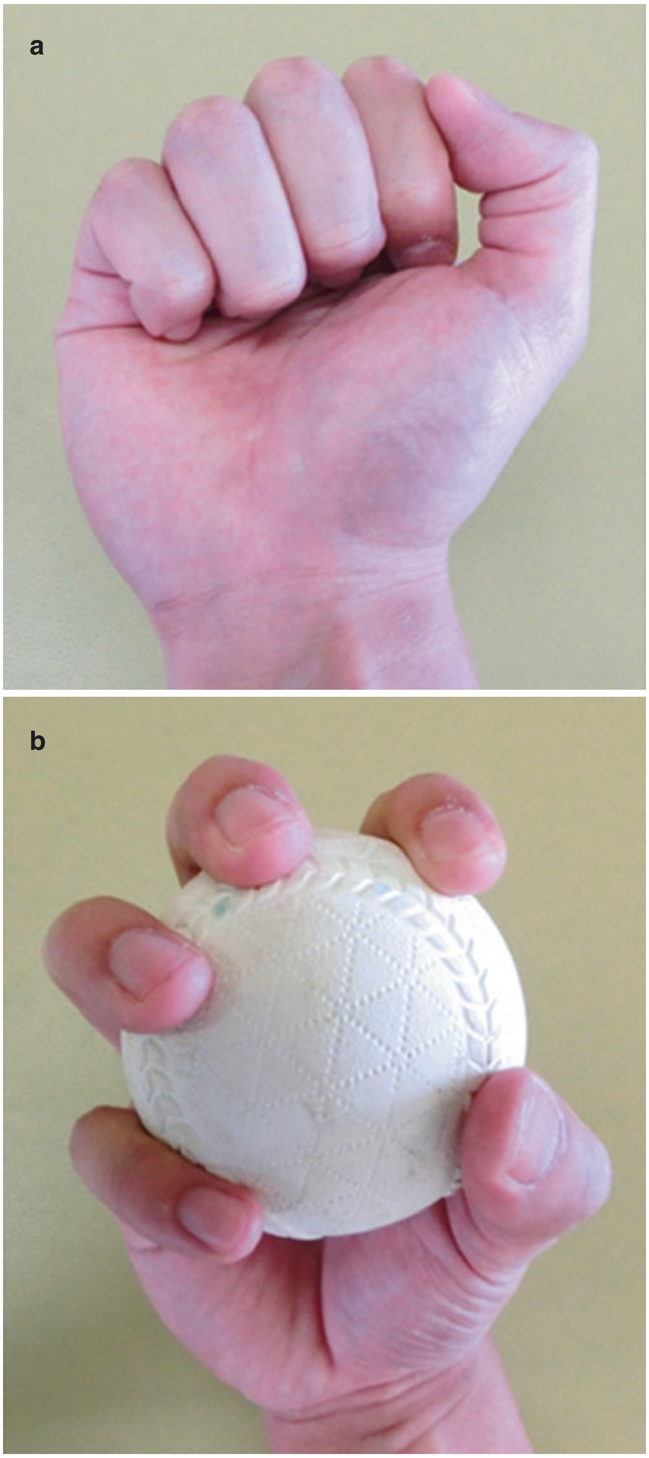

Fig. 3.14 The 1st type of accessory movement of the MP joint. (**a**) Gripping without a ball: The MP joints rotate to some degree. (**b**) Grasping a ball: The range of motion of the MP joint becomes larger than in (**a**). This available range of motion of the MP joint is given by the accessory movement 1st type

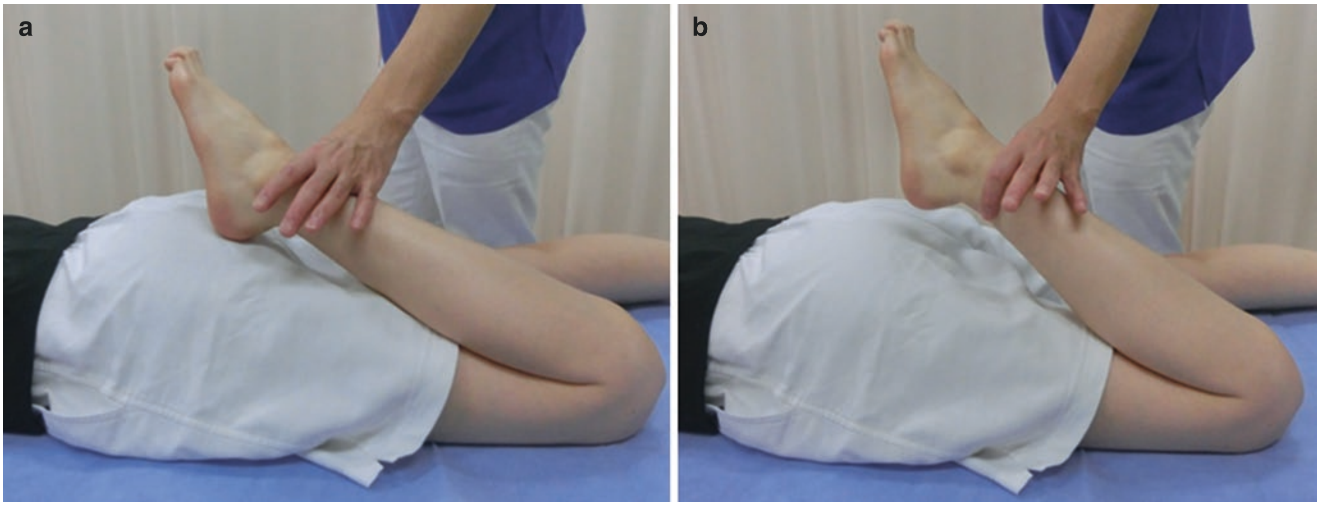

Fig. 3.15 The 1st type of accessory movement of the knee. Passive flexion of the knee in prone position. Sensing the end feel in the joint is quite important to know the 1st type of accessory movement. (**a**) The end feel is soft when the tibia is flexed by the examiner's palm. (**b**) The end feel is not soft and the motion is limited when gripping the leg firmly

3.6 Symptoms Originating from SIJ Dysfunction

Observations of patients after AKA-H treatments for SIJ revealed symptoms originating from SIJ dysfunction as follows: low back pain, buttock pain, and restricted trunk movements, as well as referred pain, numbness, edema, and muscle tightness in the lower extremities.

3.7 Causes of SIJ Dysfunction

3.7.1 Primary SIJ Dysfunction

SIJ can be in a subluxation state when it is caused by accidental damage in the loose-packed position. In the joint having a small range of motion, such as SIJ, spinal facet joints, and so on, some subluxation of the joint can continue for a long time and symptoms originating from the joint cannot be relived. Because SIJ does not have muscles to move by itself, it is difficult for the subluxation to be adjusted at will by patients. If the same thing occurs in the joint having a large range of motion, such as the hip joint, the glenohumeral joint, etc., a little subluxation will be no problem and will adjust naturally with daily life joint movement.

Acute low back pain often occurs in the stooping position when lifting heavy baggage. Acute subluxation of the SIJ can occur in such a loose-packed position, and is the origin of acute low back pain (Fig. 3.16).

Fig. 3.16 The stooping position for lifting heavy baggage. This position is a loose-packed position in the sacroiliac joint. Acute subluxation of the sacroiliac joint can occur in this position, and a patient complains of acute low back pain

3.7.2 Secondary SIJ Dysfunction

The limitation of the knee extension due to osteoarthrosis of the knee joint can cause a loose-packed position of the knee joint. SIJ also could occur in the loose-packed position when standing while keeping heel contact. This has a risk of SIJ dysfunction. Kyphosis due to degenerative spondylosis could cause spinal facet joint dysfunction and SIJ dysfunction. Additionally, an overload in the adjacent segment of the spine after spinal fusion surgery as well as a pelvic inclination and scoliosis due to leg length discrepancy could cause SIJ dysfunction.

3.8 Arthrostatic Hyper-Reflex Chain

SIJ dysfunction causes ipsilateral joint dysfunction via an arthrostatic hyper-reflex chain. SIJ dysfunction causes ipsilateral joint dysfunction of the hip joint, the knee joint, the ankle joint, facet joints, costovertebral joints, and other joints having a small range of motion, but on the same side of the body. For example, right SIJ dysfunction caused ipsilateral joint dysfunction only on the right side of the body. The contralateral SIJ alone can be influenced by SIJ dysfunction on the opposite side of the body (Fig. 3.17).

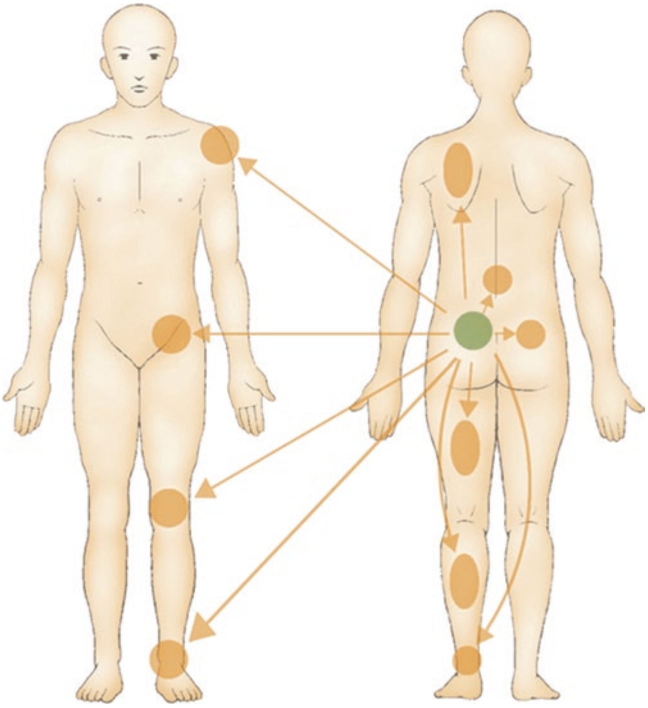

Fig. 3.17 The arthrostatic hyper-reflex chain. The sacroiliac joint dysfunction causes ipsilateral joint dysfunction of the hip joint, the knee joint, the ankle joint, facet joints, costovertebral joints, and other joints having a small range of motion. The contralateral sacroiliac joint alone can be influenced by sacroiliac joint dysfunction on the opposite side of the body

3.9 Joint Dysfunction and Inflammation

Aseptic inflammation is sometimes observed in the knee, shoulder joint, and so on. Simple arthritis of the hip in infants and adults is also one type of aseptic inflammation of joints. Hydrarthrosis often accompanies aseptic inflammation of joints. Overuse could cause such primary inflammation in joints. In general, this kind of inflammatory state could continue for several days, or occasionally, for 2 or 3 months. Aseptic inflammation also occurs in SIJ and can continue for 2 or 3 months. The AKA-H for these inflammatory conditions is not effective.

The primary inflammation of the SIJ is named “simple sacroiliac arthritis,” which is similar to simple arthritis of the hip. The onset of this condition is either acute or chronic. The pain is very severe in acute simple sacroiliac arthritis and is relieved only after several days, disappearing in 2–3 months. Complication of SIJ dysfunction with simple sacroiliac arthritis could lead to chronic pain. If the AKA-H treatment for SIJ dysfunction is performed, SIJ pain will not continue for over 2 or 3 months, this is a period when simple sacroiliac arthritis disappears naturally.

3.10 Silent Afferent Neurons

Type IV joint receptors, which are nociceptive receptors, are inactive for stretching a joint capsule or ligaments in normal conditions. Degeneration or deformity of an articular cartilage alone doesn’t cause the pain, because nociceptive receptors are absent in the cartilage.

Type IV joint receptors respond with a high threshold and become active against nociceptive mechanical stimulation. Type IV is inactive in the normal physiological state of joint movements. In case of joint dysfunction, the type I receptor reacts, resulting in a hyper-state of arthrostatic reflex. Strained joint capsules and ligaments lead to a response of the type IV receptors, which cause the joint pain.

About 30% of type IV receptors are called “silent afferents” and react only to inflammation. Michaels et al. reported that numerous silent afferent neurons were located in visceral and deep somatic tissue, and they didn’t response to physiological stimulation. However, they did respond to inflammatory stimulations. The silent afferents contain neuropeptides, and these peptides are released when activated under inflammation stimulations, triggering vasodilation and swelling. Under activation of silent afferents, joint pain could occur when a joint capsule and ligaments are stretched. Tenderness of a joint, painful sensation with soft palpitation, and rest pain could also occur under inflammatory conditions [11, 12]. Complication of SIJ dysfunction with severe inflammation could lead to severe SIJ pain.

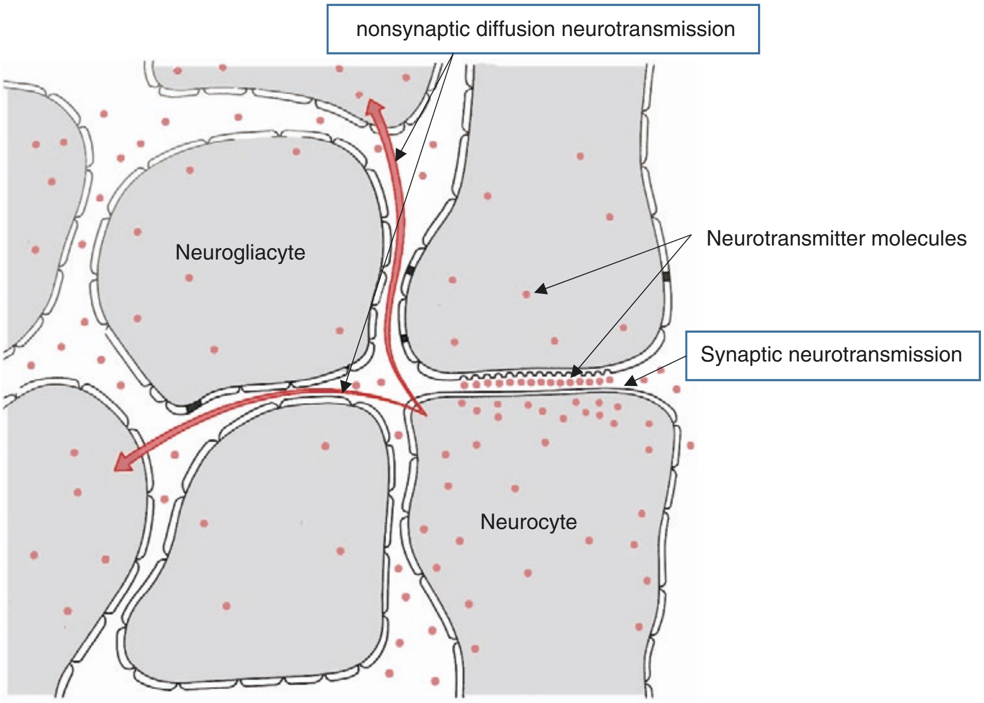

Fig. 3.18 Nonsynaptic diffusion neurotransmission (NDN). This schema is cited from Ref. [15]—with slight modification

3.11 The Transmission Pathway of Joint Pain

Pain is recognized when stimulation caught by nociceptive receptors is transmitted to the brain. Synaptic neurotransmission and neuroendocrine secretion are well known. Additionally, nonsynaptic diffusion neurotransmission (NDN) has become known recently (Fig. 3.18) [13–16]. Initially, stimulation is received by nociceptive joint receptors and is transmitted quickly via the synaptic transmission pathway. On and off transmission is clear in this synaptic pathway. For example, signals of bone fractures and ligament injuries are transmitted quickly.

In the NDN pathway, neuropeptides released from silent afferents are transmitted to an algesthesia center of the brain bypassing the synaptic pathway. These signals reach and diffuse in the algesthesia center of the brain a little bit behind the synaptic neurotransmission. Both pathways transmit stimulations around the joint to the algesthesia center of the brain and joint pain is recognized. Therefore, it is considered that pain immediately relieved after an AKA-H treatment is pain transmitted via synaptic neurotransmission, and pain relieved 3–10 min after an AKA-H treatment is via the NDN.

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