Pathomechanics of Stance

Clinical Concepts for Analysis

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This paper presents some basic biomechanical considerations that can form a basis for analysis of stance deviations. The floor reaction forces and their effects on the lower extremity are reviewed as a basis for understanding the pathomechanics of stance. Pathomechanics includes the unwanted motions that tend to be created by the floor reaction forces in the absence of critical muscle activity. Compensatory postures that a patient may assume to prevent these unwanted motions and compensatory postures that a patient may assume at one joint because of abnormal positioning at another joint are discussed. Finally, the influence of joint posture from one phase of gait to another is explained.

Key Words: Biomechanics, Gait.

The observational gait analysis techniques introduced by the physical therapy and pathokinesiology departments at Rancho Los Amigos Hospital have made clinical gait analysis more objective than in the past. By emphasizing observational skills, therapists have learned to identify critical events in the gait cycle and any deviations of these events.1 After accurate identification of gait deviations, the therapist must determine the causes of the deviations to determine treatment effectively. Attempting to eliminate a deviation without determining its cause may lead to inadequate and ineffective patient care. For example, quadriceps femoris muscle strengthening or application of a knee orthosis would be inappropriate treatment for a patient whose knee flexion posture in stance is due to weakness of the soleus muscle.

Stance deviations may be caused by motor control deficit, deformity, or sensory deficit; compensations for these problems; compensation for pain that results in an antalgic posture; or compensation for postures occurring at other joints.

This paper will present 1) a review of the floor reaction forces and their effects on the lower extremity in stance as a basis for understanding the remainder of the text, 2) the unwanted motions created by the floor reaction forces in the absence of critical muscle activity and the subsequent compensatory postures that a patient may assume to prevent these unwanted motions, 3) the compensatory postures that a patient may assume at a joint because of a position at another joint, and 4) the influence of posture from one phase of gait to the next. I have not intended to include every possible cause of stance deviations but to include some biomechanical considerations that can form an elementary basis for analysis of stance for patients of any etiology. Some content is my opinion based on these biomechanical considerations. When content has been supported by the literature, I have cited references.

USE OF FLOOR REACTION FORCES

Understanding the biomechanics of maintaining stability of body parts is fundamental to interpreting the causes of gait deviations occurring in stance. The floor reaction forces are the principal external forces that render a joint either inherently stable or unstable.² If unstable, muscle action is necessary to counteract an external floor reaction force.

Conventionally, many physical therapists analyzing gait deviations use alignment of the body's center of gravity over the foot to explain stance stability.³ This application of the laws of gravity, although useful, is in error. The walking individual's center of gravity lies outside of the base of support during loading and unloading of his weight to the stance limb. Only during the single-limb support periods of mid-stance and early terminal stance should the center of gravity truly lie over the base of support.^{2, 4, 5} In addition to the center of gravity's position, the floor reaction forces account for the effect of acceleration of the center of gravity and the inertia of its movement.² Use of the position of the floor reaction force vectors (FRFVs) in relationship to the joints of the stance limb rather than the center of gravity's relationship to the joints or foot gives the clinician more accurate information regarding stance stability.

Floor reaction forces are vertical, medial-lateral, and fore-aft. The vertical and fore-aft forces are important for motion in the sagittal plane, and the vertical and medial-lateral forces are important for motion in the frontal plane. For each plane, the two forces may be combined into a single force. This resultant force depicts the total floor reaction forces acting in the plane. Visualization of these forces is accomplished by the use of vectors drawn over the figure of a walking subject (Fig. 1).

A floor reaction force is equal in magnitude and opposite in direction to the force that the body applies to the floor through the foot. Its relationship to a joint determines what motion tends to occur at that joint because of external forces.² The greater the perpendicular distance of a FRFV from a joint's axis, its lever arm, and the greater the length of the vector, its magnitude, the greater is the FRFV's tendency to produce rotation at the joint, its moment or torque. A resultant FRFV depicts the combined floor reaction forces and can be used to compute the total floor reaction torque for each plane (Fig. 2).

Recently, investigators have been able to display instantaneously the resultant FRFVs superimposed on an image of the walking subject without using tedious mathematical hand calculations.^{2, 6}

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Figures for the normal resultant FRFVs included here are estimated from the work of Boccardi et al² and Perry et al.⁶ The lengths of the vectors have been drawn to show relative magnitudes. No precise scale is intended. Figures for the resultant FRFVs in pathological stance are conceptual and based on the literature as cited or on my experience.

NORMAL RESULTANT FLOOR REACTION FORCE VECTORS

Sagittal Plane

At initial contact, the FRFV normally lies anterior to the hip and knee and posterior to the ankle (Fig. 3).^{2.6} The hip, therefore, tends to flex, the knee is stable in extension, and the ankle tends to plantar flex. These tendencies are not large because the vector's magnitude is small at initial contact.

As body weight shifts to the limb during loading response, the vector shifts behind the knee and causes a flexor moment and the tendency for the knee to flex (Fig. 4). In normal gait, EMG output of the hip and knee extensors is greatest in this phase⁷ and emphasizes the magnitude of the floor reaction moment, which tends to cause hip and knee flexion.² The relationship of the FRFV posterior to the ankle causes a plantar flexion moment. The pretibial muscles contract and control the plantar flexion to about 15 degrees.^{1,2,7} In late mid-stance at normal velocity, the vector shifts behind the hip and anterior to the knee (Fig. 5).^{2.5} This shift is partially dependent on the momentum achieved by the contralateral swing limb.¹ The hip and knee joints are, therefore, stable in extension. At the ankle, the vector shifts anteriorly, creating a dorsiflexion moment. The triceps surae muscle becomes active, stabilizes the ankle, and allows smooth progression of the body over the plantigrade foot.^{1.7}



TORQUE (MOMENT) AT ANKLE = AxB

A Lever arm for ankle dorsiflexion.
B Magnitude of floor reaction force (arbitrary unit

B Magnitude of floor reaction force (arbitrary units)

Fig. 2. Method to calculate the floor reaction torque at the ankle, using the magnitude of the resultant FRFV and its lever arm.



Fig. 3. Normal resultant FRFV at initial contact: Vertical and sagittal forces.

This same relationship of the FRFV to the joints of the lower extremity remains constant in terminal stance and necessitates continued use of the triceps surae muscle (Fig. 6).⁷ At the end of terminal stance, the vector shifts behind the knee and causes a knee flexion moment. The knee rapidly flexes. As unloading occurs in preswing, the magnitude of the force vector diminishes considerably.^{2.6} The relationship of the vector to the joints continues as in late terminal stance (Fig. 7).

Frontal Plane

In the frontal plane, the FRFV lies lateral to the hip and knee at initial contact and produces a small moment of hip abduction and genu valgum (Fig. 8).² At the subtalar joint, the vector is neutral and therefore has no effect.

As the body weight advances onto the limb in loading response and in midstance, the vector shifts medially to the hip and knee and lateral to the subtalar joint (Fig. 9). This alignment results in moments of valgus (eversion) at the foot, varus at the knee, and adduction at the hip. Valgus is a normal occurrence at the foot,⁷ and the knee varus is opposed by ligamentous support. At the hip, action of the hip abductors maintains lateral hip stability.^{1,2,7}

In terminal stance, the FRFV shifts somewhat laterally and decreases its medial moments at the hip and knee (Fig. 10). At the foot, the vector shifts medially to the subtalar joint and produces a varus (inversion) moment. Foot varus occurs as the heel rises.⁷

In preswing, the FRFV lies neutral to the foot and knee and lateral to the hip (Fig. 11). The vector, therefore, creates a small abductor moment at the hip. Hip adductor muscles may become active at this time.⁷

PATHOLOGICAL POSITIONING OF RESULTANT FLOOR REACTION FORCE VECTOR

When weakness of a stabilizing stance muscle occurs, the floor reaction force is unopposed and stance stability may be lost. Stance muscle weakness, therefore, tends to cause unwanted motions. Primary weaknesses, however, are often compensated for by the patient who has adequate sensation and motor control. Through joint positioning, the patient may be able to move the FRFV to ren-



Fig. 4. Normal resultant FRFV at loading response: Vertical and sagittal forces.

der the joint stable. Positioning at one joint may necessitate other joint positioning to keep the body in upright alignment. Finally, because gait is not a static but a dynamic event, joint postures in one phase of gait tend to influence postures in the following phase.

UNWANTED MOTIONS CREATED BY WEAKNESS AND COMPENSATORY POSTURES ASSUMED

Sagittal Plane

Initial contact and loading response. Weakness of the pretibial muscles causes the foot to advance rapidly to the floor in loading response because the plantar flexion moment of the floor reaction force is unopposed. To avoid this plantar flexion moment, the patient may contact the ground with the foot flat while the FRFV passes neutral or anterior to the ankle; this action eliminates the plantar flexion moment (Fig. 12).

If the quadriceps femoris muscle is weak, the knee tends to flex excessively in loading response. To avoid this uncontrolled flexion of the knee, the patient may contact the ground with a flat plantar flexed foot (Fig. 12). As I have stated, floor contact with the foot flat moves the FRFV anteriorly. As a consequence, the FRFV's moment causing knee flexion will be diminished, as the FRFV remains anterior to the knee. The



Fig. 5. Normal resultant FRFV at midstance: Vertical and sagittal forces.



TERMINAL STANCE

Fig. 6. Normal resultant FRFV at terminal stance: Vertical and sagittal forces.



Fig. 7. Normal resultant FRFV at preswing: Vertical and sagittal forces.

patient may also lean the trunk forward to align the FRFV anterior to the knee (Fig. 13).⁸⁻¹¹ In either case, the normal loading response of knee flexion of 15 degrees will not occur. An undercut or cushion heel is another compensation designed to aid a patient with knee instability by moving the FRFV anteriorly (Fig. 14).¹²

Hip extensor weakness would allow the hip to fall into flexion. If the limb is stable on the floor, the pelvis will drop into a symphysis-down posture.^{13, 14} To



Fig. 8. Normal resultant FRFV at initial contact: Vertical and frontal forces.

prevent instability in flexion, the patient may position the FRFV posterior to the hip joint by leaning the trunk backwards (Fig. 15).^{13, 15}

Mid-stance and terminal stance. If a patient has a weak triceps surae muscle, advancement of the FRFV anterior to the ankle in mid-stance and terminal stance will tend to cause uncontrolled tibial advancement into excessive dor-





Fig. 11. Normal resultant FRFV at preswing: Vertical and frontal forces.

siflexion.¹⁶⁻¹⁸ To prevent this, the patient may keep his ankle in plantar flexion with his foot flat on the floor.¹⁸ This posture will tend to align the FRFV behind the ankle and eliminate the need for calf control (Fig. 16). The patient walks with a "step to" gait.

The hip and knee are stable in the sagittal plane during mid-stance and ter-

Fig. 12. Resultant FRFV with foot flat at initial contact to eliminate the plantar flexion moment.

minal stance. If velocity is decreased or if loading-response flexion is not restrained to 15 degrees at the knee and 30 degrees at the hip, the floor reaction force may cause hip and knee flexion.¹⁹ The same compensations for an unstable hip and knee that were used at initial contact and loading response may therefore be used here (Figs. 12–14). **Preswing.** In preswing, the FRFV diminishes considerably in magnitude.² The limb is being unloaded, and very little muscle activity is necessary for stability.¹ Weakness would not tend to produce any unwanted motions.

Frontal Plane

The primary instability resulting from muscle weakness occurs at the hip. During most of stance, the FRFV produces an adduction moment at the hip. In a patient with hip abductor weakness, a contralateral pelvic drop tends to occur.²⁰ To compensate, the patient may lean his trunk to the stance limb.¹⁷ This places the FRFV lateral to the hip and maintains hip stability (Fig. 17).

The varus moment at the knee is opposed by ligamentous forces. If a patient has a knee flexion posture causing slack in these ligaments or if he has inadequate ligaments, a varus knee posture tends to occur. The valgus knee posture that often occurs in patients with rheumatoid arthritis²¹ may be due to lateral shifts in the FRFV, which these patients tend to display.^{22, 23}

POSTURES COMPENSATORY TO OTHER ABNORMAL POSTURES

I will now consider the influence of one joint's posture on that of the other joints. Just as a primary scoliotic curve may eventually produce a compensatory curve in the opposite direction to form a balanced scoliosis, primary gait postures may necessitate compensatory postures at other joints to maintain an upright position and stability. These compensatory postures seem to be most critical when velocity is decreased and momentum cannot aid stability. For ease of interpretation, the postures discussed are assumed to occur during single limb support when the center of gravity is truly over the base of support.

Plantar Flexion

The posture of plantar flexion may be compensated for by heel rise. Heel rise allows advancement of the tibia over the foot. No other joint compensations are necessary. A plantar flexed ankle with the foot flat, however, causes the tibia to incline posteriorly.^{9,24} The FRFV is aligned posterior to the ankle and the



Fig. 13. Resultant FRFV with trunk forward lean at initial contact to eliminate the knee flexion moment.

tibia may not advance forward (Fig. 18a).²⁵ Necessary compensatory postures to maintain the body upright are knee hyperextension^{16, 24, 25} in Figure 18b or trunk forward lean in Figure 18c.^{10, 16, 25}

Knee Hyperextension

A primary position of knee hyperextension (Fig. 19a) may be compensated for by ankle plantar flexion in combination with hip hyperextension (Fig. 19b) or by backward trunk lean (Fig. 19c).¹⁰

Trunk Forward Lean

A primary deviation of trunk forward lean (Fig. 20a) is compensated by ankle plantar flexion (Fig. 20b) or less likely by knee flexion (Fig. 20c). As can now be appreciated, plantar flexion and knee hyperextension (Figs. 18b, 19b) and plantar flexion and trunk forward lean (Figs. 18c, 20b) are interrelated postures. If a patient displays one of these individual postures, its pair is likely to occur.

Ankle Dorsiflexion or Knee Flexion

Ankle dorsiflexion and hip and knee flexion as well as dorsiflexion and trunk backward lean are related in the same manner as ankle plantar flexion, knee hyperextension, and trunk forward lean. Excessive dorsiflexion with the foot flat places the FRFV anterior to the ankle and causes the tibia and body to advance



Fig. 14. Resultant FRFV at initial contact with an undercut heel to eliminate the plantar flexion moment.



Fig. 16. Resultant FRFV with plantar flexion in single limb support to eliminate the dorsiflexion moment.

forward (Fig. 21a).¹⁶ To compensate, the hip and knee flex⁹ (Fig. 21b), or the trunk leans backwards (Fig. 21c).¹⁰ Following this same reasoning, one can determine that a posture of knee flexion is compensated by ankle dorsiflexion and hip flexion (Fig. 21b). If the knee flexion is severe, dorsiflexion range may not be adequate to maintain body alignment over the foot. To further compensate, the heel rises (Fig. 22).¹⁷

Hip Flexion

A hip-flexed posture may be compensated for by knee flexion and resulting dorsiflexion (Fig. 21b).¹⁴ The more common position of hip flexion, symphysis down, is compensated by a lumbar lor-



Fig. 15. Resultant FRFV with trunk backward lean at initial contact to eliminate the hip flexion moment.



Fig. 17. FRFV with trunk lateral lean toward the stance limb to eliminate the hip adduction moment in single limb support.

dosis to keep the trunk upright (Fig. 23).^{13, 14}

Heel Rise

Heel rise in terminal stance occurs during single limb support. To have a normal heel rise during single limb support, the ankle is held at neutral as body weight advances forward of the foot (Fig. 6).¹ If the heel does not rise, then either







Fig. 19. Knee hyperextension posture in single limb support; A) no compensation, B) compensation by ankle plantar flexion, and C) compensation by trunk backward lean.

the ankle is positioned in excessive dorsiflexion or the body weight has not advanced forward.¹¹ If the patient is in excessive dorsiflexion, then a compensatory posture of knee flexion occurs (Fig. 21b) or the patient falls onto the opposite limb (Fig. 21a) and decreases stride length and single-limb support time.¹¹ If body weight is not advanced, the ankle is most likely positioned in slight plantar flexion (Fig. 18a). Compensatory postures of knee hyperextension or trunk forward lean occur (Figs. 18b,c).

Trunk Backward Lean

A posture of trunk backward lean requires dorsiflexion of the ankle to maintain balance (Fig. 21c).²⁶ Thus, for the paraplegic to be able to maintain hip stability by trunk backward lean, his knee-ankle-foot orthoses must allow his ankles to dorsiflex.

Varus and Valgus

Abnormal alignment in the frontal plane also produces compensatory postures. Because of the alignment of the subtalar joint, internal rotation of the tibia produces foot valgus and external rotation produces foot varus. Foot varus and valgus postures produce the corresponding rotation of the tibia.^{27, 28}

Knee varus and valgus will tend, respectively, to produce hip abduction and adduction and a tendency to foot valgus and varus if the foot remains flat (Fig. 24).²⁹ An abducted hip posture necessitates trunk lateral lean to the same side, and adduction of the hip necessitates trunk lateral lean to the opposite side (Fig. 25).¹³

Combination of Weakness, Compensatory Posturing, and Secondary Compensatory Posture

Now that the effects of weakness, the compensatory postures for that weakness, and the compensatory postures that may occur at joints because of abnormal posturing of another joint have been considered, an example combining these concepts is appropriate. A patient with quadriceps femoris muscle weakness must align his FRFV anterior to his knee to maintain knee stability. To do this, he may lean his trunk forward. This trunk forward lean then necessitates a compensatory posture of plantar flexion or knee hyperextension for him to maintain an upright alignment (Figs. 18b, c).

INFLUENCE OF POSTURE FROM PHASE TO PHASE

Terminal Swing—Initial Contact

Walking is a dynamic activity. Events in one phase of gait necessarily affect events in subsequent phases. Terminal swing postures affect the methods of floor contact in initial contact. The neu-

PRACTICE





Fig. 22. Heel rise in single limb support because of knee flexion posture with inadequate dorsiflexion.

Fig. 20. Trunk forward-lean posture in single limb support; A) no compensation, B) compensation by ankle plantar flexion, and C) compensation by knee flexion.



Fig. 21. Excessive dorsiflexion posture in single limb support; A) no compensation, B) compensation by hip and knee flexion, and C) compensation by trunk backward lean.

tral ankle and extended knee provide for heel contact (Fig. 3).¹ If the ankle is plantar flexed greater than 30 degrees or if the knee is flexed greater than 30 degrees or if a combination of plantar flexion and knee flexion achieve greater than 30 degrees, initial contact will be with the foot flat or with the forefoot (Fig. 26).

Initial Contact—Loading Response

In the same manner, the method of floor contact influences loading response. If initial contact is made with the foot flat or with the forefoot, the ankle plantar flexion moment will tend to be decreased and the FRFV will tend to remain anterior to the knee in loading



Fig. 23. Hip flexion posture with symphysis down compensated by lumbar lordosis.



Fig. 24. Knee varus (A) and valgus (B) in single limb support showing compensatory hip abduction and adduction, respectively.



Fig. 25. Hip adduction (A) and abduction (B) postures in single limb support with resulting trunk lateral lean to opposite and stance sides, respectively.



Fig. 26. Combination of ankle plantar flexion and knee flexion postures producing initial contact with forefoot.



Fig. 27. Effect of loss of plantar flexion in loading response: A) posture at initial contact and B) resultant posture in loading response.

response (Fig. 12).^{8,9} Loading response knee flexion will not occur. If a patient is unable to plantar flex because of an orthosis with an ankle joint downstop, loading-response plantar flexion is lost, and the tibia will advance rapidly forward as the foot falls to the floor.⁹ The forward motion of the tibia increases the knee flexion moment, and the patient tends to flex excessively at the knee (Fig. 27).

Loading Response—Mid-Stance and Terminal Stance

Mid-stance occurs with contralateral swing when the patient perceives that his limb is stable. His method of floor contact and loading response may be chosen as above to assure mid-stance and terminal stance stability. If the patient is unstable in mid-stance, singlelimb support time will be decreased and the FRFV may not advance forward in terminal stance.¹⁶ If the vector does not advance to the forefoot, heel rise may not occur (Fig. 18a). If the patient's ankle advances into excessive dorsiflexion in mid-stance, the heel may also tend to stay on the floor in terminal stance (Fig. 21a).

Terminal Stance—Preswing

Preswing knee flexion occurs as the limb is unloaded after heel rise. If advancement of body weight is diminished or if the heel does not rise in terminal stance, the resultant posture tends to keep the FRFV anterior to the knee (Fig. 21a). The knee, therefore, does not passively flex to 35 degrees in preswing.¹¹

Preswing—Initial Swing

Finally, preswing postures affect the motions that occur in initial swing. The knee flexes 25 degrees in initial swing from the preswing posture of 35 degrees to the initial swing posture of approximately 60 degrees.¹ This knee flexion ensures toe clearance in initial swing.1 Lack of preswing knee flexion necessitates excessive knee flexion in initial swing¹¹ or compensatory motions at other joints to achieve floor clearance.25 Methods of compensation that may be used include pelvic hiking, circumduction, trunk lateral lean toward the stance limb, vaulting, or a combination of any of these motions (Fig. 28).



Fig. 28. Compensations used in ipsilateral swing phase for inadequate preswing knee flexion.

CONCLUSION

In this analysis of stance-phase deviations, I considered the effects of one weakness or one posture on stance stability. Analysis of gait of the patient with multiple weaknesses or deformities or both is obviously more complex. Furthermore, some patients may not have the proprioceptive awareness to perform the compensations mentioned. Others may be affected by compulsory patterns of motion that limit their ability to compensate. These deficits must also be considered in analysis of stance deviations. The concepts presented here, however, can be used as an elementary basis for analysis. Clinicians should consider the mechanical factors that may affect their patients with neurological deficits and consider the possible abnormal neurological influences when they analyze their patients' gaits.

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