Catastrophic Shifts in Instantaneous Axes of Rotation

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When one bone moves upon another the movement is usually a rotation. Rotations always occur about a directed line in space called the axis of rotation. The location and direction of the axis of rotation for a movement are often fairly constant, but there may be shifts in their location and/or direction as the movement progresses. When the axis of rotation moves at the movement progresses, the axis at any given moment during the movement is called the instantaneous axis of rotation (IAR). In this discussion we will consider situations where the location and/or the direction of the axis of rotation shifts very rapidly from one state to another, essentially discontinuously. Such transitions will be called catastrophic shifts, following the nomenclature of Rene Thom, a French mathematician who was one of the pioneers of the field of topology called catastrophe theory, that deals with such discontinuous surfaces in phase spaces.

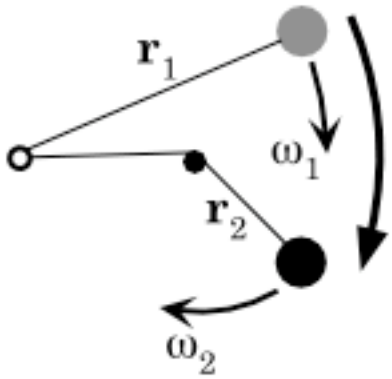
A catastrophe is a sudden, discontinuous change in the state of a system. They may occur with violence, such as an explosion that results when a quantity of a chemical mixture suddenly experiences a very rapid, exothermic, change. On the other hand, a catastrophe may be simple transition such as a dead leaf suddenly parting company with a twig that it has been attached to for a considerable time, or a superheated or supercooled fluid suddenly boiling or freezing as a result of a very minor disturbance. Such discontinuous transitions are commonly modeled as folded surfaces in a state space. It may be demonstrated mathematically that there are a limited number of ways that such surfaces may be formed for a given number of state variables. The study of these surfaces is the subject matter of a branch of topology concerned with non-linear dynamical processes.

We will not be concerned with the topological mathematics in this examination, but with the causes and consequences of such rapid transitions in joints. Catastrophic changes in the axis of rotation are a common phenomenon in the body's joints. They occur when a structure, such as a ligament, restrains a movement when the movement reaches a particular point in its excursion. Prior to that point the movement occurs about axis that may be determined by any of a number of factors, including muscle tensions and the contours of the joint surfaces. The ligament is lax so the movement progresses smoothly until the ligament is suddenly taut and will not stretch. If movement is to occur at that point, then it must occur about the attachments of the ligament to the bones. Consequently, there is a very rapid shift in the location and/or the direction of the axis of rotation. Similar rapid shifts may occur when two bones come into abutment and the axis

of rotation shifts to the point of contact. The two situations that tend to lead to catastrophic changes in the IAR are those that stop a joint angle from increasing and those that stop a joint angle from decreasing.

Frequently, such catastrophic changes in the axis of rotation are a normal component of the movement. For instance, a movement progresses until it takes up the slack and then the attached bone is drawn into the movement, allowing further rotation to occur in a more distal joint in which it participates. Such catastrophic shifts are beneficial and normal. However, there is a possibility of injury when such a shift occurs unexpectedly and the joint is under considerable force. When the axis of rotation shifts, there is a sudden change in the distribution of force couples about the joint. If the nervous system can not compensate fast enough, then there is the possibility of torn soft tissues or even broken bones. It is possible that catastrophic shifts of axes of rotation may be a significant cause of back injuries.

A Simple Example



Consider a very simple example of a catastrophic change in IAR. A cord, with a weight attached, is swinging about a central axis when it encounters a post. The axis of rotation shifts to the location of the post, which now acts as the fulcrum for the movement. If you try this experiment you will see that when the axis of rotation shifts to the post, the weight and the portion of the cord distal the post follows to a smaller arc and there is a sudden increase in the speed with which the cord swings. This is because the described system is closed to external energy gain or loss or change in mass. The angular momentum is conserved. The principles are

the same as apply when a skater draws their arms in while spinning to increase the rate at which they revolve.

Let $\mathbf{p}$ stand for the angular momentum, which is defined as a quantity that is directed perpendicular to both the vector that extends from the center of the coordinate system to the revolving object, $\mathbf{r}$, and to both of the vectors that extend in the direction of movement of the object, its velocity and momentum, $\mathbf{v}$ and $\mathbf{p}$.

$$\mathbf{L} = \mathbf{r} \times \mathbf{F} = \mathbf{r} \times \frac{\mathbf{dp}}{\mathrm{dt}}, \text{ where}$$

$\mathbf{F} =$ a force and

$\mathbf{r} =$ vector from the center of the coordinate system to the point of application of the force,

$\mathbf{p} = \mathrm{m}\,\mathbf{v}$ = the momentum of the moving object, but

$$\frac{\mathrm{d}}{\mathrm{dt}}(\mathbf{r} \times \mathbf{p}) = \left(\frac{\mathbf{dr}}{\mathrm{dt}} \times \mathbf{p}\right) + \left(\mathbf{r} \times \frac{\mathbf{dp}}{\mathrm{dt}}\right) = (\mathbf{v} \times \mathbf{p}) + \left(\mathbf{r} \times \frac{\mathbf{dp}}{\mathrm{dt}}\right)$$

$$= \mathrm{m}(\mathbf{v} \times \mathbf{v}) + \left(\mathbf{r} \times \frac{\mathbf{dp}}{\mathrm{dt}}\right) = \mathbf{r} \times \frac{\mathbf{dp}}{\mathrm{dt}}, \text{ consequently}$$

$$\mathbf{L} = \mathbf{r} \times \mathbf{F} = \frac{\mathrm{d}}{\mathrm{dt}}(\mathbf{r} \times \mathbf{p}).$$

If $\mathbf{L}_1$ is the angular momentum of the swinging cord prior to encountering the post and $\mathbf{L}_2$ its angular momentum after doing so, then it follows that -.

$$\mathbf{L}_1 = \mathbf{L}_2$$

$$\frac{\mathrm{d}}{\mathrm{dt}}(\mathbf{r}_1 \times \mathbf{p}_1) = \frac{\mathrm{d}}{\mathrm{dt}}(\mathbf{r}_2 \times \mathbf{p}_2)$$

$$\mathrm{m}_1 \cdot \frac{\mathrm{d}}{\mathrm{dt}}(\mathbf{r}_1 \times \mathbf{v}_1) = \mathrm{m}_2 \cdot \frac{\mathrm{d}}{\mathrm{dt}}(\mathbf{r}_2 \times \mathbf{v}_2); \quad \mathrm{m}_1 = \mathrm{m}_2$$

If the radius of the swing immediately prior to contacting the post is 'η' times the radius after and the radius and velocity are orthogonal sinusoidal function of the angle between the cord and some reference direction,

$$\mathbf{r} = \kappa \cdot cos(\omega\,\mathrm{t}) * \mathbf{i} + \kappa \cdot sin(\omega\,\mathrm{t}) * \mathbf{j}$$

$$\mathbf{v} = \frac{\mathbf{dr}}{\mathrm{dt}} = -\kappa\omega \cdot sin(\omega\,\mathrm{t}) * \mathbf{i} + \kappa\omega \cdot cos(\omega\,\mathrm{t}) * \mathbf{j}$$

then the

$$\mathbf{r}\times\mathbf{p} = \kappa^2\omega\cdot\left[cos(\omega t)*sin(\omega t)+sin(\omega t)*cos(\omega t)\right]*\mathbf{k}$$
$$= \kappa^2\omega\cdot sin(2\omega t)*\mathbf{k}$$

and

$$\mathbf{r}_1\times\mathbf{v}_1 = \mathbf{r}_2\times\mathbf{v}_2\ ;\quad \mathbf{r}_2 = \eta\mathbf{r}_1$$
$$\kappa^2\omega_1\ sin(2\omega_1 t)*\mathbf{k} = \eta^2\kappa^2\omega_2\ sin(2\omega_2 t)*\mathbf{k}\ ,\ t = t_c = 0$$
$$\omega_2 = \frac{\kappa^2\omega_1}{\eta^2\kappa^2} = \frac{\omega_1}{\eta^2}\ \text{ at } t_c = 0\ .$$

By a suitable choice of parameters we can set the time equal to zero at the instant of contact with the post and the momentum immediately prior to contact is the same as that immediately after contact. Therefore, the angular velocity immediately after contact will be equal to the angular velocity immediately before contact multiplied by the inverse of the square of the ratio of their lengths. If the post contacts the swinging cord midway between its ends then the angular velocity is four times a great after the contact. Note, however, that the radius is half as long so the speed of the swinging object, s, is only twice as great after the contact with the post.

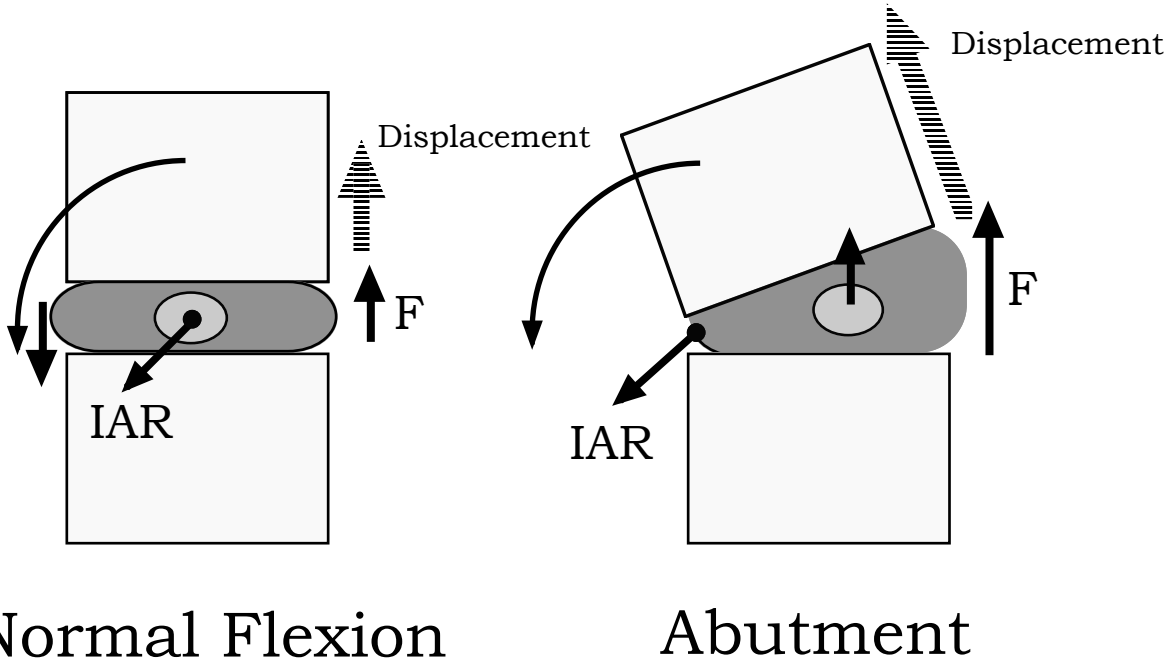
$$\mathbf{v} = -|\mathbf{r}|\cdot\omega\cdot sin(\omega t)*\mathbf{i}+|\mathbf{r}|\cdot\omega\cdot cos(\omega t)*\mathbf{j}\ ;$$
$$s = \sqrt{\left[|\mathbf{r}|\cdot\omega\cdot sin(\omega t)\right]^2+\left[|\mathbf{r}|\cdot\omega\cdot cos(\omega t)\right]^2}$$
$$= \sqrt{|\mathbf{r}|^2\cdot\omega^2\cdot\left[sin^2(\omega t)+cos^2(\omega t)\right]} = |\mathbf{r}|\cdot\omega$$
$$\frac{s_1}{s_2} = \frac{|\mathbf{r}_1|\cdot\omega_1}{|\mathbf{r}_2|\cdot\omega_2} = \frac{|\mathbf{r}_1|\cdot\omega_1}{\frac{1}{2}|\mathbf{r}_1|\cdot 4\omega_1} = \frac{1}{2} \quad\Leftrightarrow\quad s_2 = 2s_1$$

By similar logic, it follows that the force required to stop the swinging mass is increased in inverse proportion to the change in cord length.

$$-\mathbf{L}_1 = \mathbf{r}_1\times-\mathbf{F}_1 = \mathbf{r}_2\times-\mathbf{F}_2 = -\mathbf{L}_2\ ;\quad \mathbf{F}\perp\mathbf{r}$$
$$|\mathbf{r}_1|*|\mathbf{a}_1| = |\mathbf{r}_2|*|\boldsymbol{a}_2|$$
$$r_1*a_1 = r_2*a_2$$
$$a_2 = \frac{a_1}{\eta} \quad\Leftrightarrow\quad |\mathbf{F}_2| = \frac{|\mathbf{F}_1|}{\eta}$$

A Clinical Example: Back Strain

One of the most puzzling aspects of back injury is that one can cause a significant injury doing a lifting action that previously caused no problem. It is proposed here that one possible mechanism for such a injury is that there is a sudden shift in the instantaneous axis of rotation (IAR) of one disc upon another causing a rapid change in the loads being experienced by muscles and ligaments and of the stress patterns in the disc itself. Such sudden changes in the axis of rotation will occur when two hard surfaces come into abutment and the impinging structures are forced to rotate about the new point of contact. Normally the axis of rotation of one disc upon another lies approximately through the center of the disc. If the two discs are being allowed to approximate anteriorly, by a flexion movement, then the two anterior rims will eventually abut and the axis of rotation suddenly shifts to the anterior margins of the discs. When this happens the anterior part of the disc suddenly shifts from a state of compression to a state of tension. The posterior part of the disc is now about twice as far from the fulcrum of the action and the stress is consequently about twice as great. Similar changes in the stress upon soft tissues will apply to the muscles and ligaments that surround the spinal canal.



Also, notice that the direction of movement is more horizontal with the IAR located at the margin of the disk, which means the force couplings that held the movement in check prior to

the shift will be unable to do so after the abutment. The upper vertebra will begin to shear forward relative to the lower vertebra.

Because of the sudden change in the tension through the muscles, they are forced to elongate until their internal tension is once again equal to the demands of the job. That initial increase in tension is largely due to passive stretching of the muscle. Subsequently, given time for the situation to be detected by the Golgi tendon organs and the muscle spindles and conveyed back to the spinal cord, the motor neuron pool for the muscle may be able recruit more contractile elements and the tension is shifted back to the active tension of the muscle. In the mean time, the sudden stretching of the contracted muscle will cause the contractile actinomyosin arrays to slip internally, possibly causing tearing in the muscle fibrils.

The muscles usually control joint position and the orientation of the bones, therefore we would normally expect the ligaments and joint capsules not to be stressed. However, if the muscle is suddenly passively lengthened, then the separation of the discs may be great enough and fast enough to place a substantial tensile force through a ligament or joint capsule to cause a tearing of its tissues.

Such catastrophic stretching of soft tissues may also apply to other structures in and around the spinal column, so there might be tearing of capillaries or small arterioles or venules, perhaps of nerves. Most tissues will stretch to accommodate to stress, but they may be more readily torn by sudden high tensions that by a gradual build up of tension.

The force generated in a stretched tissue is proportional to the elasticity of the tissue.

$$\mathbf{F} = k \circ \Delta \mathbf{L} \text{ ; where } \mathbf{L} \text{ is the length of the structure.}$$

If we invert this relationship, then we can express the dependence of length upon the force use to stretch the structure.

$$\Delta \mathbf{L} = \frac{|\mathbf{F}_L|}{k} \quad \Rightarrow \quad \frac{d\mathbf{L}}{dt} = \frac{1}{k} \circ \frac{d\mathbf{F}}{dt}$$

However, the linear relationship between the force and the length of the soft tissue is an equilibrium relationship. If the length of the tissue were monitored during a change in force, one would expect there to be a relaxation time. It is not unreasonable to assume that the time course

of the relaxation would be approximately exponential. Therefore, it is likely that the viscosity of the soft tissue would be proportional to the rate at which the length is changing.

$$-\upsilon \circ \frac{d\mathbf{L}}{dt} + k \circ \Delta\mathbf{L} = \mathbf{F}$$

The viscosity would probably be variable in a manner that reflected the contractile tension in the muscle. The constant for steady state tension would also be related to the contractile tension in the tissue. In tendons and ligaments the values would be relatively fixed, although there may be some minor changes with repeated use. Probably most of the loosening up that comes with warming up is due to changes in the contractile tension of the muscle, changes in tone. Tone is a neural attribute and it takes time to react to changes in the stresses in the muscles, tendons and ligaments. For very rapid changes there is not time for adaptive changes from the nervous system.

Implications

By shifting the axis of rotation, we change the manner in which the rotating object is moving. We change the speed with which it is moving. We change the forces that it impressing onto other structures. For instance, if a weight is causing a joint to rotate about its center and there is a sudden limiting of its movement by a ligament or an abutment, so that it is rotating about an edge, then the same force is suddenly producing twice the torque, because of the doubled lever arm length. Similarly, with the same forces and energy, the velocity of a movement may change drastically, faster than the nervous system can react to brake the movement. During that uncontrolled interval, there may be injury.

Normally, we operate our joints in ranges that do not include the catastrophic transition or if we are including it, we built it into the control of the movement. The catastrophic transition is apt to be destructive only when it is unexpected or particularly abrupt.

Some ligaments and joint capsules are built to restrain movement continuously: medial and lateral collateral ligaments of the knee, ligaments that bind the hand and foot bones together) and some to provide restraint only when further movement in a particular direction is potentially injurious (alar ligaments of odontoid process, ligaments of the hip joint). Many ligaments are so

restrictive as to prevent movement between bones at all times or to allow it to occur primarily to absorb stress (pelvic ligaments), .