2 Biomechanics and the Mathematics of Positioning

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The proper flow of blood and other bodily fluids is crucial to achieving successful surgical outcomes and maintaining patient health. In general, neutral positions of the body, head, and neck are recommended in order to achieve optimum blood flow [1]. Despite this, other positions may be necessary or preferred for particular surgeries, and in these cases it is important to understand how these positions affect flows in the head and brain.

The major flows in the head and brain can be modeled by considering the major sources and sinks of fluid, namely arterial flow into the head, venous flow out of the head, and cerebrospinal fluid (CSF) production. Let P_a denote the mean (carotid) arterial pressure, P_d denote the mean pressure in the dural venous sinuses (equivalent to the pressure at the top of the internal jugular), and P_{CSF} denote the component of intracranial pressure (ICP) due to the formation of CSF. Then the ICP is given by [2]

$$P_{ICP} = P_{CSF} + P_d, \tag{2.1}$$

the cerebral perfusion pressure (CePP) is given by

$$P_{CePP} = P_a - P_{ICP} = P_a - P_d - P_{CSF}, \tag{2.2}$$

and the capillary perfusion pressure (CaPP) in the head is given by

$$P_{CaPP} = P_a - P_d. \tag{2.3}$$

Following [2], $P_{CSF} = R_{out} I_{formation}$, where R_{out} is the resistance to outflow of CSF and $I_{formation}$ is the formation rate of CSF. $I_{formation}$ can be expected to be around 0.45 mL/min [3] while values of R_{out} below 13 mmHg/(mL/min) are considered normal [4]. Thus, P_{CSF} can be expected to be around 5.8 mmHg or below. In [2], average P_{CSF} was 5.7 mmHg. Under normal circumstances, P_{CSF} can be taken to be constant [5], and hence we only need to determine the effects of position on the arterial and venous pressures. Furthermore, P_{CePP} and P_{CaPP} differ by a constant amount and so any trend in one is also displayed in the other.

It is now necessary to define some geometrical parameters to describe the position of the patient as well as their head and neck. We can first describe the patient's basic position as supine, prone, or lateral. In general, the arterial pressure is not affected by this basic position [6–8], while the venous pressure can be expected to be the same in either the supine or lateral position, but raised by about 2 mmHg in the prone position [6, 7]. By ignoring the arrangement of the patient's arms and legs, their remaining body position can be described by a single parameter, namely the *tilt angle* τ. τ is the angle between the horizontal and the line from the heart through the center of the neck and head (see Fig. 2.1), with positive values corresponding to head-up tilt and negative values corresponding to head-down tilt.

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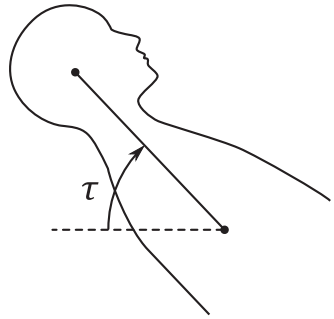

Fig. 2.1 The *tilt angle* τ that describes the angle of the patient's body relative to the horizontal (supine position shown)

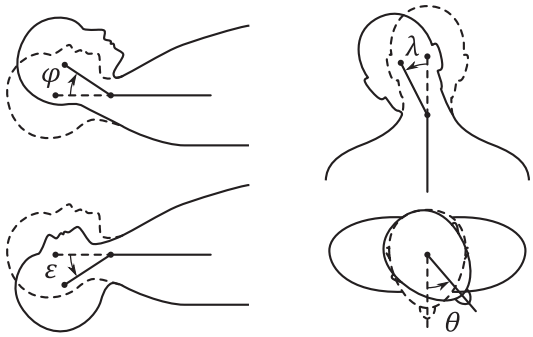

Fig. 2.2 The four angles that describe the position of the patient's head relative to their body. These are the *flexion angle* φ, the *extension angle* ε, the *lateral flexion angle* λ, and the *rotation angle* θ

Four angles can be used to describe the position of the head and neck. These are the *flexion angle* φ, the *extension angle* ε, the *lateral flexion angle* λ, and the *rotation angle* θ (see Fig. 2.2). Although the flexion and extension of the neck could be described by a single angle, the following model of blood pressure becomes simpler when two separate angles are used. Furthermore, for simplicity we will assume that the neck is never rotated and laterally flexed at the same time. Then λ is the angle between the line from the heart to the top of the neck (extended) and the line from the top of the neck to the top of the head, projected into the coronal plane. Positive values of λ correspond to lateral flexion toward the patient's right and negative values correspond to lateral flexion to the patient's left. θ is the angle between the line from the center of the head through the center of the face, projected into the transverse plane, and the line through the center of the head perpendicular to the coronal plane. Positive values of θ correspond to head rotation toward the patient's left, and negative values correspond to rotation to the right.

Finally, φ is the angle between the line from the top of the neck to the center of the head and the intersection of the coronal plane with the transverse plane of the head, whenever the head is bent toward the chest (and thus φ is always either positive or zero). ε is the angle between the line from the top of the neck to the center of the head and the intersection of the coronal plane with the transverse plane of the head, whenever the head is bent toward the back (and thus ε is always positive or zero).

It is a well-established principle that within a communicating fluid system[1], gravity will create a hydrostatic pressure gradient [9]. The hydrostatic pressure difference between two points in the system is given by

$$\Delta P = \rho g \Delta h, \tag{2.4}$$

where ρ is the fluid density, g is the acceleration due to gravity, and Δh is the difference in height between the two points. Thus, we can expect that both the arterial and venous pressures will decrease toward the head for positive tilt angles, and increase toward the head for negative tilt angles. By itself, this understanding is only sufficient to determine pressure differences rather than actual pressure at any point in the body (particularly since the circulatory system is able to respond to changes in body position). To proceed, we must utilize the concept of a *hydrostatic indifference point* (HIP), that is, a point at which the hydrostatic pressure remains the same no matter the orientation of the system [9]. Then, if the blood pressure at a zero tilt angle is known (i.e., when there is no hydrostatic gradient present), the pressure change to any given body orientation is governed by the change in height relative to the HIP.

[1] A fluid system is called communicating if fluid is freely able to pass between any two points in the system.

The HIP for arterial flow to the head is located just above heart level [9], and so the heart location can reasonably be taken to represent the arterial HIP. The venous HIP, in contrast, is located around the level of the diaphragm [2]. Thus, we can model the effect of tilt angle on arterial pressure as

$$P_a = P_a\left(0^\circ\right) - \rho g L_{\text{heart}} \sin\left(\tau\right), \tag{2.5}$$

where $P_a(0^\circ)$ is the arterial pressure at zero tilt angle—i.e., in a "standard" supine, prone, or lateral position, ρ is the density of blood, g is the acceleration due to gravity, and L_{heart} is the distance between the center of the head and the heart. Based on standard values, $\rho g \approx 78$ mmHg/m.

The model for venous pressure is complicated by the fact that as venous pressure in the jugular falls to zero, the vein can collapse and divide the venous flow in the head from the rest of the body [10]. In this case, the reference point for venous pressure in the head is no longer the venous HIP, but the point of collapse of the jugular. Although there are other venous pathways flowing out from the head (thus maintaining *some* communication with the rest of the venous system), observations indicate that blood flow is divided between those and the jugular so as to maintain zero pressure at the point of jugular collapse [2]. Thus, the effect of tilt on venous pressure becomes

$$P_d = \begin{cases} P_d(0^\circ) - \rho g\left(L_{\text{heart}} + L_{\text{heart–HIPvein}}\right)\sin(\tau), \tau < \tau_{\text{collapse}} \\ -\rho g L_{\text{collapse}} \sin(\tau + \eta), \tau \geq \tau_{\text{collapse}} \end{cases} \tag{2.6}$$

where $P_d(0^\circ)$ is the venous pressure at zero tilt angle, $L_{\text{heart – HIPvein}}$ is the distance between the heart and the venous HIP (around the level of the diaphragm [2]), L_{collapse} is the distance between the center of the head and the point of collapse of the jugular,

$$\eta = \begin{cases} \varphi - \varepsilon, \text{supine position} \\ \varepsilon - \varphi, \text{prone position} \\ \lambda, \text{left lateral position} \\ -\lambda, \text{right lateral position} \end{cases} \tag{2.7}$$

is the neck tilt angle and τ_{collapse} is the tilt angle at which the jugular first collapses. Note that $P_d(0^\circ)$ can be expected to be around 2 mmHg higher in the prone position compared to the supine or lateral positions [6, 7]. From the definition of the various parameters, we can solve for τ_{collapse} as

$$\tau_{\text{collapse}} = \sin^{-1}\left(\frac{P_d(0^\circ)}{\rho g\left[L_{\text{heart}} + L_{\text{heart–HIPvein}} - L_{\text{collapse}}\right]}\right). \tag{2.8}$$

Based on fitting done in [2], we can assume that in general, $L_{\text{heart – HIPvein}} \approx 0.09$ m and $L_{\text{collapse}} \approx 0.11$ m. The neck tilt angle is only required for calculations following collapse of the jugular since the reference point in this case is located within the neck. Note that according to Eq. (2.6), P_d will be negative for a range of tilt angles (even for some angles below τ_{collapse}). There is no theoretical problem with this since all pressures are measured relative to ambient (i.e., atmospheric) pressure and so negative values simply indicate pressures below ambient.

We can reproduce some trends seen in the literature when we apply this model of gravitational effects to the expressions for ICP and CePP. As reported in numerous studies (e.g., see [2, 11–14]), ICP decreases with increasing tilt angle. This effect is seen in the model by the hydrostatic decrease in venous pressure with increasing tilt angles. However, CePP has been reported both to decrease with tilt angle in some studies (e.g., [13]) and to be unaffected by tilt angle in others (e.g., [11, 12]). An examination of the current model shows that CePP can actually be expected to *increase* with tilt angle for $\tau < \tau_{\text{collapse}}$ (due to the greater distance between the head and the venous HIP compared to the arterial HIP) but then decrease with tilt angle for $\tau > \tau_{\text{collapse}}$. Furthermore, the value of τ_{collapse} can vary significantly between individuals [2], presumably due to the wide variation in venous pressure at zero tilt. Thus, the differences between studies can be attributed in part to individual patient differences and in part to other factors that influence blood flow (e.g., the study in [13] was conducted on

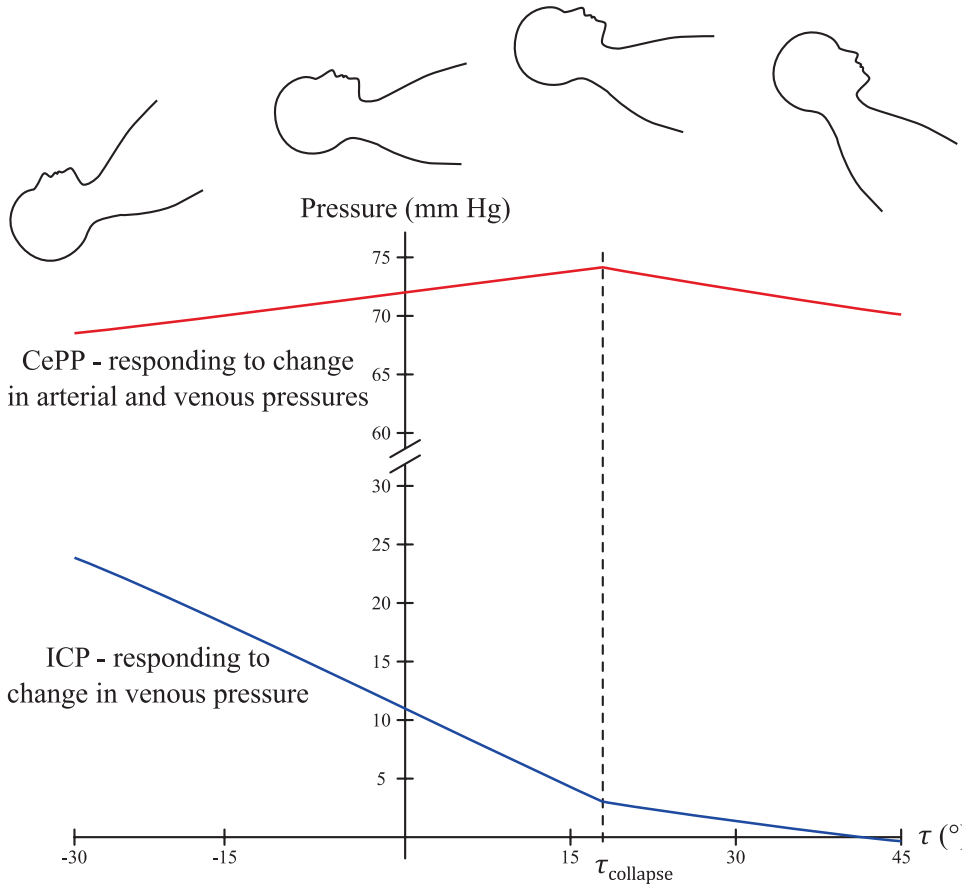

Fig. 2.3 A numerical example of the effects of tilt angle τ on intracranial pressure (ICP) and cerebral perfusion pressure (CePP). In this example, the patient is supine with their neck in a neutral position, $P_a(0^\circ)$ = 83 mmHg, $P_d(0^\circ)$ = 5.3 mmHg, P_{CSF} = 5.7 mmHg, L_{heart} = 0.24 m, $L_{heart\ -\ HIPvein}$ = 0.09 m, and $L_{collapse}$ = 0.09 m. ICP always decreases as τ increases, but the rate of decrease diminishes for $\tau > \tau_{collapse}$. CePP, and thus flow through the brain, has a maximum at $\tau = \tau_{collapse}$

patients who had suffered middle cerebral artery stroke). See Fig. 2.3 for an example of ICP and CePP changing with τ.

In contrast to the effects seen in this model for positive tilt angles, negative tilt angles can be expected to increase the ICP (due to the hydrostatic increase in venous pressure) and decrease the CePP (due to the venous pressure increasing more rapidly than the arterial pressure). Thus, this model predicts that in general, any position with head-down tilt can be expected to impair blood flow in the head.

While the hydrostatic effects of body position on blood pressure are relatively well understood, the effects of head and neck position are more difficult to model. The dominant effect of neck flexion, extension, rotation, and lateral flexion on blood flow appears to be the bending and compression of the blood vessels in the neck [14–19]. In principle, this should manifest as increased resistance to flow and thus greater pressure drops in these vessels. This increased resistance arises from the fact that the ratio of the perimeter to cross-sectional area of a blood vessel increases as the vessel is compressed, as well as the fact that increased blood velocity through the narrower section will lead to increased frictional losses. Therefore, it is reasonable to expect that any head motion away from a neutral position could decrease P_a and increase P_d (since the pressure

drops occur in the direction of flow), leading to increases in ICP and decreases in CePP and CaPP. Furthermore, since veins are much more compliant than arteries, we can expect that the effects on venous pressure to be more significant than those on arterial pressure. In fact, for general patients, normal neck motions appear not to have a significant effect on vertebral arterial flow [17] or central venous and arterial pressure [14]. Moreover, while neck motion can change the geometry of the arteries in the neck and the distribution of flow between them, these effects vary between individuals and there does not appear to be a general trend or a significant effect on flow into the head [20]. This suggests that there is no need to model the effects of neck motion on arterial flow into the head.

Perhaps due to the lower pressures involved and increased vascular compliance, neck motion can have a significant effect on venous pressure and hence ICP [14, 21]. In one study that examined neck flexion, extension, lateral flexion, and rotation [21], every motion away from the neutral position resulted in an increase in ICP, although not all of these individually achieved statistical significance. Nevertheless, the trends were consistent enough to warrant a model that captures effects of each considered motion. Rotation-induced pressure increases were observed to usually be linear with increasing rotation angle [14]. Therefore, in the absence of contrary evidence, we will assume that venous pressure increases linearly with each motion of the neck. Next, it was found that the combination of flexion/extension with lateral flexion or rotation decreased the influence of the second motion [21]. One possible explanation for this is that flexion of the neck slackens the blood vessels somewhat, reducing the bending or compression required for the second motion (lateral flexion or rotation). In contrast, extension of the neck can stretch the blood vessels somewhat, decreasing their compliance and thus decreasing the effects of the second motion. Since the effect of neck motion is to change the resistance to flow of the venous system, the induced change in pressure should be proportional to the pressure difference that is driving flow through the head. Together, all of these effects suggest the following model for venous pressure in response to neck position.

$$P_{\mathrm{d}} = P_{\mathrm{d}}\left(\text{neutral}\right) + \frac{P_{\mathrm{a}} - P_{\mathrm{d}}\left(\text{neutral}\right)}{\Delta P_{\mathrm{ref}}} \left[\begin{array}{l} P_{\varphi_{\mathrm{lim}}} \dfrac{\varphi}{\varphi_{\mathrm{lim}}} + P_{\varepsilon_{\mathrm{lim}}} \dfrac{\varepsilon}{\varepsilon_{\mathrm{lim}}} + \\ \dfrac{\lambda}{\lambda_{\mathrm{lim}}}\left(P_{\lambda_{\mathrm{lim}}} - P_{\lambda_{\mathrm{dec}}}\left(\dfrac{\varphi}{\varphi_{\mathrm{lim}}} + \dfrac{\varepsilon}{\varepsilon_{\mathrm{lim}}}\right)\right) + \\ \dfrac{\theta}{\theta_{\mathrm{lim}}}\left(P_{\theta_{\mathrm{lim}}} - P_{\theta_{\mathrm{dec}}}\left(\dfrac{\varphi}{\varphi_{\mathrm{lim}}} + \dfrac{\varepsilon}{\varepsilon_{\mathrm{lim}}}\right)\right) \end{array}\right], \tag{2.9}$$

where ΔP_{ref} is a reference arterial to venous pressure difference and for each variable x, x_{lim} is a limiting value of the angle and $P_{x_{\mathrm{lim}}}$ is the pressure increment observed at that limiting value (when $P_{\mathrm{a}} - P_{\mathrm{d}}(\text{neutral}) = \Delta P_{\mathrm{ref}}$). Furthermore, $P_{\lambda_{\mathrm{dec}}}$ and $P_{\theta_{\mathrm{dec}}}$ are the decreases in pressure increments for lateral flexion and rotation, respectively, when the neck is flexed or extended to its limiting angle. There is no *a priori* reason why $P_{\lambda_{\mathrm{dec}}}$ and $P_{\theta_{\mathrm{dec}}}$ should apply to both flexion and extension of the neck, instead of requiring separate parameters for each motion (particularly as the two motions individually have different effects on pressure). However, the data collected in [21] suggested that both flexion and extension cause equivalent decreases in the effect of rotation and the effect of lateral flexion. The values for P_{a} and $P_{\mathrm{d}}(\text{neutral})$ in Eq. (2.9) should be those given by Eqs. (2.5) and (2.6), respectively. Since jugular collapse significantly changes the flow through the venous system (and renders any other distal geometrical changes to the jugular irrelevant), the parameter values in Eq. (2.9) can be expected to change for $\tau \geq \tau_{\mathrm{collapse}}$.

Equation (2.9) does not include any direct dependence on blood viscosity because even with geometrical changes to the blood vessels, vascular resistance should be proportional to viscosity. Therefore, even though the resistance of the altered neck vasculature will increase with increased viscosity, the resistance of the rest of the cerebral system will also increase in proportion, leading to the same distribution of pressure. However, since increased blood viscosity leads to

decreased flow, patients with higher blood viscosities can be expected to have higher arterial pressures as the body attempts to maintain cardiac output. This will, in turn, increase the effects on neck motion on venous pressure and thus lead to higher values of ICP.

Based on the results in [21], we can estimate that for general patients and $\tau < \tau_{\text{collapse}}$, $\Delta P_{\text{ref}} \approx 80$ mmHg, $P_{\varphi_{\text{lim}}} \approx 4.8\text{mmHg}$, $\varphi_{\text{lim}} \approx 45^{\circ}$, $P_{\varepsilon_{\text{lim}}} \approx 1.5\text{mmHg}$, $\varepsilon_{\text{lim}} \approx 60^{\circ}$, $P_{\lambda_{\text{lim}}} \approx 2.6\text{mmHg}$, $\lambda_{\text{lim}} = 45^{\circ}$, $P_{\lambda_{\text{dec}}} \approx 1.4\text{mmHg}$, $P_{\theta_{\text{lim}}} \approx 4.2\text{mmHg}$, $\theta_{\text{lim}} \approx 60^{\circ}$, and $P_{\theta_{\text{dec}}} \approx 1.9\text{mmHg}$, where some angles are taken from the original study and others are estimated according to the patients' age range using [22]. This suggests that the largest increases in P_{d} and ICP occur with flexion and rotation of the neck, while extension of the neck can actually reduce the pressure rise from rotation. See Fig. 2.4 for an example of how various neck positions affect ICP and CePP. There does not appear to be any existing studies that would allow for the estimation of parameter values for $\tau \geq \tau_{\text{collapse}}$.

Although only rotation was considered in [14], their results suggested a potentially much larger value of $P_{\theta_{\text{lim}}}$, up to 12.5 mmHg. This discrepancy can potentially be attributed to the different patient populations in the two studies, with [21] excluding patients with high ICP, increased CSF volume or any impairment on the Glasgow Coma Scale, while [14] only included patients with intracranial tumors. Since the numerical value of arterial pressure was not reported in [14], it is possible (but unknown) that patient hypertension contributed to the observed effects. Furthermore, the patients with the highest initial ICP in [14] exhibited the largest (and nonlinear) pressure increases. This, coupled with the amount of individual variation seen in arterial studies (e.g., [18, 20]) suggests that this model of the effects of neck motion will not apply to any

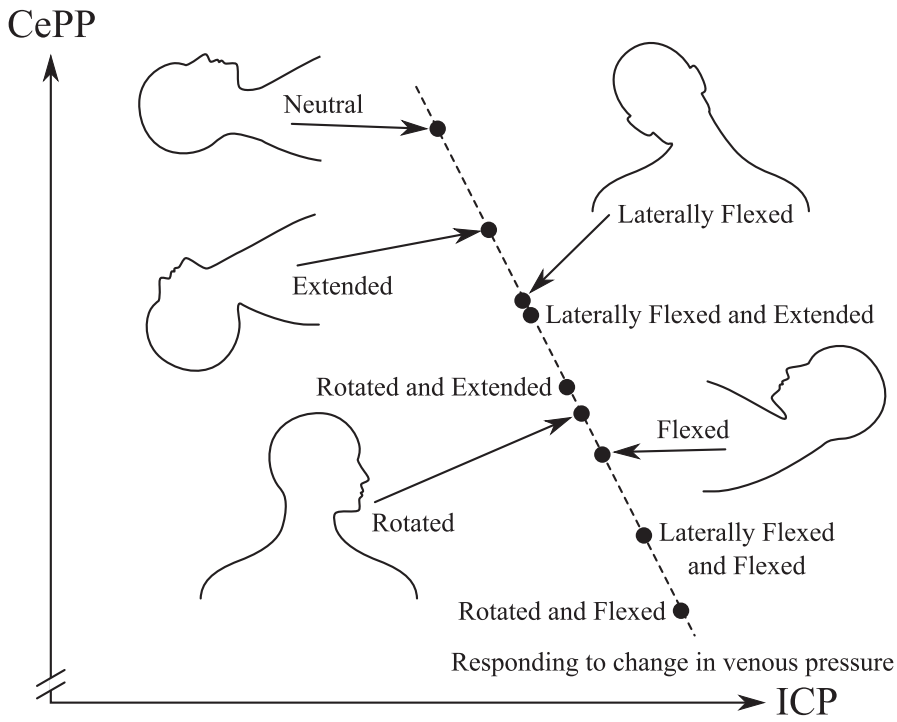

Fig. 2.4 An example of the effects of different neck positions on intracranial pressure (ICP) and cerebral perfusion pressure (CePP). In this example, the patient is supine with zero tilt angle. Compared to the neutral position, any other neck position increases ICP and decreases CePP. All cases shown correspond to the limiting values of the angles used in Eq. (2.9)

patient with existing impairments in blood flow to the head and neck. In principle, any disease state distal to the neck should not influence the model, but only if they do not induce any changes to the geometry or distribution of flow in the neck. Finally, the amount of variation seen in studies of neck motion suggest that there is an increased possibility that positioning the patient's neck without due care could adversely affect blood flow to the head. In particular, moving the head and neck past their normal range of motion (e.g., by hyperextending the neck [23]) can be expected to increase the risk of complications.

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