Isolation of the physiological regulation component of the arterial pressure time variation after postural stresses by a model of the gravitational and arterial kinking effects

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ISOLATION OF THE PHYSIOLOGICAL REGULATION COMPONENT OF THE ARTERIAL PRESSURE TIME VARIATION AFTER POSTURAL STRESSES BY A MODEL OF THE GRAVITATIONAL AND ARTERIAL KINKING EFFECTS

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INTRODUCTION

Prompt active postural manoeuvres induce an immediate arterial pressure variation followed by a period of regulation (4). For the squatting manoeuvre, initial hypertension was explained by a rise of cardiac filling pressure due to "squeezing blood out of the veins of the legs", leading to an increase in stroke output by Frank-Starling mechanism. For a minor part, it was also explained by "kinking" of the femoral arteries. O'Donnel and Mc Ilroy observed an increase in central blood volume and accepted the idea of a rise of cardiac filling (2). However, they did not observe so consistent circulatory variations when postural changes were realized in a water tank. Therefore, they concluded that kinking of the arteries and veins of the legs could not be very important. Moreover, the immediate pressure variations, most often appearing in the first beat succeeding the postural manoeuvres cannot be easily explained by the previously invoked modifications of cardiac filling pressures. When Hoffman *et al* lifted dogs until they stood erect, the right ventricular stroke volume usually fell in the first beat after the postural change, but the left ventricular stroke volume did not fall for another 1-3 beats (1). When the dogs where rapidly lowered to standing on four legs again, a delay of 2-3 beats was also observed.

Thus, another interpretation of immediate hypotension must be added. It should especially take into account the natural gravitational fluid mechanics phenomena imposed to the arterial blood. Besides, to allow the investigation of the orthostatic regulation of arterial blood pressure, it would be necessary to separate the cardiovascular regulation component of the arterial pressure time course from the pressure evolution that would naturally appear in the network without physiological contribution. It is the aim of this study.

HYDROSTATIC MODEL

The gravitational fluid mechanics component of the initial blood pressure fall is first modelized using an hydrostatic approach in a single very distensible tube representing the whole arterial net.

It is shut at both extremities, filled up with liquid under pressure and is first laid horizontally. The pressure diagram is evidently constant in the conduit. The tube is suddenly stood up in the gravity field. To study the impact of the deformability of the wall and for an easier understanding, let us first suppose that to the initial pressure diagram is a priori superposed a triangular diagram due to the weight of the liquid. This situation would not respect hydraulic equilibrium.

In fact, the increase of pressure at the base generates a dilatation of the conduit and consequently a movement of liquid towards the base. The weakening in fluid in the superior part develops a constriction and a drop of pressure. The hydraulic load being weaker at the top than at the base, the fluid is then drawn upwards and consequently, a decrease of section and of pressure at the base appears. Thus, compared to the supposed diagram of pressure, the respect of the hydrostatic condition of constant load in the equilibrium status towards the system tends, progressively induces a drop of pressure linked to the deformability, identical on all the fluid height. The final configuration will correspond to the figure 1.

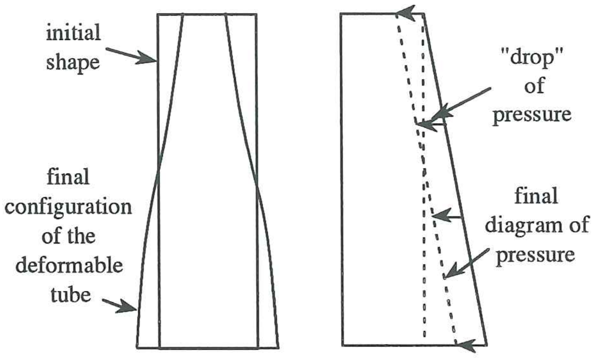

Figure 1. Final geometrical configuration of a flexible conduit after standing it up and corresponding pressure diagram.

The equality of the volumes of fluid can be used to calculate this drop of pressure. Supposing the horizontal conduit prismatic and the cross-section area only function of the pressure, the conservation of the volume between the horizontal and vertical positions lead to the following conclusion. In the median case where the cross-section area is a linear function of the pressure, the drop is exactly equal to the half of the hydrostatic difference of pressure between the top and the base of the tube. The drop is then independant of the rheological law of the wall. It is easy to show that in this particular situation, the diagrams of pressure along the tube have the same area in both geometrical cases. In general, the drop of pressure oscillates around the median value, according to the concavity of the cross-section - pressure law (3).

When this study is realized for more complicated postural changes, such as those described on figure 2 for the sitting to standing and squatting to standing manoeuvres, similar conclusions can easily be derived. The notation h representing the length along the tube from the top down to the base, the median drop of pressure at the level of the heart or of the finger, which of course depends on the manoeuvre, is expressed (with ρ the volumic mass and g the gravity)

for the **sitting to standing** manoeuvre by:

$$\Delta p = \frac{\rho g}{2 h_4}\left(-h_1(2h_4 - h_1) + h_2(2h_4 - h_2)\right)$$

and for the **squatting to standing** manoeuvre by:

$$\Delta p = \frac{\rho g}{2 h_4}\left((2h_4 - d)(h_3 - h_1) - h_3^2 + h_1^2 - (h_4 - h_3)(h_4 - h_3 - 2d)\right)$$

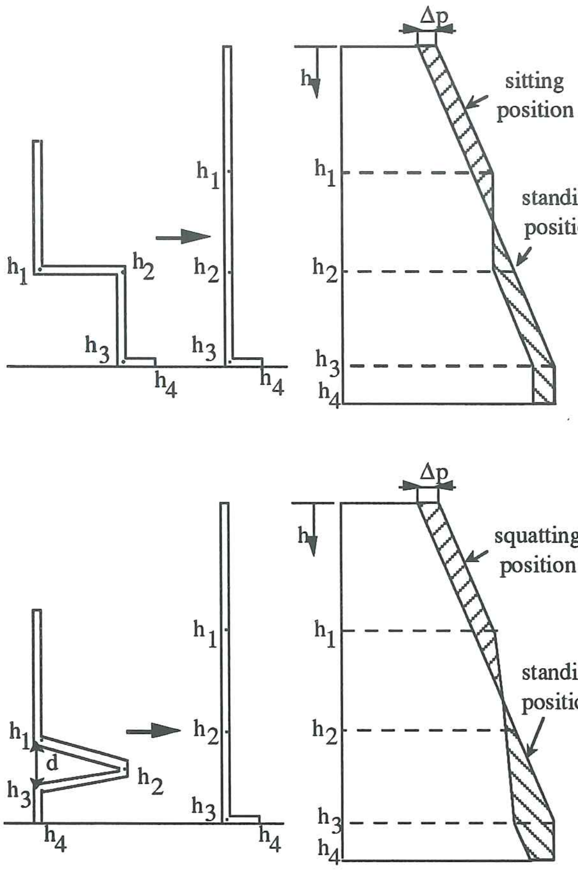

Figure 2. Modelization of the sitting to standing manoeuvre (up) and of the squatting to standing manoeuvre (down) with corresponding pressure diagrams.

EXPERIMENTAL COMPARISON

Both manoeuvres were performed by a set of 36 adult, healthy, normotensive subjects. Arterial blood pressure was continuously measured at a finger by means of the non-invasive Finapres device (Ohmeda) based on the Penaz's photoplethysmographic technique. During the whole duration of measurements, the finger cuff was maintained at heart level. Beat-to-beat mean arterial pressure (MAP) was calculated.

Change from sitting to standing position induces a very reproductible and prompt decrease of MAP with a nadir of -15 (SEM 3) mmHg after about 7 sec. Considering a mean individual (h_1=0.70m, h_2=1.15m, h_3=1.70m, h_4=1.90m), our model leads to a drop due to gravity of 18 mmHg. This is slightly higher than the experimental value, suggesting the beginning of the effects of regulation.

The mean experimental decrease for the squatting to standing manoeuvre is 32 (SEM 9) mmHg, also appearing 7 seconds after the postural change. The hydrostatic model leads in the same conditions to a fall of 25 mmHg (with d=0.2m). The main origin of the difference can be found in the crushing of the arteries in the hollow of the knee due to the weight of the body. Equilibrium calculation in the squatting position, associated with assumptions on the concerned region, allows to estimate the induced exterior pressure on these arteries. In the hydrostatic model (fig. 2), the pressure must then be replaced by the transmural pressure which increases the pressure fall. Our first simplified estimations lead to an increase of 7 to 12 mmHg, which is confirmed by our first studies on leg crossing.

The total fall is then of about 35 mmHg, again slightly higher than the experimental one.

HYDRODYNAMIC MODEL

To go further, the time course of the mean pressure due to the gravitational effects was simulated using a fully non linear one dimensional fluid mechanics model. The geometrical configuration of the tube changes like the body one. Two numerical methods are used: a fixed grid method of characteristics and a Petrov-Galerkin finite element method with special test functions and prediction-correction. Technical elements and data are detailed in (3). For the sitting to standing manoeuvre, figure 3 gathers the simulated pressure evolution as estimated by the model and a representative recorded curve of mean pressure.

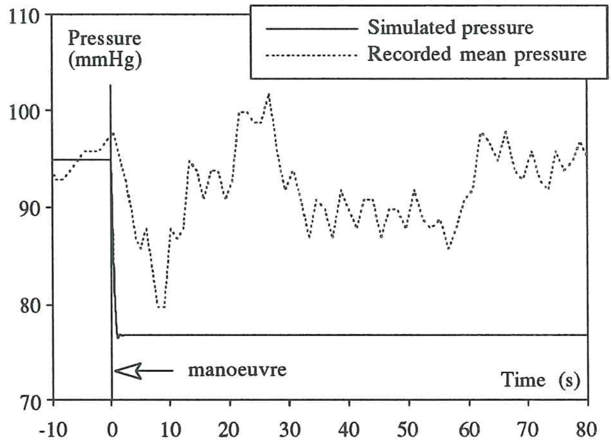

Figure 3. Simulated pressure evolution due to gravitation and a representative recorded mean pressure curve.

CONCLUSIONS

In spite of the geometrical simplicity of the model, it proves that the gravity impact on the arterial network, sometimes associated with the arterial kinking, plays an essential rôle in the initial pressure variation observed after the studied postural manoeuvres. Of course, in each individual case, the influences are combined with the others (cardiac filling, respiration...).

The difference between the simulated curves and the measured ones should be used for physiological interpretation, since such an approach can help in the quantification of the physiological regulations induced by postural stresses. To allow an efficient individual use, this approach still need to be improved in several ways : a more geomotrically representative network, adaptation of the model to the personal morphology, in some aspect superposition of the flow, standardisation of the manoeuvres...

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