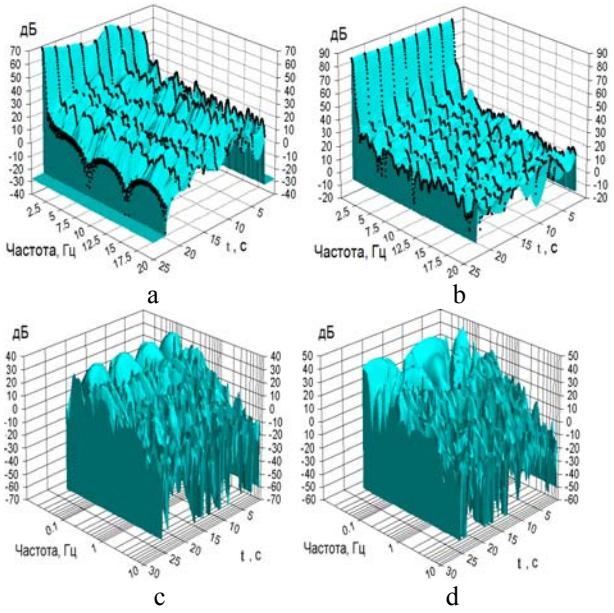

Fig.2. DSTFT/ DWT distributions of the $x_{COM}(t)$ (a/c) and $y_{COM}(t)$ (b/d) curves of the two-leg stance of a healthy individual.

$\sim(\theta_j)^2$ one can obtain the quasi-linear system of ODE governing oscillations of the segments in the form

$$M\cdot\frac{d^2}{dt^2}\vec{\theta}+K(\frac{d}{dt}\vec{\theta},\vec{\theta})+N\cdot\vec{\theta}=\vec{u}(\frac{d}{dt}\vec{\theta},\vec{\theta}) \qquad (1)$$

where $\vec{\theta}^T=(\theta_1,\theta_2,\theta_3,\theta_4)$, T is transposition sign, M is the mass-inertia matrix, K is centrifugal matrix, N is gravity matrix, $\vec{u}$ is the control function which is usually supposed to be proportional to deviations of angles and velocities. Components of the matrices M, K, N are long complex expressions which are not presented here for brevity. The quasi-linear system (1) has been solved numerically. Amplitudes and frequencies of body oscillations have been computed, analyzed and compared to the measured data.

It was sown the COM trajectories during the two-leg and one-leg stances, as well as at the step forward off the force platform by the left/right leg contain important information for differential clinical diagnostics of the spine and joint pathologies.

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FLOW–WALL INTERACTION IN THE ANEURYSM MODEL

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The aneurysm occurs when part of a blood vessel inflates abnormally due to damage or weakness in its wall. As the wall is weakened, it balloons out under the action of transmural pressure at its weakest point creating a saccular or fusiform bulge called aneurysm. As the aneurysm grows, the deformation and the displacement of the wall become large, the stress increases which leads to the rupture of wall of the aneurysm. The aneurysm is likely to form as a result of a biological processes caused by biochemical or structural inherited defects, infection disease and specific hemodynamic factor [1]. The fact that the aneurysm often occurs at a specific location in arteries and veins, characterized by unique hemodynamic conditions, strongly suggests that the hemodynamic plays an important role in the formation and in the development of the aneurysm [2].

Several nonlinear mathematical models describing the long term evolution of the aneurysm have been developed in the literature, in those models, the replacement rate of elastin fibers by collagen fibers is supposed to be dependent on hemodynamic conditions. As the time scale of the dynamic considered by these models, which is of the order of several years, is very large in comparison of the heart cycle, which is of the order of one second, the hemodynamic conditions (pressure and stress) are found by solving Navier–Stokes equations in a fixed domain. The fixed domain is updated every once in a while to take account to the evolution of the geometry of the aneurysm. Therefore, they implicitly supposed that the movement of the wall does not affect the flow. The present work is mainly concerned by the action of the movement of the wall on the flow and vice versa, therefore, a full interaction between the flow and the wall is considered. The aim of the simulation is to predict the tension in the elastic moving wall of the aneurysm for inferring its rupture

and to compute the shear stress at the elastic moving wall. The latter, namely the shear stress, is used by the endothelial cells as a sensor allowing the arteries to update their geometrical form, their wall thickness and their diameter to adapt to the hemodynamic status.

A simplified nonlinear integral-partial-differential equation is devised here to describe the aneurysm's wall dynamic. In order to handle large displacement and large deformation, the Lagrangian variables are used, as they are most suited to describe large displacement associated with large deformation. In some interesting particular cases a formal solution of the nonlinear integral-partial-differential equation governing the wall motion is found. In peculiar, the obtained solution shows that the form of the wall under a constant transmural pressure is an arc of a circle in 2d case. A first integral of the wall equation is found when inviscid flow velocity is quasi uniform. i.e. the axial velocity component is much larger than the span-wise velocity component.

A digital simulation taking into account a full coupling between the wall and the flow inside the aneurysm has been made. The wall motion is described by the mentioned integral-partial-differential equation written in Lagrangian representation, and the flow is described by two dimensions Navier–Stokes equations. Three categories of numerical experiments are done here. In the first experiment the curve aneurysm wall is maintained fix (supposed to be rigid) and the inlet flow is a steady one. It is found that the flow in this case is a steady one, and that for both Reynolds number Re=1500 and Re=2000. It is found that two quasi symmetric counter rotating rolls take places under the curve aneurysm walls, inverting by the way the direction of the shear stress at the fluid/solid interface (Fig. 1).

The second experiment is done using a steady inlet flow and elastic wall, i.e. the wall allowed to move under the variation of transmural pressure over time. It is found that the flow is asymmetric in this case. The flow is found to be an unsteady one for Re=1500 and Re=2000 and transmural pressure to density ratio Dp/r=10 m^2/s^2 as well as for Re=1500, Re=2000 and transmural pressure to density ratio Dp/r=15 m^2/s^2. However, the wall displacement and the variation of the streamlines of the flow over time are very small. Two asymmetric rolls appear under the curve aneurysm wall, which inverts the direction of the shear stress exerted by the flow on the wall. Fig. 2. As the symmetry is broken in this case, it can be inferred that the friction forces in the vicinity of the walls must be of different amplitude which could lead to asymmetric evolution of the aneurysm shape over long period.

In the third experiment a periodic pulsatile flow at the inlet of the channel and a curve elastic wall are involved. After a long period so that the effect of the initial condition are removed, the flow becomes quasi periodic. Samples, in six stations in the laps of one period, are selected to examine the flow. Two sub cases are considered here, in the first one a relatively long aneurysm subjected to a relatively low transmural pressure to density ratio is considered. In the second one a relatively short aneurysm subjected to a relatively high transmural pressure to density ratio is considered. In both case the flows obtained by numerical simulation are asymmetric. Fig. 3. The presence of two rolls under the curve walls is confirmed in these cases.

However, during the systolic period, the rolls are quasi removed and the flow tends to be unidirectional.

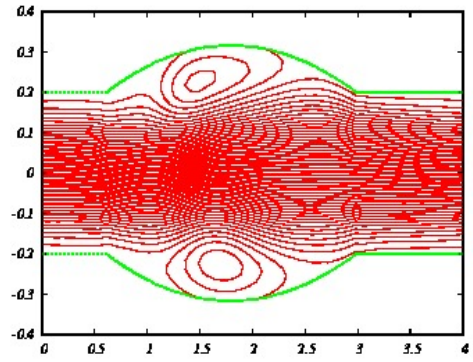

Fig. 1. Streamlines obtained with rigid wall ans steady inlet flow. Re= 1500.

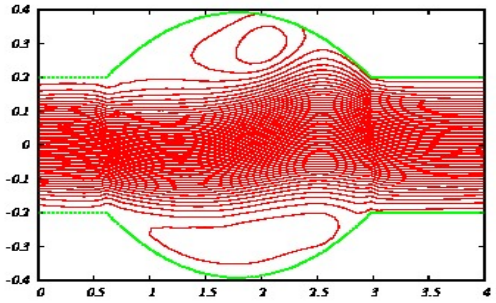

Fig. 2: Streamlines obtained with elastic wall and steady inlet flow. Re=1500

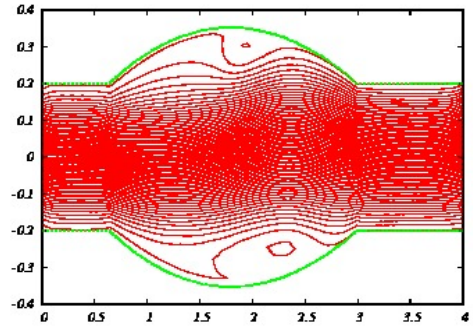

Fig. 3. Streamlines obtained with elastic wall and pulsatile inlet flow.

An interesting feature of the pulsatile flow which appears in the present numerical experiments is that, there is a gap between the instant where the inlet rate flow reaches its maximum and the instant where the maximum expansion of the wall occurs. i.e. the maximum of the inlet flow and the maximum of walls displacement do not occur simultaneously.

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