Letter to the Editor Re “Measurement of the uniaxial mechanical properties of healthy and atherosclerotic human coronary arteries”

Dear Editor,

We would like to comment on the above titled paper which recently appeared in Materials Science and Engineering C 33:2550–4 (2013). First we refer to deficiencies in the analysis and interpretation of the results, and then we follow this with some brief comments on the inappropriate experimental protocol used. It is important to highlight the problems with this paper so that unsuspecting readers are not tempted to perpetuate the errors.

The authors refer to the Young’s modulus, which is a concept borrowed from linear elasticity, but it is inappropriate for the deformations involved in soft biological tissues such as coronary arteries. It seems that the authors mean by Young’s modulus the value of the tangent at a point on the stress–strain curve – for the physiological modulus at the point corresponding to the mean arterial pressure – and for the maximum modulus the point of inflection on the stress–strain curve (incidentally, there are no stress–strain curves plotted in the paper). Young’s modulus is not a useful concept in nonlinear elasticity, and in any case it is dependent on the choice of stress and strain measures, which the authors do not make explicit (in particular, in Tables 1 and 2). They refer to ‘maximum stresses’ but they have not indicated how these were calculated — the data they have shown is only for the forces. They do refer to the true stress in one or two places, which presumably means Cauchy stress, and they also refer to true strain, but this is not defined.

They say “It is believed that the morbidity of coronary artery is related to the magnitudes of stress and strain within the arteries; thus, quantification of these values is of great importance”. Again it is unclear what these values are meant to be — what measures of stress and strain are used? Of course, if the stresses or strains are very high then failure of an artery will result, but all this needs to be quantified, not hidden in vague statements without specification of the measures used.

It is mentioned that “In the studies related to the mechanical properties of carotid or aorta found in the literature, the true stress versus engineering strain or engineering stress versus engineering strain are usually used for calculations.” and the following statement related to this is completely false: “However, this method is only valid for the strains of less than 10%. Since most of the uniaxial tensile tests of human tissues have shown deformation of more than 50%, it is not possible to assess the real mechanical behavior of tissues with this method.” This represents a lack of knowledge of nonlinear mechanics and taking for granted what is mentioned in the paper [18]. True stress and engineering strain are perfectly valid measures in nonlinear elasticity and can be found in standard books on the subject.

In Section 2.2 the authors indicate that for an incompressible material Poisson’s ratio is 0.495, and this is repeated on page 2554 with the statement “Here, the human coronary arteries are considered incompressible, meaning that the Poisson ratio is 0.495 which enables us to report the true stress and strain values.” Poisson’s ratio is 0.5 for an incompressible material and the statement “which enables us to report the true stress and strain values.” is meaningless. The phrase “the spontaneous dimensions of the tissue.” following this also has no meaning.

Another false assertion: “An important point to be noted is that most investigations on the mechanical properties of the arteries have adopted a rigid artery wall assumption which could be acceptable for clinical studies of atherosclerosis, small arteries, and coronary laminar flow simulations.” This refers to hemodynamic studies and it was true maybe 30 years ago but there have been many studies of hemodynamics which take the wall of the artery to be elastic, either linearly elastic or nonlinearly elastic.

The force-displacement plots in Fig. 3 are of no value. First, no information about the initial dimensions of the individual specimens is identified, so the cluster of curves tells us very little. Second, the undeformed areas of the cross-sections of the specimens on which the forces were applied should be recorded, and the engineering stress reported, not the force. Third, the initial length of each specimen is needed so some measure of strain can be calculated, without which the extension has no meaning.

In the Conclusions section there are vague statements that are just dreamlike and illustrate the lack of knowledge of the authors. One example is “The results of this study can be used for modeling and simulation of plaque vulnerability using accurate methods such as finite element analysis.” There is nothing in the paper related to plaque vulnerability. Such studies would first of all need constitutive models to describe all the materials involved, and there are no such models presented in the paper, and no relevant tests or data that could be used for the identification of plaque vulnerability. In any case analysis of plaque vulnerability is very delicate and complex. There is nothing in the paper that could be used for this purpose.

The experimental protocol reported in the paper is completely unsuited to evaluating the properties of human arteries. Material properties vary significantly along the length of an artery. The specimens are highly non-uniform in shape (exemplified in Fig. 2). The method of measuring extension is suspect — to measure the extension of the specimen by the change in distance between the grippers is not good experimental practice. Uniaxial tests in any case convey only very limited information about material properties and tell us nothing about any anisotropy in the material, and coronary arteries are known to be highly anisotropic.

The test results do not correspond to a physiological protocol since inflation seems not to have been used. The authors mention that the “Physiological modulus was derived from the pressure range of 80–120 mm Hg which is considered almost linear in
stress–strain curves [18].” In order to determine the so-called physiological modulus the specimen would need to be inflated to the pressure in question, but the authors have not reported that they applied any pressure, only the uniaxial stress, so how can they derive the modulus?

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