Introduction:
The autonomic nervous system (ANS) controls both heart rate and vascular tone, which are known to be impaired during septic shock (SS). Acute inflammation is presumed to increase arterial stiffness of large arteries in experimental studies [1]. The objectives of this work are to verify if standard SS resuscitation modulate mechanical vascular properties and to verify if alterations in these vascular properties and ANS activity are correlated.

Methods:
A protocol of fecal peritonitis septic shock and standard resuscitation (fluids and noradrenaline) was applied on 6 pigs. The arterial blood pressure waveform was recorded in the central aorta and in the femoral and radial arteries. The characteristic arterial time constant $\tau$ was computed at the three arterial sites, based on the two-element Windkessel model [2]. The total arterial compliance (AC) and the total peripheral resistance (TPR) were also estimated. Baroreflex sensitivity (BRS), low frequency (LF, 0.04-0.15 Hz) spectral power of diastolic blood pressure, and indices of heart rate variability (HRV) were computed to assess ANS functionality.

Results:
Septic shock induced a severe vascular disarray, decoupling the usual pressure wave propagation from central to peripheral sites, as shown by the inversion of pulse pressure (PP) amplification, with a higher PP in the central aorta than in the peripheral arteries during shock. The time constant $\tau$ together with AC and TPR were independently decreased. A decrease in BRS, LF power, and HRV describe an ANS dysfunction. After the administration of fluids and noradrenaline, both vascular and autonomic dysfunction persisted and these were found to be significantly correlated.

Conclusion:
Measures of mechanical vascular function and ANS activity could represent an useful end-point to guide further clinical investigations and refine our understanding of SS mechanisms, especially under medical treatment.

References:
1. Hatib et al., JAP, 111(3), 853-60, 2011