Introduction

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Organic erectile dysfunction is frequently associated with cavernosal arterial vascular disease and/or corporeal veno-occlusive dysfunction (*Krane et al, 1989*) and (*Shabsigh et al, 1989*).

Hemodynamic studies are important in evaluation of patients with erectile dysfunction (*Govior et al, 1995)* and *(Cornio et al, 1996)*. Among various methods of hemodynamic studies; color flow imaging with doppler waveform analysis is currently considered the best (*Levine et al, 1996*), (*Montorsi et al, 1996)* and *(Wegner et al 1995)*.

In view of the central role of cavernous smooth muscle relaxation in inducing penile erection, the data suggested that vascular impotence might be linked to cavernous malfunction rather than to impaired flow in the cavernous arteries alone (*Saenz de Tejada, 1989*). However flow characteristics in the cavernous arteries, best measured with color-coded duplex sonography, did not indicate oxygenation itself (*Lopez et al, 1991)* and *(Meuleman et al, 1992)*.

Influence of oxygen tension on contractility of vascular smooth muscle as demonstrated in several studies (*Grote et al, 1988),* might have an important role in penile erection, since smooth muscle relaxation is regarded as the main event in erection. Nitric oxide has been shown to be the chief producer of relaxation of penile smooth muscle (*Knispel et al, 1992)* and *(Bloch et al 1998).*

Kelm et al (1991) found that,the half-life of nitric oxide depends mainly on the concentration of molecular oxygen, superoxide anions and hydroxyl radicals which are directly proportional to tissue oxygen tension. So, the importance of cavernous oxygen tension is evident from its influence in contractility of vascular smooth muscle.

Therefore, the reduction in cavernous oxygen tension can be considered to be a common mechanism in arterial and venous erectile dysfunction (*Romero-Tejada et al 1998).*

*Aim of the work:

The aim of this work is to evaluate vasculogenic impotence by monitoring the cavernous oxygen tension.