INTRODUCTION
When the hair becomes grey and scanty, when specks of earthly matter begin to be deposited in the tunics of the artery and when a white zone is formed at the margin of the cornea, at this same period the prostate gland usually – I might perhaps say invariably becomes increased in size’ (Sir Benjamin Brodie).

Benign prostatic hyperplasia (BPH), the most common benign tumour in men, is responsible for urinary symptoms in majority of men older than 50 years of age. BPH has been known for centuries to be a cause of urinary dysfunction. It was mentioned in the Egyptian papyri as early as 1500 BC and was discussed by Hippocrates 1000 years later. From time of birth until puberty, there is little change in the size of prostate. At puberty, a rapid increase in size occurs that continues up to third decade. Prostatic growth at this time increases at the rate of 1.6 g per year. There after its growth markedly decreases to 0.4 g per year in men of age 31 to 90 years (Berry et al, 1934). At age 55, approximately 25% of men note a decrease in the force of their urinary stream, at age 75, this increase linearly to 50% (Arrighi et al, 1990).

Etiology of BPH is unclear. Several hypotheses have been put forth based on histologic, hormonal and age related changes. Two factors, necessary for BPH to occur are:
1. Presence of dehydrotestosterone (DHT).
The importance of DHT in prostatic growth is dramatically manifested in patients with congenital deficiency of 5 alpha reductase, the enzyme responsible for conversion of testosterone to DHT. Affected males have ambiguous genitalia at birth, but at puberty the availability of normal levels of testosterone causes normal virilisation, functional erections, and ejaculation. The low level of serum DHT, however, result in a vestigial prostate, that never develops BPH.

The relative roles of androgens and estrogens in inducing BPH, however, are complex and not understood completely. It is known that castration prior to puberty prevents BPH, castration in patients with established BPH, however, does not uniformly result in prostatic atrophy. A probable explanation may be that androgens are required for initiation of BPH but not for its maintenance.

Anatomically, the prostate gland is of the shape of a compressed inverted cone. It is situated in the true pelvis below inferior border of the pubis symphysis and lies in front of the ampulla of the rectum. Its upper end is continuous with the neck of the bladder.

The prostate is a complex organ consisting of acinar, stromal, and muscular elements. It starts to develop at the 12th week of the fetal life under the influence of androgenic hormones from the fetal testes.

Cystometry at its inception more than a century ago was used only for experimental physiology followed by Human application much later in 1927. The rapid surge of interest with wider clinical
application gained momentum with the development of improved methods of measuring urinary flow rates by Vongarrelts in 1956.

Electromyographic assessment can be attributed to the efforts of Franksson and Petersen 1955 and measurement of Bladder and Urethral pressure to Enhorning 1961.

Cystometry is the recording of the pressure-volume relationship of the Bladder. The method yields important information concerning accommodation of the Bladder to increased filling volumes and sensory perceptions. The term cystometry usually indicates measurement of the Detrusor pressure during controlled Bladder filling and subsequent voiding with measurement of synchronous flow rates (filling and voiding cystometry).