FEMININE URINARY INCONTINENCE AND URODYNAMIC EXAMINATION

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SUMMARY

The Authors performed urodynamic examinations on all patients affected by urinary incontinence. This examination enabled them to classify incontinence precisely, from the etiopathogenetic point of view.

Incontinence may be due to an altered transmission of the abdominal pressure, to sphincteral lesions or insufficiency, or to stimuli.

This classification provides clear indications for the choice of the most suitable therapy (surgical, medical, instrumental). It thereby contributes to improving the outcome and decreasing the number of relapses so far reported by all Authors using any of the suggested surgical techniques.

The urodynamic examination has proved to be a valid method to detect the causes of feminine urinary incontinence – a syndrome related to various etiopathogenetic moments – overcoming all uncertainties entailed by the examination of the mere anamnestic and morphologic data.

A differential urodynamic diagnosis of the various forms of incontinence is therefore absolutely necessary to select patients accurately and choose the most suitable therapy.

However, the reliability of this kind of examination is highly dependant on the instruments and methods that are used. The adequate instruments allow (fig. 1) the simultaneous evaluation of many parameters, three pressures at least (abdominal, vesical and differential or detrusorial), the electric activity of the sphincter or the pelvic diaphragm, and the urinary flow. Fig. 2 shows the recorded values of all these parameters in a healthy person.

The examination is divided into different phases. In the beginning, simple expedients, like cough (C), are resorted to verify the perfect synchronism of the pressure and the sphincteral electric activity.

During the following phase (filling cystometrogramme) the detrusor pressure (DP) slightly and constantly grows with a maximum increase of 4 cm of water, up to a volume of 280 ml in the examined case. This is the maximum vesical functional capacity. In fact, immediately afterwards (fig. 3), as miction can no longer be inhibited, the detrusor contracts leading pressure up to 75 cm of water, and the tracing of the mictional flow appears on the record.

During this phase (mictional cistometrogramme) the relaxing of the urethral sphincter and floor, testified by the electric silence to EMG, shows the perfect synchronism of the detrusorial and sphincteral activities.

The last examination phase is the recording of the urethral pressure profile

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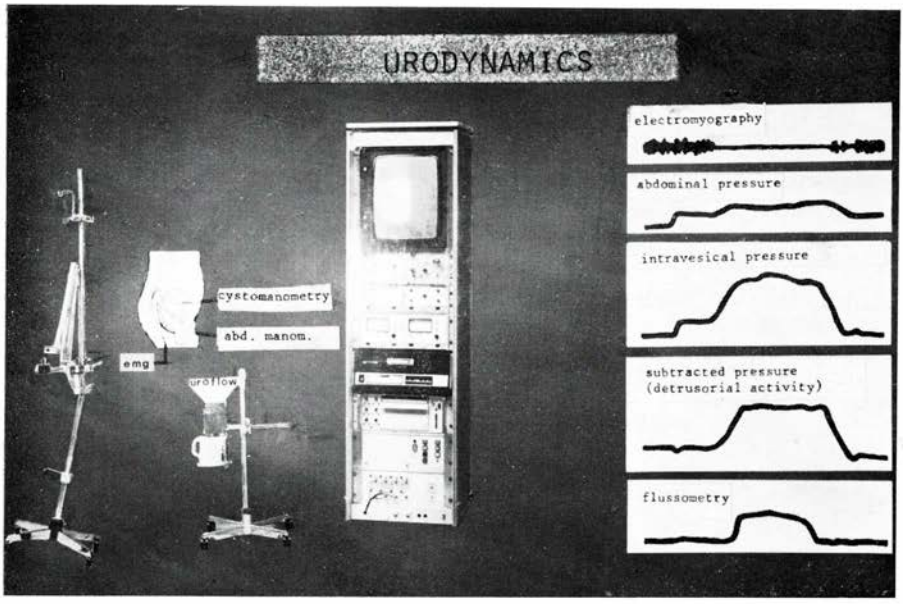
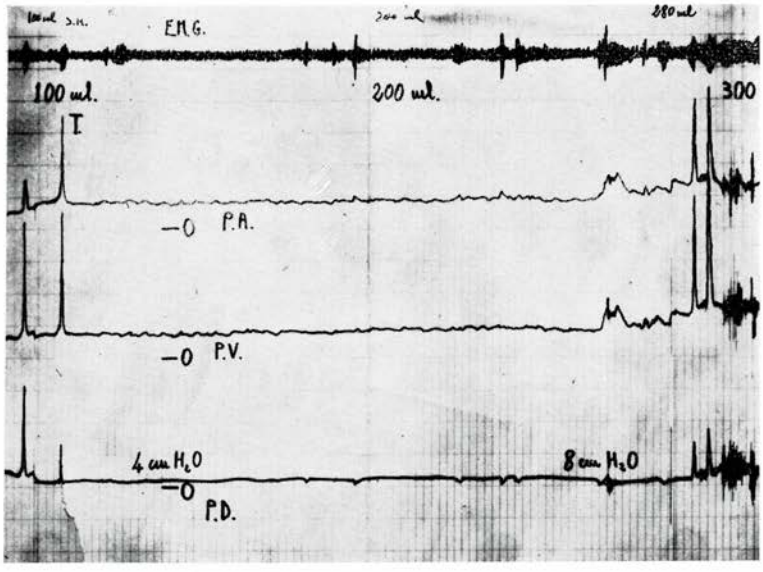
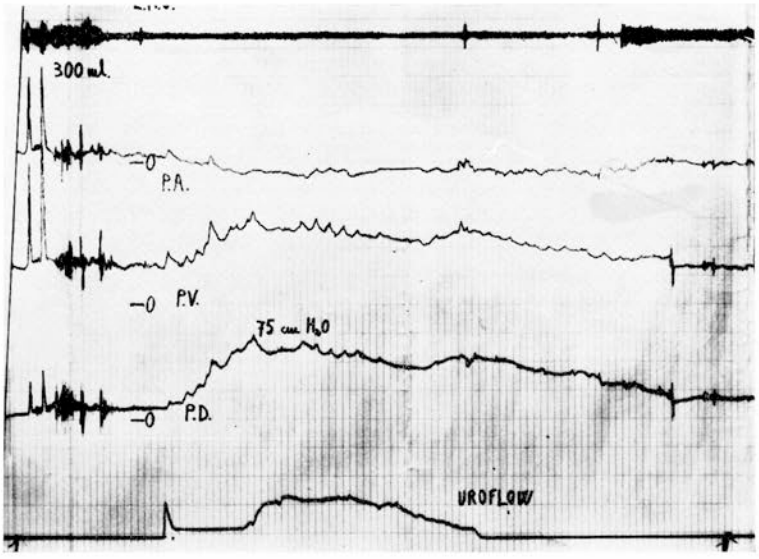


Fig. 1.



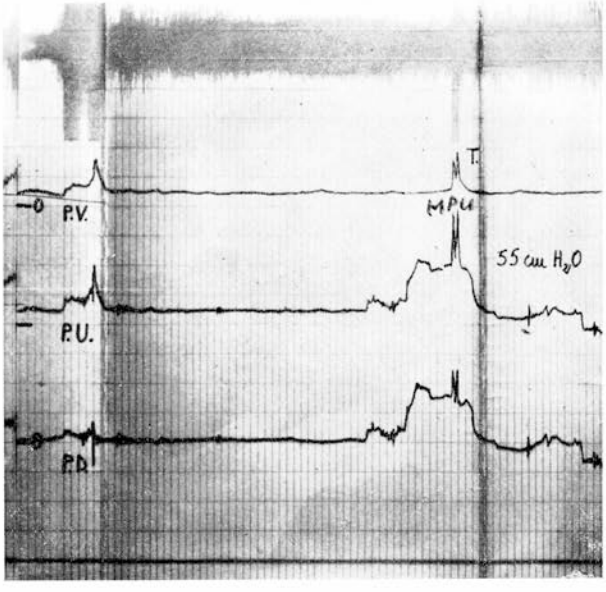
P.A., Abdominal pressure; P.V., Vesical pressure; P.D., Substracted pressure.

Fig. 2.



P.A., Abdominal pressure; P.V., Vesical pressure; P.D., Substracted pressure.

Fig. 3.



P.V., Vesical pressure; P.U., Urethral pressure; P.D., Substracted pressure; M.P.U., Maximal urethral pressure.

Fig. 4.

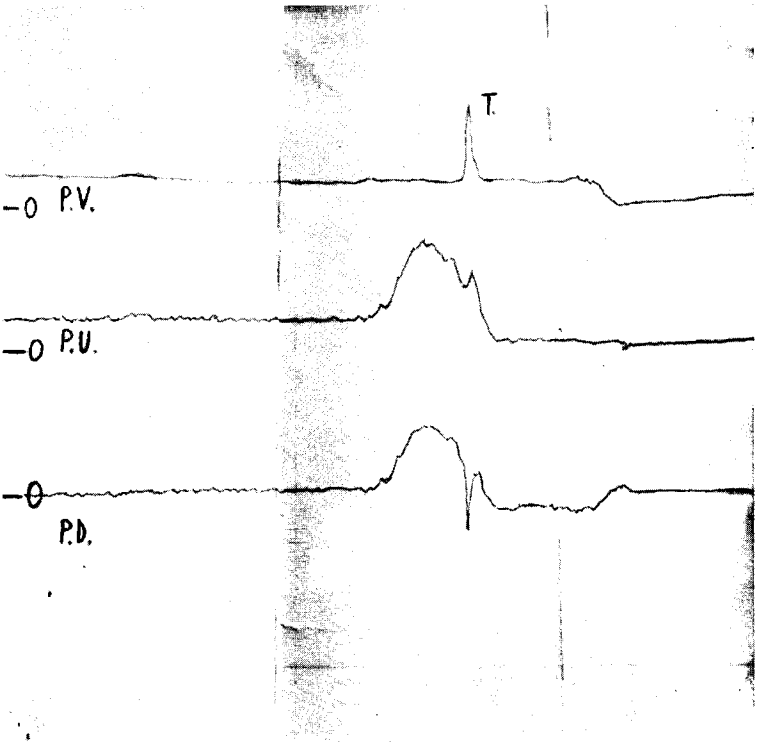
(UPP). Normally, a thin catheter is used, opened at one end to measure the vesical pressure, and with a few holes five centimetres from that end to measure the urethral pressure.

The tracing reported in fig. 4 was obtained by extracting the catheter at the same constant speed as that of the paper coming out of the polyrecorder, so that the record was just as long as the catheter.

The tracing shows: the vesical pressure (VP); the urethral (UP) and differential (DP) pressures.

A positive peak in the point of maximum urethral pressure (MUP) shows an even transmission of the abdominal pressure (cough) to the vesical and urethral areas with no change in the differential pressure.

This positively proves that the vesical neck and the urethra are in their right positions within the abdominal pressor girdle.



P.V., Vesical pressure; P.U., Urethral pressure; P.D., Substracted pressure; T., Cough.

Fig. 5.

The parameters measured through the urodynamic examination highlight three forms of urinary incontinence and their possible association.

The first originates from sphincteral insufficiency; the second from an altered transmission of the abdominal pressure, and the third from the detrusor hyperactivity.

Fig. 6.

The first two forms are referred to as 'stress incontinence'; the third as 'urge incontinence'.

Stress incontinence, caused by an altered transmission of the abdominal pressure, occurs when the urethra and the vesical neck change position thus becoming functionally extraabdominal organs. This may follow upon any lesion of the support structures (delivery trauma, tissue relaxation, hysterectomy). In these cases, in both the filling and the mictional cystometrogramme, the UPP tracing (fig. 5) shows a negative peak of the differential pressure.

The cause is an uneven transmission of the increasing abdominal pressure, favouring the vesica as against the urethra.

The clinical consequence is urinary leakage.

Usually, in these cases, colpocystography shows a change in the posterior urethrovesical angle.

Fig. 6 reports the colpocystogramme of the examined patients affected by cystocele stress incontinence.

The vesica has prolapsed downwards and towards the anterior vaginal wall; the normal vesical base-plate morphology has changed and, notably, the vesicourethral angle tends to become obtuse.

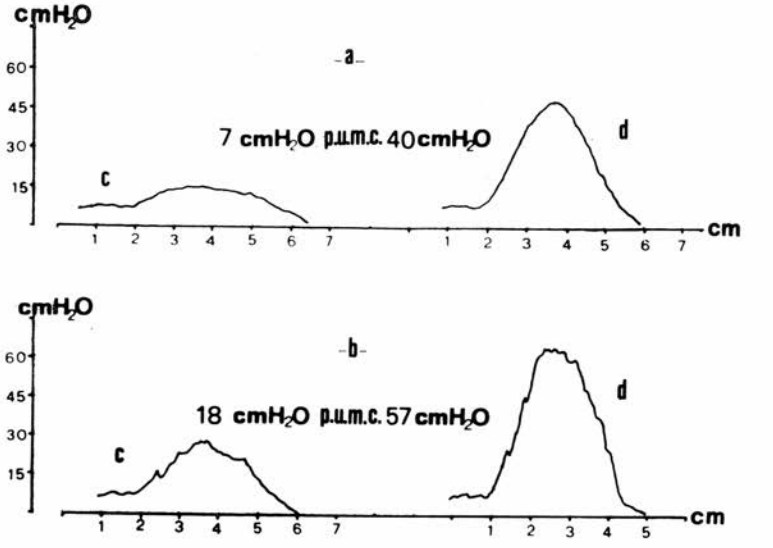
Post-operatory colpocystography (fig. 7) shows that the pelvic floor has resumed its normal anatomic position with respect to the vesica lifted 'in toto' and in its base, and to the posterior vesico-urethral acute angle. This is kept in its position by the stretch of the anterior vaginal wall, where the traction stitches are.

In stress incontinence due to mere sphincteral insufficiency, the UPP shows a decrease in the maximum closing urethral pressure and the urehtral functional length. In these cases the UPP is usually taken with a microtransuder catheter too.

Unlike the above described catheter, this measures the real pressure, not the urethral resistance. It is therefore particularly useful to study the pathology of the urethral sphincter.

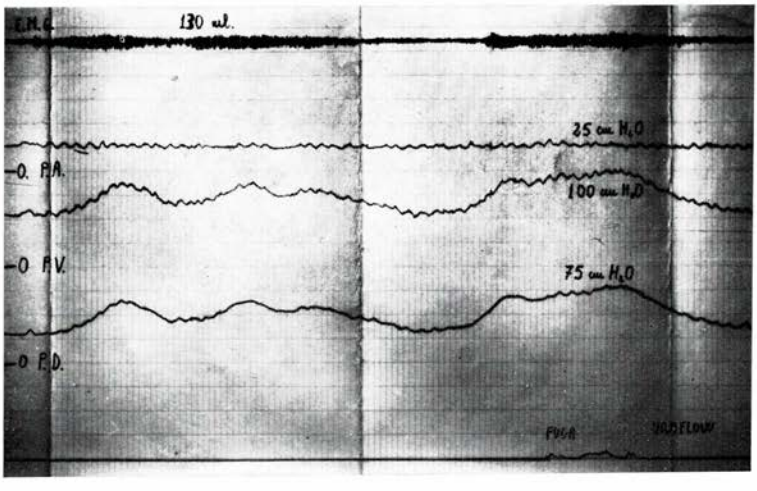
Fig. 8 shows the UPP (basal and during electrostimulation) of a patient affected by stress incontinence following upon a

Fig. 7.



a)= H_2O infusion b)=microtransductor
c)= basal pressure d)=during electrostimulation
pumc= maximal urethral closure pressure

Fig. 8.



P.A., Abdominal pressure; P.V., Vesical pressure; P.D., Substracted pressure; Fuga, Escape of urine.

Fig. 9.

surgical intervention to remove paraurethral cysts. An urethral sphincter lesion was suspected.

UMP values and the functional length are clearly reduced. Their rise during stimulation, though moderate, indicates that the sphincter can resume its functionality, through adequate electrostimulation.

Finally, urinary incontinence due to the detrusor hyperactivity (urge incontinence) from the urodynamic point of view, is characterized by contractions that the patient cannot inhibit. They raise the vesical pressure beyond the closing MUP, causing urinary leak.

From the physiopathologic point of view, many mechanisms can be held responsible for the contractions that the detrusor fail to inhibit. First of all, a deficient inhibition of the mictional response, caused by a wide range of neurologic diseases; then, an anomalous stimulation of the positive feed-back mechanism between the proximal urethra nervous terminations and the detrusor.

Under physiologic conditions, this mechanism contributes to holding the miction and can be activated under particular urethral pathologic conditions, like the presence of ectropion, stenosis or papillomas.

Lastly, another cause can be a lesion in the mechanisms inhibiting the mictional response.

Fig. 9 shows the urodynamic picture of a patient affected by urge incontinence due to ectropion of the urethral mucosa.

Unlike stress forms, this form presents an anomalous filling cystometrographic tracing with normal sphincteral functionality. In fact, when the vesical volume attains 150 ml, contractions occur that are not inhibited by the detrusor. They reach a maximum value of 75 cm of water, thus producing a moderate urinary leak, shown by the flow record.

The electric activity of the perineal plane muscles increases to offset the endovesical hypertension and prevent urinary leak.

CONCLUSION

Urinary incontinence forms must be classified according to etiopathogenetic categories to allow the performance of a precise surgical, medical or instrumental therapy. This is the only way to prevent distressing failures due to indiscriminate surgical treatment. Urodynamics can provide today accurate indications for alternative treatments.

Wherever this approach was followed there has been a remarkable improvement in the results obtained. This confirms that the high number of relapses was due to the unsuitability of the surgical intervention when alternative therapies were possible, rather than to the kind of surgical intervention that was performed (there is an extremely wide range of suggested surgical interventions, but none of them is successful in all cases).

However, the urodynamic examination must meet precise requirements.

We have described technical details at length not to repeat overmeticulously and uselessly things that should already be well-known, but, on purpose, to state principles that must absolutely be complied with if the urodynamic examination is to produce comparable, repeatable and, above all, reliable results.

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