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BRAIN IMAGING AND URODYNAMIC CORRELATION IN PATIENTS WITH CEREBROVASCULAR STROKE

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Objective: To identify urodynamic abnormalities in patients with cerebrovascular accidents and correlate both with CT or MRI findings.

Patients and Methods: From September 2001 to March 2003, a total of 44 males and 16 females were prospectively examined urodynamically in different phases after cerebrovascular accidents, and as early as two days after stroke.

Results: In most cases, the urodynamic findings could be correlated with CT or MRI findings. The most determining factor was the site of the lesion followed by the size. Small lesions were frequently silent unless located in critical sites. It was found that frontal, frontoparietal, parietal, basal ganglia and internal capsular ischemic lesions were associated in most cases with detrusor hyperreflexia, whereas thalamic, pontine and cerebellar infarcts were linked to detrusor hyporeflexia. Multiple lesions within the same group produced the same effect, while mixed lesions produced variable effects. There was no effect of laterality or dominance and an initial shock phase could not be identified. Detrusor-sphincter-dyssynergia (DSD) and hence upper tract deterioration were not observed. The effect of stroke was also modified by already present or predominant conditions such as BPH.

Conclusion: Correlating urodynamic and CT findings is very difficult in stroke patients because of the diffuse nature of the lesions, the unknown function of many brain centers in micturition control, the innumerable connections between the different brain regions and the extremely complicated influences that the brain regions exert upon each other and upon the bladder. The optimal understanding of the problem is dependent upon the better understanding of the function of each part of the brain. Further studies in this direction are recommended.

Keywords: cerebrovascular accident (CVA), urodynamics, brain imaging, incontinence.

INTRODUCTION

In spite of the marked reduction in mortality after cerebrovascular accidents (CVA), they remain the third leading cause of death after heart disease and cancer, in addition to the high incidence of temporary or permanent disability. Many patients suffer from lower urinary tract symptoms (LUTS) and urodynamic abnormalities[1]. Some authors describe detrusor hyperreflexia in the majority of symptomatic patients[2-4]. Others claim the presence of detrusor-sphincter-dysynergia (DSD) in some lesions[2,3], which is not to be expected in any suprapontine lesion[5]. The usefulness of CT or MRI in the urologic management of these patients is still unclear. Previous studies that tried to correlate the urodynamic results with CT findings failed to disclose a definite pattern of diagnostic or prognostic significance. The morbidity associated with surgery in this patient group and the difficulty in differentiating irritative voiding symptoms secondary to hyperreflexia from those due to outlet obstruction have raised the importance of studying this high-risk group of patients.

The aim of this work was to identify the related urodynamic abnormalities and to correlate them with CT or MRI findings regarding

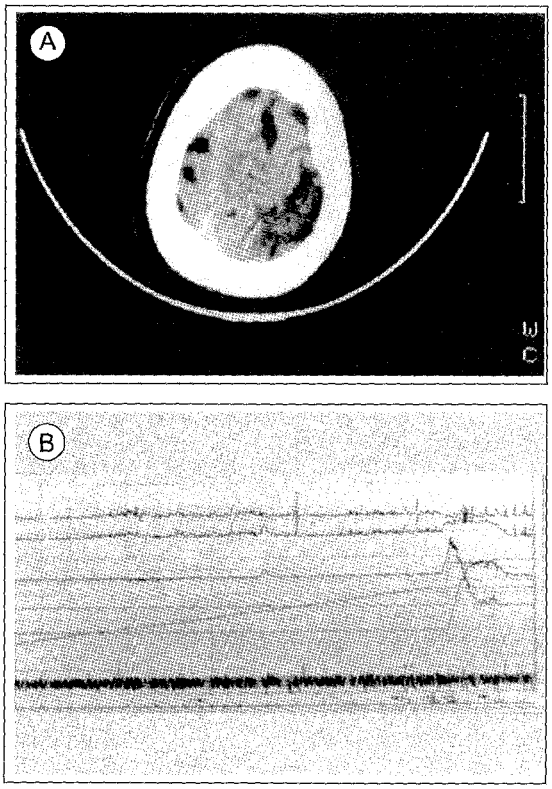

Fig. 1: A: CT scan of a patient with a frontal lesion affecting the sphincteric area. **B:** Cystometrogram of the same patient showing involuntary low amplitude detrusor contraction with concomitant sphincter relaxation accompanied by leakage.

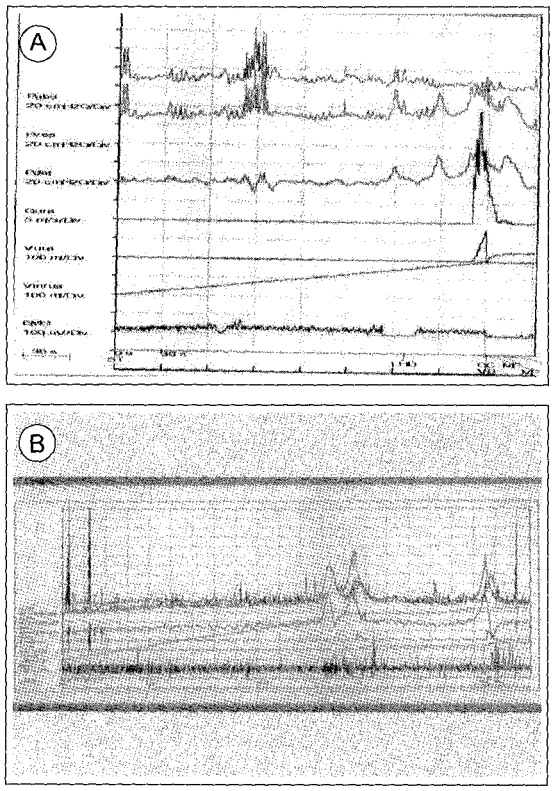

Fig. 2: A: Patient with uninhibited sphincter relaxation (USR), category 3. Note the hyperactive waves which are not accompanied by reflex sphincter contraction. **B:** The same patient has a lesion in the internal capsule.

Table 1: Patient Distribution According to Evaluation Date

Post-stroke evaluation date	No. of Pts	%
2 days	4	7%
3 days	5	8%
4 days	4	7%
5 days	3	5%
7 days	6	10%
Second week	17	28%
> 2 weeks	21	35%

laterality (and hence dominance), site and size of the lesion. We tried to detect the interaction between CVA and other underlying conditions such as BPH, diabetes or neuropathy and eventually to suggest a urodynamic-oriented treatment of resultant incontinence.

PATIENTS AND METHODS

The study which was carried out between September 2001 and March 2003 included 44 males and 16 females in different phases after CVA admitted to the hospital for treatment. Their age ranged from 35 to 85 years with a mean of 64.8 years.

Male or female patients with a stroke (ischemic or hemorrhagic) or transient ischemic attacks (TIA) with or without LUTS were included in the study, while patients with a disturbed level of consciousness who were unlikely to communicate and those in a general condition too bad to permit such a relatively invasive examination, e.g. those with coma or

Table 2: Results of Urodynamic Evaluation

	No. of Patients	%
Intact bladder sensation	all	100%
Unstable bladder contractions	19	31%
Asymptomatic detrusor hyperreflexia	2	3%
Post-void residual (PVR)	15	25%
Uninhibited sphincter relaxation (USR)	5	8%
Pseudodyssynergia	9	15%

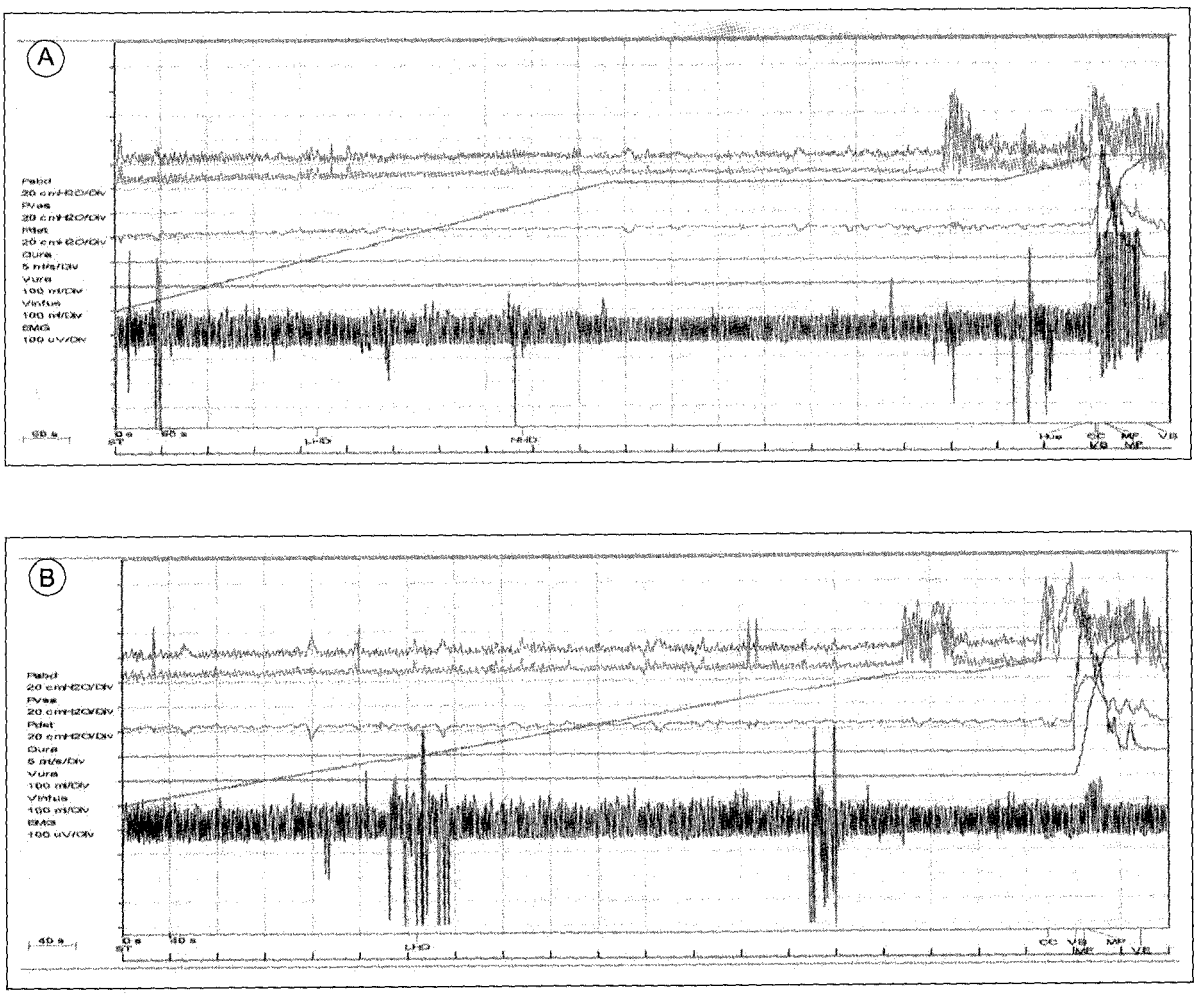

Fig. 3: A: An example of pseudo-dyssynergia. Note the very high sphincter activity during voiding simulating DSD with impaired flow. **B:** The same patient was instructed to relax. Note the marked reduction of EMG activity during voiding with a marked improvement of the flow.

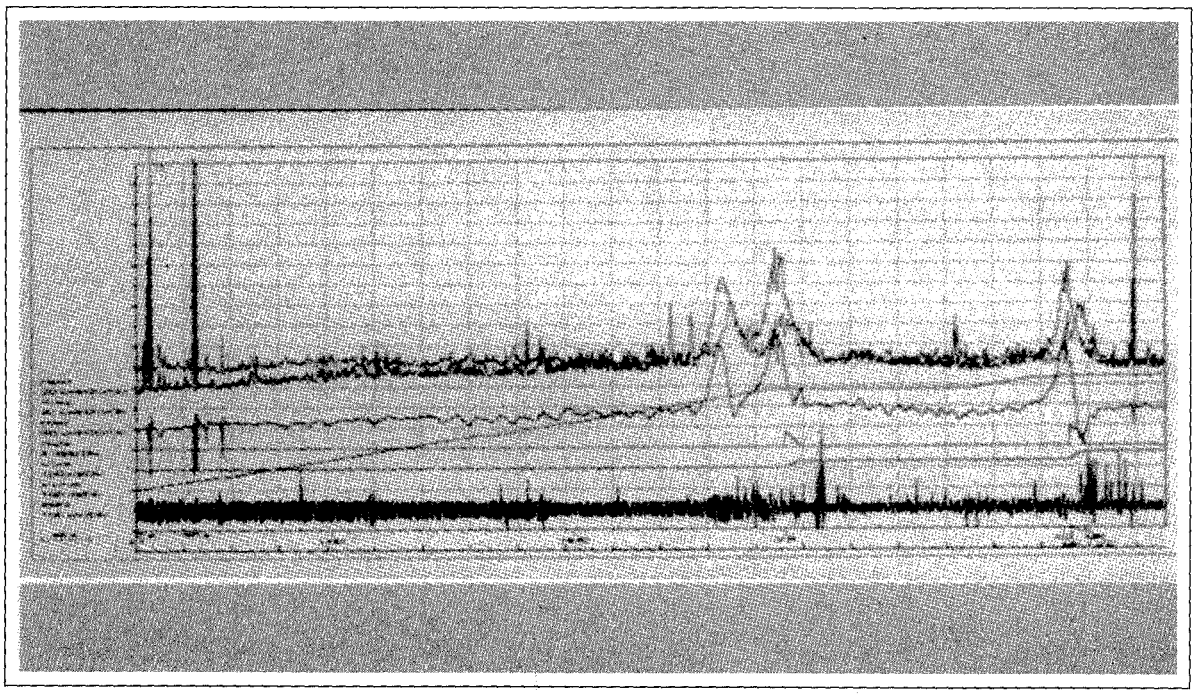

Fig. 4: Cystometrogram of a patient with hyperreflexia with reactionary sphincteric response

on artificial ventilation, were excluded. Aphasia was not a contraindication as long as the patient could understand and communicate.

A total of 22 patients were examined during the first week after the onset of CVA (four patients were examined as early as two days after the stroke), 17 cases during the second week and the remaining patients (n=21) more than two weeks after the stroke (Table 1).

All patients were subjected to a full neurologic assessment, cranial CT or MRI, voiding diary, ultrasound assessment of the bladder capacity and the condition of the upper urinary tract, and eventually a full fluoroscopy-guided urodynamic evaluation.

RESULTS

The results of urodynamic evaluation (Table 2) revealed that bladder sensation was preserved in all patients.

The bladder volume varied widely from 100 to 630 cc. In patients with a hyperreflexic bladder, it was much reduced (100-220 cc), while in those with hyporeflexia it ranged between 480 and 630 cc.

Unstable bladder contractions were observed in 19 patients (31%) and asymptomatic detrusor hyperreflexia in two (3%). The magnitude of involuntary contractions did not correlate with incontinence. In one of our patients, who also had uninhibited sphincter relaxation, leakage occurred after a weak involuntary contraction with a magnitude of 10 cm water (Fig. 1 A,B)

A post-void residual (PVR) was observed in 15 patients (25%); in four of them it was attributed to bladder hypoactivity, in two due to preexisting LMNL and in nine due to BPH.

Uninhibited sphincter relaxation (USR) or absent volitional control of the sphincter (AVCS) was observed in five patients with

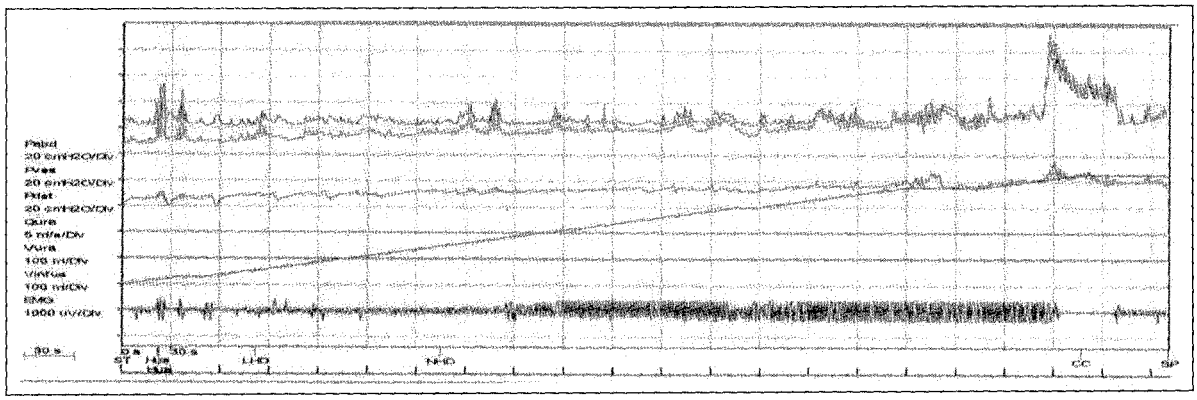

Fig. 5: Cystometrogram of a patient with hypoactive bladder (category 4). Note the complete absence of detrusor contraction and the electric silence of the sphincter during voiding.

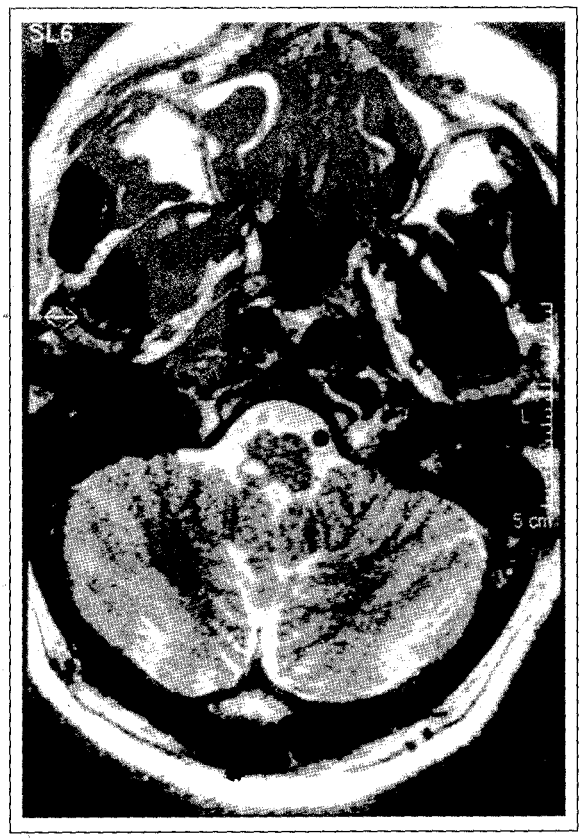

Fig. 6: Significance of the site and site of the lesion. This MRI shows a small peripheral medullary lesion in a urodynamically normal patient.

frontal lobe lesions or internal capsular lesions (Fig. 2 A, B).

Pseudodyssynergia was observed in nine patients (15%). In contrast to true DSD pseudodyssyneriga was characterized by its changing pattern on repeated examination and by its reduction or even disappearance once the patient was instructed to relax (Fig 3 A,B). True DSD was not seen in our series.

CT and MRI findings and their correlation to urodynamic studies:

In 13 patients, no lesion could be detected by CT or MRI; the remaining 47 patients were distributed as shown in Table 3.

The correlation of urodynamic results and imaging studies enabled us to combine affected brain regions and typical bladder dysfunction patterns. Thus, a division into the following groups was possible:

1. Group I: These lesions were characterized by detrusor hyperreflexia; they included lesions of the frontal lobe, the frontoparietal area, the parietal lobe, the internal capsule and the basal ganglia. Lesions of more than 2 x 1 cm in size caused detrusor hyperreflexia in five out of six patients. In the remaining six patients the lesion was very small (0.5 cm or less) (Fig. 4).

2. Group II: These lesions are linked to detrusor hyporeflexia. They include lesions of the thalamus, pons, cerebellum and the two hemorrhagic strokes. (Fig. 5). Reasonably big lesions caused hyporeflexia (5 of 8), the other three were small or lacunar. Cerebellar infarcts caused hypoactivity in one case with a very big cerebellar infarct, while three cases with small infarcts were normal.

Table 3: CT and MRI Distribution of Brain Lesions in 47 Patients

Site	No of Patients
Frontal lobe	5
Frontoparietal area	1
Parietal lobe	1
Parietotemporal	2
Occipital lobe	2
Internal capsule	3
Basal ganglia	2
Internal capsule and basal ganglia	8
Frontal lobe with internal capsule or basal ganglia	3
Cerebellum	4
Thalamus	2
Pons	3
Thalamus with internal capsule and/or basal ganglia	4
Pons with internal capsule or basal ganglia	3
Medulla oblongata	2
Hemorrhagic strokes: 2, one of them thalamic and the other pontine	2

3. Combined lesions within the same group produced the same effect. Multiple lesions within Group 1 also produced detrusor hyperactivity (10 of 11), although some of them were lacunar

4. Mixed lesions between Groups I and II produced variable effects, where the resultant effect may be hyperactivity, hypoactivity or normal. This group was composed of seven patients, of whom five were normal or showing features not attributable to stroke.

5. Parietotemporal lesions were without effect on bladder function (2 of 2).

6. Occipital lobe lesions were also without effect (2 of 2).

7. Medulla oblongata lesions were without effect (2 patients) (Fig. 6).

8. Dominance: In patients with hemispheric strokes, affection of the dominant (left) hemisphere was observed in 16 cases; six of them had symptoms attributable to stroke and 10 had LUTS prior to stroke.

DISCUSSION

Although many higher centers have been implicated in micturition, the mechanism by which these centers exert control is one of the seriously debated topics in neurourology. Accordingly, the effect of lesions of each of these regions is still vague. Most of the previous studies have been done retrospectively and included only stable post-stroke patients.[2,4]

Our patients were examined in different phases after stroke and as early as the general and neurological condition permitted. The patients were classified into five urodynamic groups according to the behavior of both bladder and sphincter during urodynamic evaluation. Of particular interest was the uninhibited sphincter relaxation (USR). This behavior of the sphincter is characterized by sphincteric relaxation during uninhibited bladder contraction, while a normal sphincter shows reflex contraction.

Bradley and Scott described that the sensory pathways from the periurethral striated sphincter terminate in the sensory motor cortex and that the descending motor axons from this

region travel through the internal capsule[6]. Therefore it can be concluded that lesions of the frontal lobe or internal capsule result in USR. Arena et al. mentioned that patients with this abnormal behavior could suffer from a kind of psychological carelessness of their continence mechanism[7]. We have observed that patients with USR preserve the sphincteric response to coughing or increased abdominal pressure.

Khan et al. and Burney et al. claimed that they had encountered DSD in 10% and 8.3% respectively of patients with large, diffuse or bilateral lesions[4,8], while Sakakibara reported an incidence of 14%[9]. Other authors stressed that DSD did not occur from CVA and was uncommonly, if ever, seen in suprapontine lesions and that guarding or pseudodyssynergia during involuntary bladder contraction was common and should not be misinterpreted as DSD, where EMG signals increase immediately before a measurable bladder pressure rise[5,10]. The timing of EMG activity helps to differentiate true DSD from pseudodyssynergia[10]. We did not encounter DSD in our series.

Giannantoni et al. reported to have encountered pseudodyssynergia during voiding in all subtypes of strokes[11]. In our series, we also found that pseudodyssynergia did not follow a specific correlation with the site or severity of the stroke. It was differentiated from true DSD by its changing pattern on repeated examination and by reduction of the amplitude of activity or even disappearance once the patient was instructed to relax.

BPH was diagnosed by fluoroscopic observation of the bladder neck and urethral opening during pressure flow studies and by plotting maximal flow (Q_{max}) and detrusor pressure at maximal flow (Pdet Q_{max}) on the Schaefer nomogram.

As for the CT correlation, in Group I (hyperreflexic group) the lesions included the frontal lobe, frontoparietal lobe, internal capsule, and basal ganglia. Feder et al. reported an association between infarct size and urinary incontinence and that the incontinence was likely to occur when the lesion is bigger than 4 cm in diameter[12]. Gelber et al. reported similar findings[13]. We observed that in relatively large areas as the frontal lobe, lesions less than 2x1 cm are unlikely to produce effects, but lesions situated in a small area, such as the anterior limb or genu of the internal capsule, will produce marked effects, even if they are small. The superomedial part of the frontal lobe and genu of the corpus callosum are the regions responsible for bladder innervation[14,15]. Damage of this area causes detrusor hyperreflexia[16].

The cortical area concerned with the innervation of the periurethral striated musculature is located on the medial aspect of the sensorimotor cortex[14,15]. Fibers from this area travel caudally through the internal capsule and cerebral peduncles to the brain stem, continuing ultimately as corticospinal tracts[17]. Lesions of this area result in USR[17].

The first group included 12 patients, in 4 of them the lesion was very small (0.5 cm in diameter), two had lesions located in the frontal lobe away from the important detrusor and sphincteric areas. The remaining six had reasonably bigger lesions and five of them had detrusor hyperreflexia. The sixth one had a hypoactive bladder. Neurologically, this patient had a big infarct with relatively impaired cognitive function as well as high-grade hemiplegia, and his motor condition dictated examination in the lying position. Hypoactivity could have resulted from the above mentioned combinations of lack of concentration, severe paralysis and bad examination condition in the lying position which did not allow appropriate voiding.

Group 2 included 11 patients. In the small regions, such as the thalamus and pons, only the three patients with peripheral lacunar lesions did not show symptoms, while the bigger lesions resulted in detrusor hyporeflexia. Cerebellar infarcts caused detrusor hyporeflexia in one case with a big infarct, 4 cm in diameter, while the three remaining cases had smaller infarcts associated with normoreflexia. Our results agree with the results of Burney et al. that cerebellar lesions are associated with detrusor hyporeflexia[8]. We observed that, unlike internal capsular and basal ganglia lesions, only considerably big cerebellar infarcts cause symptoms.

We had two patients with hemorrhagic strokes. The site of hemorrhage was the thalamus and brain stem, areas that are usually linked to hyporeflexia. It is not clear to us whether all hemorrhagic strokes cause detrusor hyporeflexia irrespective of size[8], or they produce variable effects according to the site of hemorrhage.

We saw combined lesions, i.e. lesions involving two or more regions in the same group, in 11 patients of the first group; in 10 of them the result was detrusor hyperreflexia, although many of the lesions were lacunar. The effect seemed to be an additive one.

The analysis of mixed lesions, i.e. lesions involving multiple regions from Groups 1 and 2, is most confusing as there may be a lesion affecting multiple areas of the brain without having an effect on micturition, whereas a lesion of each area alone will cause voiding disorders. This group included seven patients; five of them were normal or had other features not attributable to stroke, the other two had detrusor hyperreflexia. From our data we conclude that the effect of these lesions was an additive one and that the net result will be normal or one lesion will determine the end result.

The medulla oblongata is the site of decussation of the pyramidal tracts, so a lesion in this area (infrapontine) is liable to result in DSD. We had two patients with medullary lesions but the lesion in both cases was of the lacunar type, 0.5 X 0.5 cm, and located dorsolaterally distant from the area of pyramidal decussation. These medullary lesions could theoretically produce DSD if they were more centrally located and slightly bigger.

In conclusion, urodynamic abnormalities can in most cases be correlated with CT or MRI findings. The most relevant factor is the site of the lesion followed by the size. The larger the size of the lesion, the higher is the tendency to cause urodynamic abnormalities. Small lesions are frequently asymptomatic unless located in a critical site. The principle of fixed dominance as that of language is doubted. There is no identifiable shock stage, and the acute retention which may occur is probably not due to the effect of stroke but due to impaired consciousness, restricted mobility or inability to communicate the desire to void. Hence, temporary detrusor failure due to acute overdistension ensues. DSD, and hence upper tract deterioration, does not occur with strokes and if DSD is encountered, one should look for an infrapontine lesion. The net result of CVA is affected by already-present or subsequently evolving diseases as BPH.

Correlating urodynamic, and CT findings is very difficult in stroke patients because of the diffuse nature of the lesions, the unknown function of many brain centers in micturition control, the innumerable connections between the different brain regions and the extremely complicated influences that the brain regions exert upon each other and upon the bladder which are not yet well-understood; this is in addition to the associated age-related changes and BPH adding to the complexity of the analysis.

The optimal understanding of the problem is dependent upon the better understanding of the function of every part of the brain. We recommend further studies in this direction.

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RESUME

Corrélations entre l'imagerie cérébrale et l'urodynamique chez les patients présentant un accident vasculaire cérébral

Objectifs: Identifier les anomalies urodynamiques chez les patients présentant un accident vasculaire cérébral (AVC) et leur corrélation avec les résultats de la TDM et de l'IRM. **Patients et Méthodes**: De septembre 2001 à mars 2003, une étude prospective de 44 hommes et 16 femmes a été réalisée. Une étude urodynamique pendant les différentes phases après accident vasculaire cérébral a été indiquée chez tous les patients et ce dès les deux premiers jours de la phase aigue. **Résultats:** Dans la plupart des cas, les conclusions de l'urodynamique pourraient être corrélées aux résultats de la TDM et de l'IRM. Le facteur déterminant était la topographie de la lésion et sa dimension. Les petites lésions étaient fréquemment silencieuses à moins qu'elle soit de topographie critique. Les lésions des lobes frontal, frontopariétal, pariétal, du ganglion basal et les lésions ischémiques capsulaires internes ont été associées dans la plupart des cas à une hyperreflexie du detrusor, alors que les infarctus thalamiques, pontiques et cérébelleux ont été liés à une hyporeflexie du detrusor. Les multiples lésions dans le même groupe ont produit le même effet, tandis que les lésions associées ont produit des effets variables. Il n'y avait aucun effet de latéralité ou de dominance et une phase de choc initiale ne pourrait être identifiée. La dyssynergie vésico-sphincterienne (DSD) et de là la détérioration du haut appareil n'a pas été observée. L'effet de l'AVC a aussi été modifié par les conditions déjà présentes ou prédominantes tel que la HBP. **Conclusions:** Corréler les résultats de l'urodynamique aux conclusions de la TDM est très difficile chez les patients présentant un AVC à cause de la nature diffuse des lésions, le rôle encore inconnue de beaucoup de centres cérébraux dans le contrôle de la miction, les rapports innombrables entre les différentes zones du cerveau et les influences extrêmement compliquées que les zones du cerveau exercent sur la vessie. La compréhension optimale du problème est dépendante d'une meilleure approche de la fonction de chaque partie du cerveau. Les études supplémentaires dans cette direction sont recommandées.

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