A DESCRIPTIVE STUDY ON URODYNAMIC ABNORMALITIES IN STROKE PATIENTS AFTER ACUTE PHASE

CERTIFICATE

This is to certify that **Dr. AUSTIN P C**, has done his thesis on **"A DESCRIPTIVE STUDY ON URODYNAMIC ABNORMALITIES IN STROKE PATIENTS AFTER ACUTE PHASE".** This study was undertaken at the **Christian Medical College & Hospital, Vellore – 632 004**, during **March 1, 1999 to Feb. 28, 2001** under my direct guidance and supervision.

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" Oh Lord you have been good
You have been faithful to all generations"

A task such as this would have been undoubtedly impossible without blessings of the Lord almighty, and collective inspiration and effort of many persons, whose names need to be mentioned, and gratitude expressed.

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This manuscript is dedicated to all the patients with hemiplegia, and their caregivers, who have reposed their faith in me.

Dr.P.C. Austin.

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Dissertation submitted during **March 1, 1999 to Feb. 28, 2001**

By

Dr. Austin P C

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INTRODUCTION

Cerebrovascular accident (CVA) is a serious neurologic event and, depending on the extent, a CVA can have temporary or permanent effects on survivors including deficits in speech, vision, memory, cognition, motor and sensory function, and volitional control of voiding.

Stroke accounts for more than 40% of acute admissions in a neurological centre. Following a stroke, 10% of the survivors, recover completely, 80% to varying levels and 10% need institutional care due to severe disability.[1] For the middle 80%, stroke rehabilitation by a rehabilitation team can significantly improve functional ability. Poor prognostic indicators of outcome are, previous stroke, older age, bladder and bowel incontinence and visuospatial deficits.[5]

Voiding dysfunction occur in a substantial percentage of stroke patients, but has not received sufficient emphasis because it improves gradually to near normal in the majority of patients. After a CVA, volitional control of voiding often is impaired. Voiding dysfunction as the result of a CVA, when compounded with other possible pre-existing conditions in the elderly can result in a challenging diagnostic and therapeutic dilemma.[1] Treatment of the bladder dysfunction should be based on the results of urodynamic findings. Subsequential urodynamic evaluations may be performed at regularly scheduled intervals based on changing symptoms or to assess success or failure of management. Proper diagnosis of the cause and

appropriate management of new onset voiding dysfunction in the poststroke patient before the patient leaves the acute care facility is desirable because urinary dysfunction can complicate patient rehabilitation.[2]

A functionally oriented approach to acute stroke care should take place in conjunction with traditional medical management, because the medical care provided during the first days and weeks after a stroke affects the ultimate disability status of the patients. Therefore, proper diagnosis and management of voiding dysfunction in the post stroke patient is important for improved patient well-being, increased patient survival, and decreased disability as well as a reduction in the health care expenditures.[1]

The symptoms indicative of voiding dysfunction are, frequency, urgency incontinence.[3] The same symptoms may be present for different voiding dysfunctions. To elucidate / differentiate the etiology of these symptoms urodynamics testing is helpful. Urodynamic findings should supplement and complement clinical examination findings. Over active bladder, is the common type of lower urinary tract dysfunction associated with cerebrovascular accidents and usually presents with irritative voiding symtoms such as frequency, urgency, and incontinence. Over active bladder is referred to as unstable when aetiology is non-neurogenic, and as hyperreflexic when the aetiology is clearly neurogenic.[2]

Urodynamics is a neuro-urologic diagnostic tool, used for evaluating the storage and evacuating function of the bladder.

There are four basic modalities for urodynamics testing. They are:

1. Cystometry (plain cystometry and voiding cystometry)
2. Uroflowmetry
3. Urethral pressure profilometry
4. Combinations with videofluoroscopy, and sphincter EMG study.

This study uses filling and voiding cystometry, to evaluate presence of voiding dysfunction in post CVA patients beyond the acute phase. Voiding cystometry is a method by which changes in bladder pressure are measured with progressively increasing bladder volumes followed by evaluation of the bladder pressure during the voiding phase. Cystometry can be done using either carbon-di-oxide gas or water.

20 patients who had MCA stroke 3 weeks to 12 weeks ago, participated in this study. Cystometric studies were done in the 20 patients with a view to describe the urodynamic abnormalities seen following stroke.

AIMS OF THE STUDY

1. To determine the incidence of voiding abnormalities due to bladder dysfunction in MCA stroke patients, subsequent to the acute sickness phase.

2. To describe the urodynamic abnormalities by using cystometry, in the above patients

ANATOMY AND PHYSIOLOGY OF STROKE

Stroke is a common life-threatening neurological disease and third leading cause of death, and leading cause of disability in adults. There are wide variations in death rates due to stroke from country to country. Survival following stroke has also improved in recent years from 49% to 62 %.[3]

Stroke is conventionally described as, an abrupt onset of non nonvulsive and focal neurological deficit, which is not progressive, due to cerebral ischemia or infarction. [17old]

The incidence of stroke increases with age and affects many people in the productive age group. It may be caused by either ischaemic infarction or intracerebral haemorrhage. Ischaemic infarction constitutes 85-90% of the total group and 10-15% are due to intracerebral haemorrhage.[5-30] The neurological deficit reflects both the location and size of the infarct or haemorrhage. Dominant hemispheric lesions cause aphasic disturbances. Most CVAs present as abrupt onset of focal neurological deficit. The deficit may remain permanent or may regress or worsen progressively. CVAs depending on its extent can have deficits in speech, vision, memory, cognition, motor and sensory function and volitional control of voiding.6 Voiding dysfunction in the acute phase of CVA is reported. Urinary retention occurs after initial stroke episode, and resolves on its own.

Blood supply of brain:

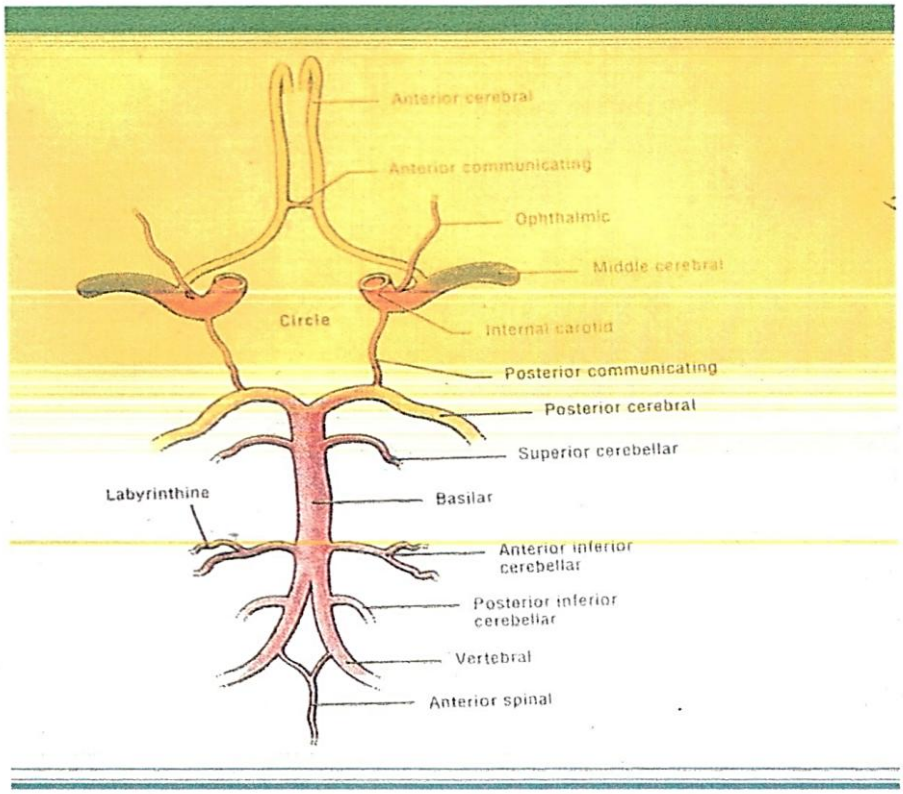
Four main arteries supply blood to the brain, they are 2 internal carotids and 2 vertebral arteries. Both vertebral arteries unite to form the basilar artery.

At the base of brain, the two internal carotids and basilar artery are interconnected to form the circle of Willis; which acts as a collateral circulation to some extent.

Each internal carotid artery gives off two terminal branches viz. the smaller anterior cerebral artery and the larger middle cerebral artery.

The territorial distribution of the middle cerebral artery includes sensorimotor areas around the central sulcus auditory areas and (in the dominant hemispehere) the speech areas.

Normal cerebral blood flow is 50-60 ml/100 gm of brain tissue / minute, Generalised decrease of cerebral blood supply is usually due to hypotension (due to any cause) and produces syncope and infarction starting from the water shed areas. Focal reduction of blood flow to any region is usually due to embolism or thrombosis secondary to cerebral vessel disease. This produces focal infarction of brain tissue, depending on the supply area of the vessel occluded.



Circle of Willis

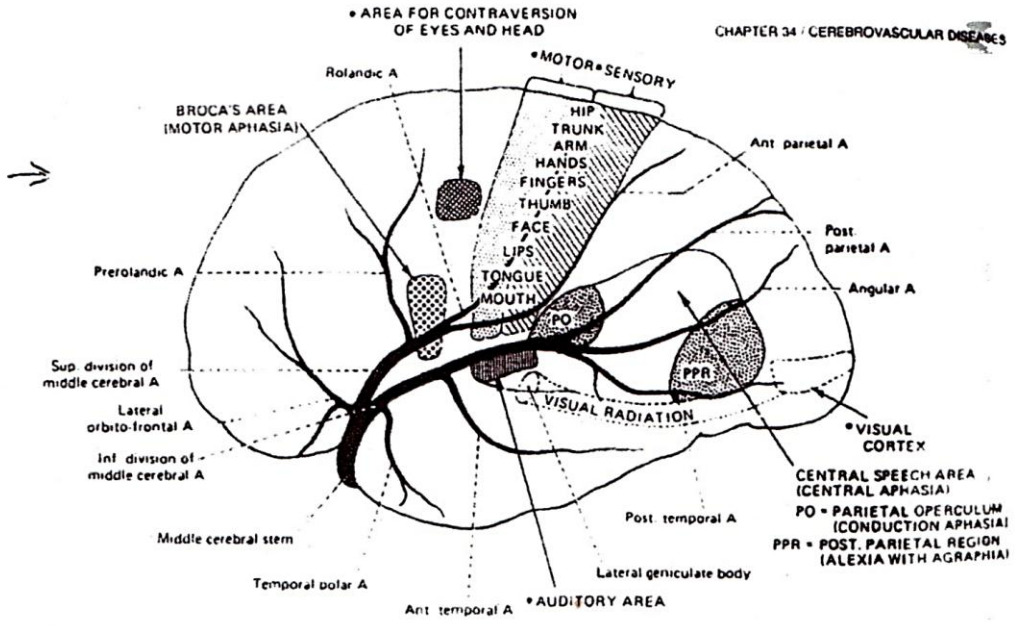


Figure 34-3

MCA Territory

Risk factors for stroke are:[6]

i. Hypertension

ii. High blood lipids

iii. Diabetes Mellitus

iv. Obesity

v. Heart Disease

vi. Environmental risk factors: Including cigarette smoking, oral contraceptives, alcohol consumption and less physical activity,

vii. Increased homocystine level.

Pathology:

Cerebral thrombosis is usually associated with softening of the brain mass. By far the commonest cause is arteriosclerosis with thromboembolism ; other causes include vasculitis, meningitis, TAO, periarteritis nodosa, encephalitis and mechanical obstructions. Atherosclerosis in the intracranial arteries usually involves the terminal portions of internal carotid, basilar, middle cerebral, anterior, posterior and pericallosal arteries, these vessels are at increased risk of sustaining an occlusive or haemorrhagic process.

If the blood perfusion abnormality persists for a few minutes, neuronal injury results, this is followed by formation of cerebral oedema. As swelling occurs, the capillaries lumen get progressively compressed. Ischemic oedema reaches its maximum 2-4 days after infarction and subsides by the end of second week.

Inflammatory changes and phagocytosis occur in the area of tissue necrosis, during the weeks that follow the brain tissues affected, show a core of severe ischemia (umbra), surrounded by a penumbra where ischemia is less severe, and neuronal cells have a chance of remaining viable.

Major categories of stroke are:

1. Hypoxic, ischaemia and infarction
2. Intracranial haemorrhage
3. Hypertensive vascular disease.
4. Thrombo embolic causes

Cerebral infarction

1. Thrombotic occlusions occur commonly at the carotid bifurcation
2. Embolic occlusion -most frequently occur in middle cerebral artery.
3. Haemorrhagic infarcts - results from breakup of emboli, allowing reflow of blood in damaged vessels.

Intracranial Haemorrhage: Types,

1. Intracerebral
2. Sub-arachnoid
3. Mixed

Intracerebral Haemorrhage:

Occurs in hypertensives, blood dyscrasias, trauma and cavernous angioma. Major sites are Putamen 55%; lobar white matter 15%; thalamus 10%; Pons 10% and Cerebellar haemorrhage 10%;

Subarachnoid Haemorrhage: Types,

1. Developmental – Berry aneurysm 95%
2. Arteriosclerotic
3. Mycotic

Causes of thrombosis, include

1. Atherosclerosis
2. Vasculitis
3. Arterial dissections
4. Haematologic disorders.

Causes of embolism, are

1. Cardiogenic causes
2. Atherothrombotic sources from diseased arteries
3. Of unknown origin.

Diagnostic studies:

1. Brain imaging

   Computed tomography

   Or

   Magnetic resonance imaging.

2. Doppler ultrasound scan of carotid arteries - To assess patency of vessels.
3. Echocardiography – To rule out cardiac source of embolism.
4. Intra-arterial digital subtraction angiography – for more precise evaluation of extracranial and intracranial vessels.

ANATOMY AND PHYSIOLOGY OF VOIDING

Urinary bladder: The wall consists of three layers.

1. Outer adventitium of connective tissue
2. Smooth muscle layer
3. Inner mucous membrane.

The trigonal area is described as the region of the posterior bladder wall between the ureteral orifices and the vesicourethral junction. The deep trigone is continuous with an integral part of the detrusor smooth muscle. Detrusor can be described as a mesh of smooth muscle, having outer longitudinal, middle circular and inner longitudinal layers.

Urethra: Female urethra is approximately 4cm in length. Extends from vesicourethral junction behind the symphysis pubis and embedded in the anterior wall of vagina.

Male: Preprostatic urethra – 1-1.5cm in length and contains smooth muscle bundles becoming continuous distally with prostatic capsule. The muscle bundles are separated by much connective tissue.

Prostatic urethra : 3-4cm in length; embedded within the substance of prostate. The urethral musculature in this part consists of inner longitudinal and outer circular layers.

Membranous portion of male urethra extends for a distance of 2.5cm distal to prostate to bulb of penis. There is continuity between inner longitudinal layer of bladder smooth muscle directly into the urethra.

External sphincter : It consists of striated muscle within the urogenital diaphragm which on contraction is capable of voluntarily stopping the urinary stream. It is annular in male, and posteriorly blends with the fibres of prostatic capsule. In female the muscle is described as tapering and being deficient posteriorly.

Internal sphincter: Located in the bladder base consists of bladder neck and proximal urethra. This sphincter consists of smooth muscle layer of urethra. Both outer circular and inner longitudinar layer of urethra are described as continuous with bladder smooth muscle layer. There is no anatomic sphincter at the bladder neck but there is a physiologic internal sphincter that consists

of bladder neck and proximal urethra, rich in elastic fibres. This smooth sphincter tone rises during normal urine storage, so that the urethral pressure exceeds intravesical pressure.

Striated sphincter / External sphincter comprises both intramural and extrinsic striated component surround the urethra in female and posterior urethra in male. Striated sphincter is not necessary for urinary continence.

PHYSIOLOGY OF VOIDING

Voiding is mediated by i) inhibition of somatic sympathetic pathways ii) activation of a spinal bulbo spinal parasympathetic reflex, passing through the pontine micturition center.

This causes inhibition of external sphincter activity, inhibition of sympathetic outflow, and activation of parasympathetic outflow.

Micturition is fundamentally a spinal reflex, facilitated or inhibited by higher centres; subject to voluntary facilitation and inhibition. Urine enters the bladder without producing much increase in intravesical pressure until the viscus is well filled. The bladder muscle has this property of plasticity.

The pressure in the bladder does not rise as it fills, in accordance with Laplace's law. This law states that the pressure in a spherical viscus is equal to twice the wall tension divided by the radius.[8] In the case of the bladder, wall tensions increases with filling, but so does the radius. Hence the net increase in pressure is slight until the organ is full.[9] (See appendix figure --- demonstrating normal cystometrogram)

During micturition, the perineal muscles and external urethral sphicter relax, and the detrusor muscle contracts; causing urine to pass out through the urethra. Relaxation of perineal muscles causes a downward tug on the detrusor muscle, to initiate its contraction.

Urgency is defined as, strong desire to void. Urge incontinence is defined as involuntary loss of urine associated with urgency, due to an overactive detrusor.

Frequency is defined as micturition more than 5 times in day, and once in night.[10]

Urinary incontinence has been defined by the International Continence Society, as involuntary loss of urine which is objectively demonstrable and is a social or hygenic problem.

Urine leakage is urine loss, without the subjective factors of social and hygiene.

Leak point pressure: is the value of the detrusor pressure, at the instant of starting of urine leak.

Obstruction implies that the urethral resistance to flow is abnormally elevated.

Bladder compliance: is defined as the change in detrusor pressure divided by the change in bladder volume.

Bladder outlet obstruction can be divided according to aetiology[10] as

1. fixed anatomic obstruction.
2. functional obstructions.

Functional in this case does not mean psychogenic, but simply indicating urethral resistance to forces of bladder emptying by neuro muscular phenomena, which may be voluntary or involuntary. Functional causes of bladder outlet obstruction include dyssynergia at the level of smooth sphincter and striated sphincter. Anatomic causes of bladder outlet obstruction include prostatic enlargement (benign or malignant) bladder neck contractive, urethral stricture.[9]

The obstructive symptoms (voiding symptoms) include weak stream, thin stream, straining, hesitancy, intermittency, terminal dribble, feeling of incomplete emptying and total retention.

Peripheral innervation of lower urinary tract is as:[11]

1. Sympathetic fibres - arise in segments T11-L2 pass via presacral hypogastric nerves and the sympathetic chain to the inferior hypogastric plexus and then to the bladder.

Sympathetic fibres convey afferent painful stimuli of over distension from the bladder to the brain. Impulse is transmitted via pelvic splanchnic nerves to sphincter urethra relaxing the latter.

2. Parasympathetic fibres derived from anterior primary division of S2-S4 segments. Fibres pass through pelvic splanchnic nerves to the inferior hypogastric plexus from which it is distributed to the bladder. Parasympathetic nerves carries the stretch reflex by distension of the bladder and causes detrusor muscle to contract.

3. Somatic innervation comes from S2, S3 and S4 sacral segments as pudendal nerves and supply urethral sphincter striated muscle.

Central nervous system influences on the lower urinary tract:

Micturition is basically a function of the peripheral autonomic nervous system. However the ultimate control of lower urinary tract function, including that of periurethral striated musculature resides at a higher neurological level.

Neural pathways:

Ascending sensory stimuli from lower urinary tract include exteroceptive (pain, touch and temperature generated in the urothelium) and proprioceptive sensory impulses (initiated in the bladder muscle). Exterioceptive impulse ascend in spinothalamic tract; synapse in the thalamus and project to sensory motor cortex.

Prioprioceptive impulses ascend in posterior column enter the dorsal portion of grey matter and turn rostrally to synapse in nucleus tegmentum of Pons. The region of cerebral hemispheres concerned with bladder muscle innervation consists of superomedial portion of frontal lobes and genu of corpus callosum.

Bradley's system of classification of neurogenic bladder is based on anatomic localisation of involved neural pathways and is as follows. [12]

Loop 1: Neural pathways connecting cerebral cortex (frontal lobe) micturition center (brain stem)

Loop 2: Pathways connecting micturition center to afferent and efferent fibres to the bladder in the sacral spinal cord.

Loop 3: Pathways connecting bladder sensory fibres to motor neurons of pudendal nucleus in the sacral cord.

Loop 4: Supraspinal and segmental innervation of periurethral striated muscle.

Cerebrovascular occlusive disease produces lesions in loop 1, and causes predominantly detrusor hyper reflexia.

REVIEW OF LITERATURE

The prevalence of CVA is approximately 60/1000 patients older than 65 years.[13]

The risk factors for stroke are, increasing age,hypertension, cardiac disease, diabetes mellitus, smoking, alcohol abuse and hyperlipidemia.[14]

The results of a CVA on the genito urinary tract are both direct and indirect. After a CVA, volitional control of voiding is often impaired.[15,16,17,18,19]

Urinary retention is the common symptom following an acute stroke. With time, this resolves to give rise to incontinence in a significant percentage of cases.

Taub et al[20] studied 639 stroke patients, of which 392 without previous disability survived, they were asessed for disability at 3 months. The results showed that 9% were severely disabled and 15% moderately disabled. In both groups initial incontinence happened to be the single best predictor of disability.[21]

The frontal cortex also bears the area concerned with voluntary control of external sphincter.[17,22] Input to Pontine Micturition Centre is derived from cerebral cortex, basal ganglia, cerebellum and spinal cord.

Bahita & Bradley[23] have demonstrated that the cerebral hemispheric region concerned with bladder detrusor muscle innervation is the superomedial portion of the frontal lobe. They further state that urinary dysfunction in patients with functional ablation of the cerebral cortical area, is characterized by a hyperactive detrusor reflex, due to impairment of inhibitory influences normally, derived from these cortical areas.[22]

Hald & Bradley[24] state that the net effect of cerebral infarction on micturition depends on the degree, size & location of the lesion. Generally, after the initial stroke episode, acute urinary retention occurs. The neurophysiologic explanation for this initial detrusor areflexia, called cerebral shock, is unknown[24]. There are relatively few prospective studies evaluating the urodynamics status of the acute stroke patient. The acute phase urinary retention may also be indirectly a result of impaired consciousness, restricted mobility, inability to communicate voiding desire, or temporary detrusor failure due to over-distension.[25] Also pre-exisiting bladder or bladder neck

dysfunction if any, may play a role in the urinary retention in the acute post stroke period.[32]

Gelber et al[17] in 1993, have prospectively evaluated bladder function in 51 patients after unilateral ischemic stroke, using urodynamic studies. Their results showed 21% of incontinent patients had hyporeflexia of the bladder; however a significant number among the group had underlying diabetes or were on anticholinergic medications.

Burney & Senapati,[4] in 1996 studied the urodynamics profile in acute stroke patients. Even thoough in acute stages, urinary retention is common, with time it normally resolves, and then incontinence becomes the more problematic urologic manifestations. They reported a high prevalence of urinary retention in association with cerebellar infarcts. Incontinence has been identified as being indicative of a poor prognosis after a stroke. (Wade & Hewer)[21]

Nishizawa et al[26] have demonstrated the influence of cerebellum in bladder behaviour in decerebrate dogs, as more of hyperreflexic nature. More studies are needed to elucidate the effect of cerebellar dysfunction or ablation on the human bladder.

Once the acute stroke phase is over the underlying bladder dysfunction becomes manifest as frequency or urge incontinence. This is due to the presence of detrusor hyperreflexia[18,19,27,28,31]

Tsuchida et al[28] performed urodynamic studies in 39 patients, after they suffered a stroke. In this, the mean interval between onset of stroke and urodynamic investigation was 19 months (Range 11 days to 13 years). In the majority of the patients the symptoms of urgency & frequency were secondary to detrusor hyperreflexia, which resulted in incontinence if suppression of these contractions did not occur.

The incidence of post stroke incontinence is reported to vary between 57% and 83% in the early stages after CVA.[17,33]

Some of these studies[15,25,29] have suggested that much of post stroke incontinence is transient, and possibly the result of forced immobility & impaired consciousness.

Borrie et al[25] report that the initial incontinence, fell to approximately half by 12 weeks. Incontinence had resolved in 80% of patients by 6 months.

Raz & Bradley[10] felt that the primary abnormality of bladder function post stroke was, a decreased sensation of gradual bladder distension, which would in the normal person give rise to a desire to micturate.

Most of the studies, assessing effects of CVA on the bladder, have been done in a retrospective manner.[16,18,19,28]

These studies reveal that detrusor hyperreflexia accounts for a large percentage of urinary dysfunction in post-stroke patients. This detrusor hyperreflexia was seen primarily in cortical lesions the incidence being 77% of cerebrovascular accidents - Khan et al.[9,10] Khan in his study of 33 patients tried to correlate the site of brain injury with the results of urodynamic testing; but was unable to draw any definitive conclusions.

Burney & Senapati[4] performed a prospective study which attempted to correlate the site of CVA as seen in imaging with findings on urodynamic testing. They evaluated 60 patients with confirmed CT or MRI lesions. Their findings suggest that the majority of fronto - parietal and internal capsular lesions resulted in detrusor hyperreflexia and AVCS (Absent Volitional Control of External Sphincter). Patients with tempero-occipital region lesions showed no abnormality of bladder function. The lesions responsible for areflexia included lesions in Basal ganglia, thalamus, pons & cerebellum. They also suggest that hemorrhagic infarcts resulted in an areflexic bladder, this is yet to be confirmed by subsequent studies.

EMG studies done by Taub, Wolfe et al[20] in supra pontine lesions, show absence of true DSD (Detrusor Sphincter Dyssynergia) in the majority of post stroke patients. However, pseudodyssynergia, characterized by voluntary contraction of the external sphincter during an involuntary detrusor contraction occurs, and should not be misinterpreted as loss of detrusor-sphincter co-ordination.

The effect of hemispheric dominance with regard to bladder dysfunction has been investigated by Khan et al[18]. He postulated that when CVA occurs in the non dominant cortex incontinence was less likely to result. However, Burney & Senapati, evaluated 44 symptomatic patients in the Parker Institute of Geriatic Care, New York but could find no definite influence of dominant hemispheric stroke on the bladder.[4,18,19]

MATERIALS AND METHODS

Patients for this study were selected from post stroke patients attending the outpatient clinics of department of Physical Medicine and Rehabilitation, and OPDs of Department of Medicine of CMC Hospital, and also from the medical OP of the Government Pentland Hospital, Vellore between January 1999 to May 2001. Study was conducted in Department of Physical Medicine and Rehabilitation, CMCH, Vellore, in collaboration with Urology Unit II.

A total number of 20 patients were included in the study. This study was presented to the Research Committee for approval and ethical clearance, which were obtained.

Inclusion criteria were:

1. Patients between 25-65 years of age.
2. Patients who have completed three weeks, but within three months, of onset of cerebrovascular accident in the middle cerebral arterial territory of either side.
3. Patients not having any pre-existing urinary complaint.
4. Patients who have had imaging, to confirm the site of the cerebrovascular accident.

Exclusion criteria were:

1. Patients with abnormal renal function, as indicated by elevated serum creatinine.
2. Patients with history of previous strokes.
3. Patients having an unstable general condition or aphasia or poor cognition status or those who were moribund.
4. Patients with a history of epilepsy, metabolic encephalopathy or prior brain trauma or surgery of any region of the brain.
5. Patients with symptomatic, lower urinary tract infection.

In order to select patients in compliance with the above mentioned exclusion and inclusion criteria, a thorough clinical and neurological examination was done. This was supplemented by CT imaging studies done during the period of acute hospitalisation for the CVA. A serum creatinine estimation was done to rule out gross abnormality of renal function as also a routine urine culture sensitivity for helping in sterilising the urinary tract prior to cystometrogram. Post void residual urine volume was determined by check catheterisation, after patient had voided voluntarily before starting to fill the bladder for cystometry. Filling of physiological body warm saline was done through the infusion pump, attached to the machine.

History of voiding difficulties were elicited in the questionairre. (Table 1; page 45) A sample of the questionnaire is attached in the appendix. (Page 58)

Patients underwent a water cystometrogram study, in the urology OP and the PMR Department, and the state of the detrusor activity and bladder compliance were deduced from the pressure change curve.

Method and Technique:

Patients were asked to empty the bladder as completely as possible. Bladder was catheterised by the sterile technique. Two infant feeding tubes (of size 6 and 8) in sterile packing, were introduced per urethrally till urine flowed freely, and the bladder was emptied. Residual urine was collected and measured. These two tubes were anchored by adhesive tape to the thigh, to prevent them slipping out during the procedure. The catheter was connected to the cystometer and calibrated. Pressure reference was the upper

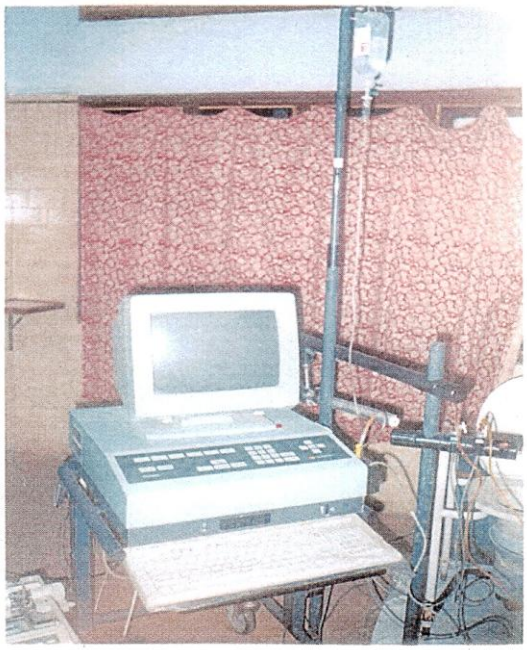
edge of the symphysis pubis. A 12 size infant feeding tube, with a condom attached to the distal end was passed 10 cms into the rectum, after lubricating with Xylocaine jelly. This served as an indicator of the intra abdominal pressure.

IV normal saline was infused into the bladder via, a volume recorder in the CMG machine, by the 8 size urethral catheter. Intravesical pressure recording was done by pressure sensitive diaphragm in the machine, which was connected to the size 6 urethral catheter.

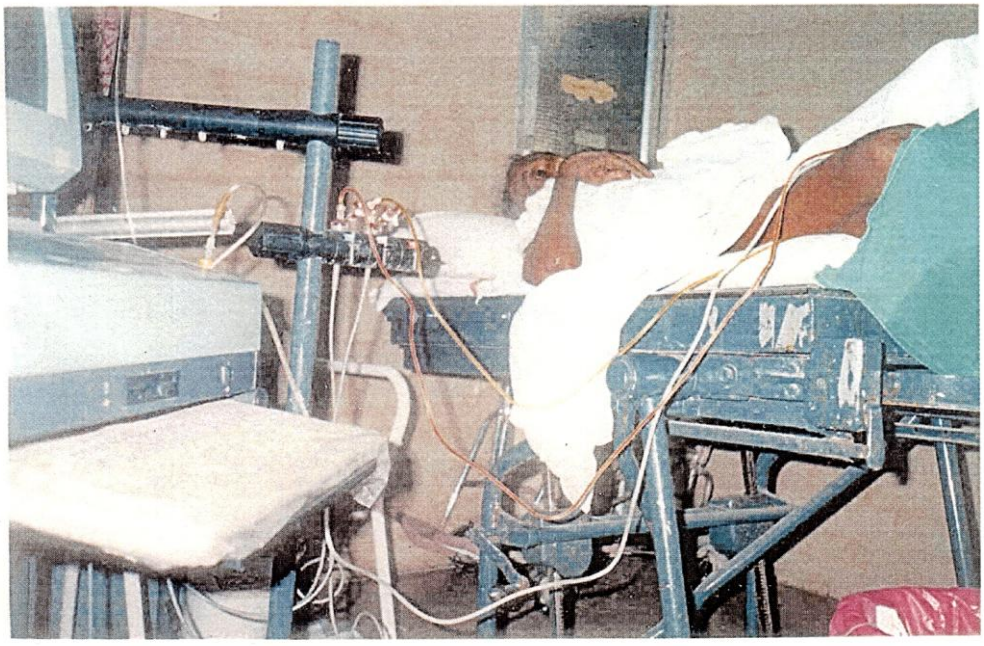
All the connecting tubes were emptied of air and filled with saline, before start of the test. The detrusor pressure was derived by subtracting the rectal pressure from the total bladder pressure and was displayed automatically by the machine.

Saline was infused into the bladder at a rate of 30-50ml per minute. The infused volumes at which patient experienced, first desire, normal desire, strong desire and urgency to micturate were recorded on the CMG infusion curve.

CMG Machine



Patient connected to CMG Machine



The machine used for recording CMG was Dantec, Menuette. This is a multichannel recorder with a weight transducer for measuring volume of infusion into the bladder. The intravesical pressure was read by Statham transducer and the flow was recorded by a rotating disc transducer. This machine had internal zeroing facility, to atmospheric pressure. Resting intravesical pressure ranges from 5-15 cm water when patient is supine and 15-40 cm water when sitting.

First desire to void:

First desire to void is the infused volume at which a desire to void or a sensation of filling begins. In a normal bladder, first desire occurs at a bladder volume of 50 - 400 ml.

Bladder pressure during filling:

The normal bladder accommodates to rapid changes in volume from zero to maximum cystometric capacity with a pressure increase of a few cm of water. Detrusor contractions are seen as more gradual, bell-shaped increases in bladder pressure.

Compliance is the change in volume divided by the change in detrusor pressure during that change.

Detrusor pressure:

Is the abdominal pressure subtracted from the bladder pressure.

Detrusor hyperreflexia is defined as overactive detrusor function caused by impaired nervous control of the detrusor reflex. An amptitude of 15 cm water is the minimum criterion for diagnosing an hyperreflexic detrusor contraction.[13]

Leak point pressure is the lowest detrusor pressure at which leakage occurs and may be used to assess the risk of upper urinary tract injury from lower urinary tract dysfunction. Patients with leak point pressure more than 40 cm of water are at high risk of complications. Leak point pressure is not an indicator for sphincteric competence or continence.

OBSERVATION AND RESULTS

Patient Profile:

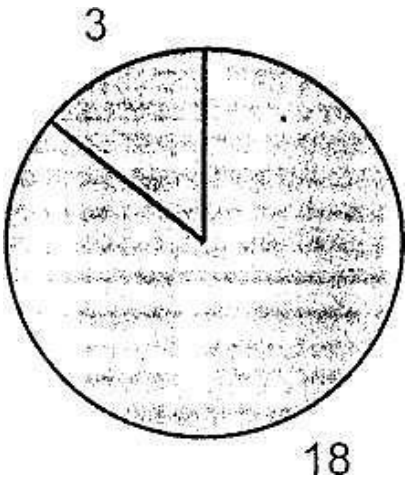
20 patients with post stroke for 3 weeks to 3 months were selected to observe the voiding pattern and urodynamic parameters. There were 17 males and 3 females in this group and their age ranged from 34 - 65 years

Study design:

Prospective descriptive study.

A standardised proforma was used to enter patient data.

PIE DIAGRAM SHOWING
GENDER DISTRIBUTION

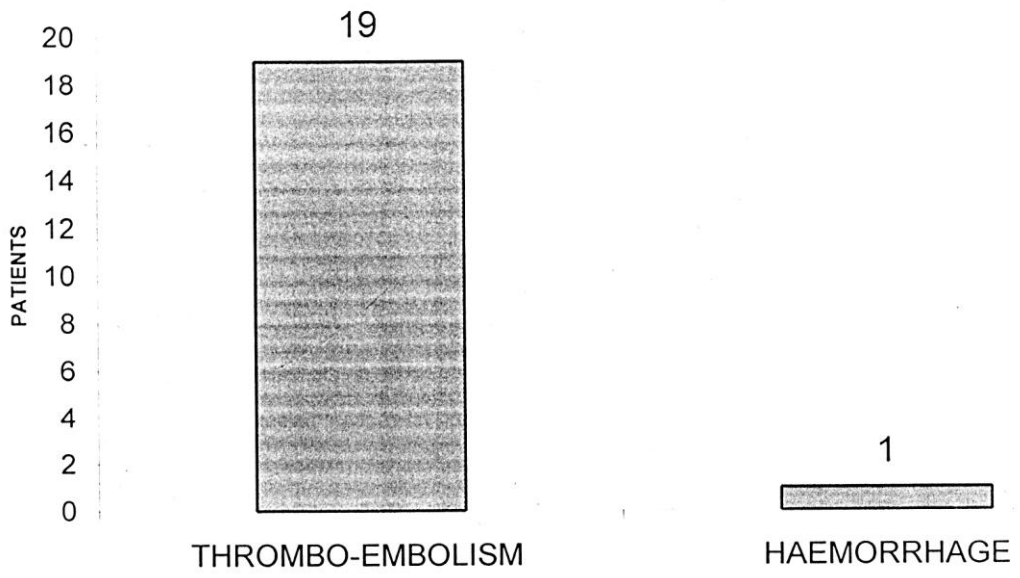


GRAPH 1

CAUSES AND TYPES OF LESION

19 patients had cerebral thrombo-embolism, and one patient cerebral haemorrage.

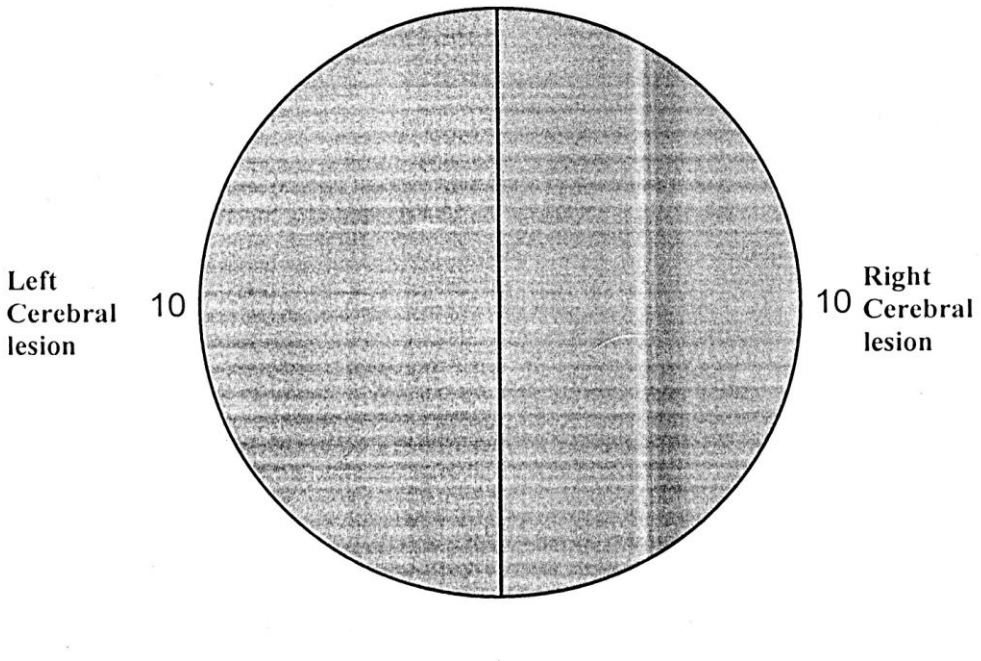
BAR DIAGRAM SHOWING CAUSES OF CVA



SIDE OF LESION:

Among the 20 patients, 10 patients had right sided cerebral lesion and 10 patients had left sided cerebral lesion. All patients were right hand dominant.

PIE DIAGRAM SHOWING SIDE OF CNS LESION

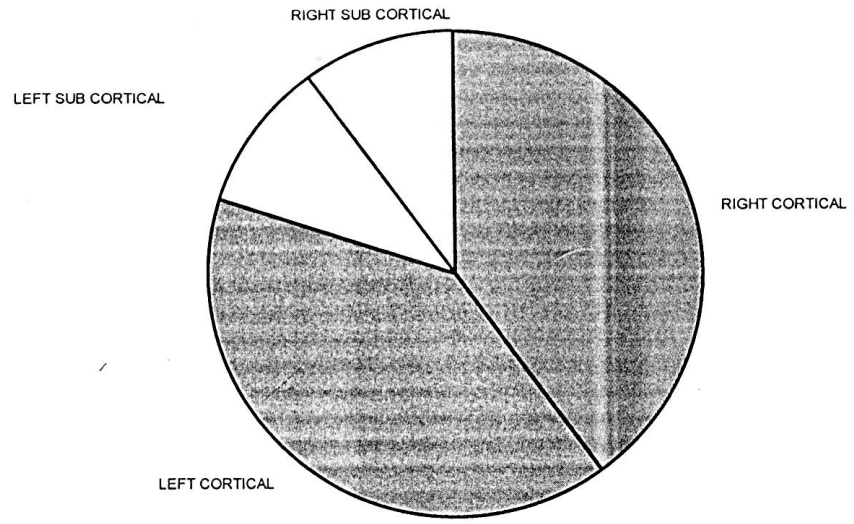


CT FINDINGS:

Site of lesions

Right Cortical	8
Right sub cortical	2
Left cortical	8
Left sub cortical	2

PIE DIAGRAM SHOWING DISTRIBUTION OF CNS LESION



VOIDING DISTURBANCES:

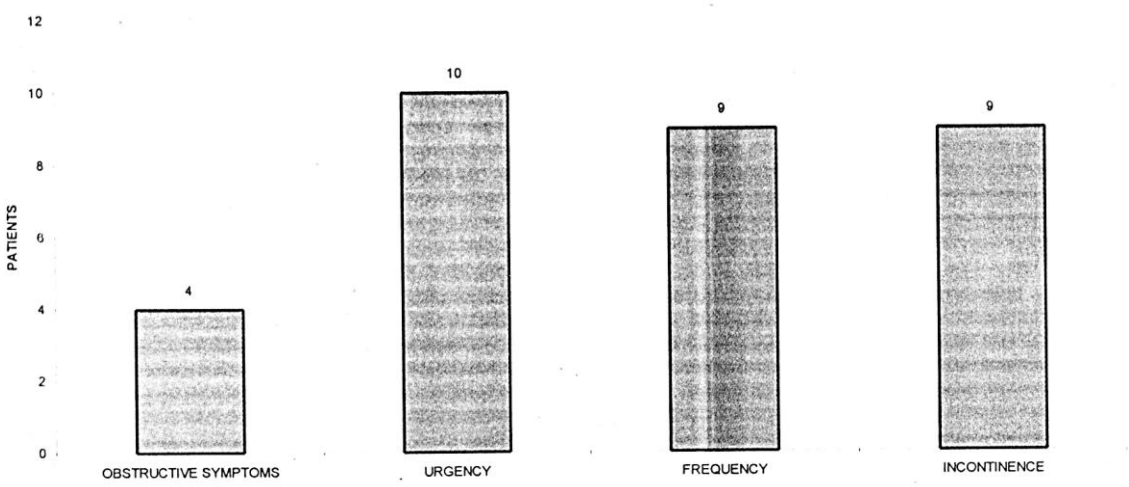
Only 4 patients had obstructive symptoms which were exacerbated by CVA.

10 patients reported urgency following the CVA.

9 patients had frequency after the CVA.

8 patients had incontinence

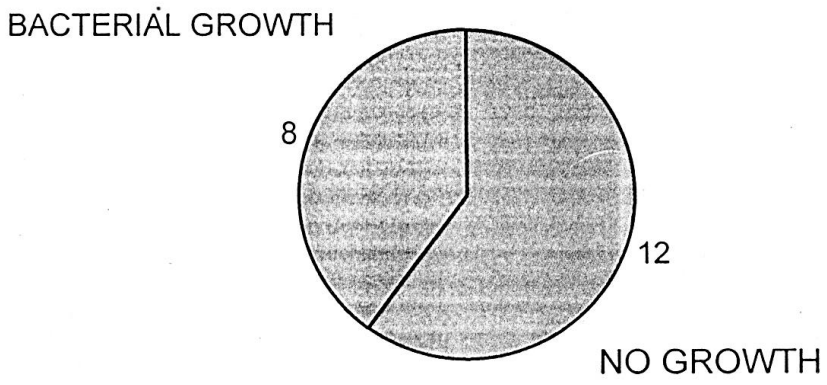
BAR DIAGRAM SHOWING INCIDENCE OF VOIDING DISTURBANCES ALL THE SUBJECTS AMONG



URINARY TRACT INFECTION

Urine culture and sensitivity was done before the CMG and among 20 patients 12 patients had no growth and 8 patients had significant bacterial growth, for which appropriate antibiotic was given.

PIE DIAGRAM SHOWING INCIDENCE OF URINARY INFECTION IN SUBJECTS



RENAL FUNCTION TEST

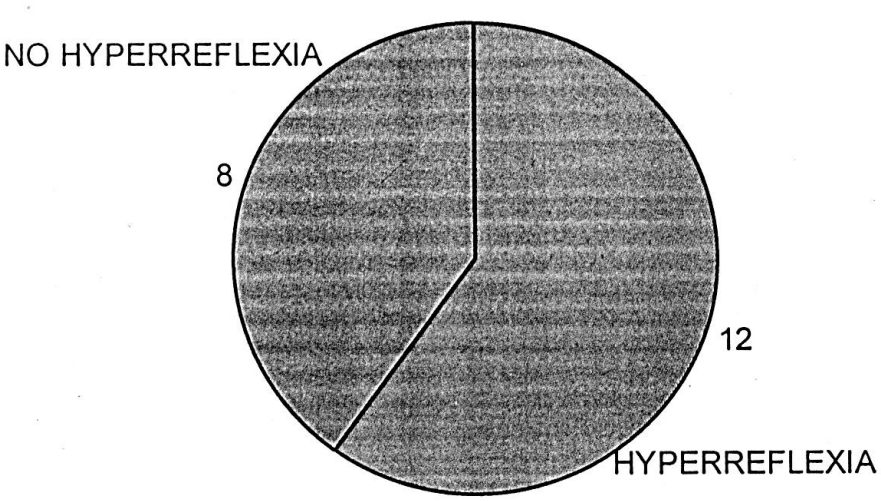
As determined by estimation of serum creatinine was within normal limit in all the patients.

There was no leak in 11 patients. The remaining 9 patients who had leak at pressures varying between 28 to 106 cms water.

CMG FINDINGS:

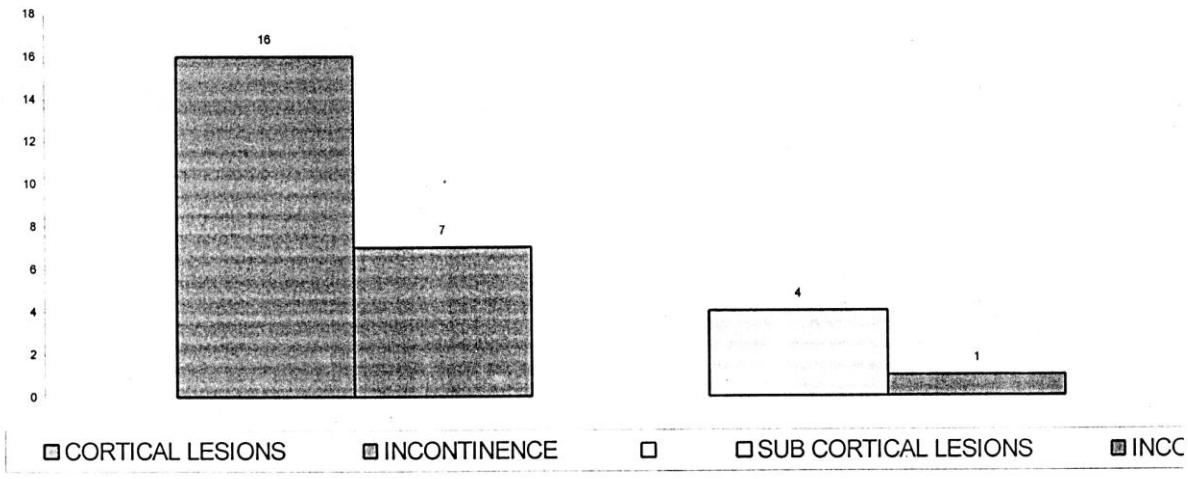
CMG tracing was done for all the 20 patients and 12 patients were found to have hyperreflexia and 8 had no hyperreflexia.

PIE DIAGRAM SHOWING INCIDENCE OF HYPERREFLEXIA IN ALL THE STUDY PATIENTS



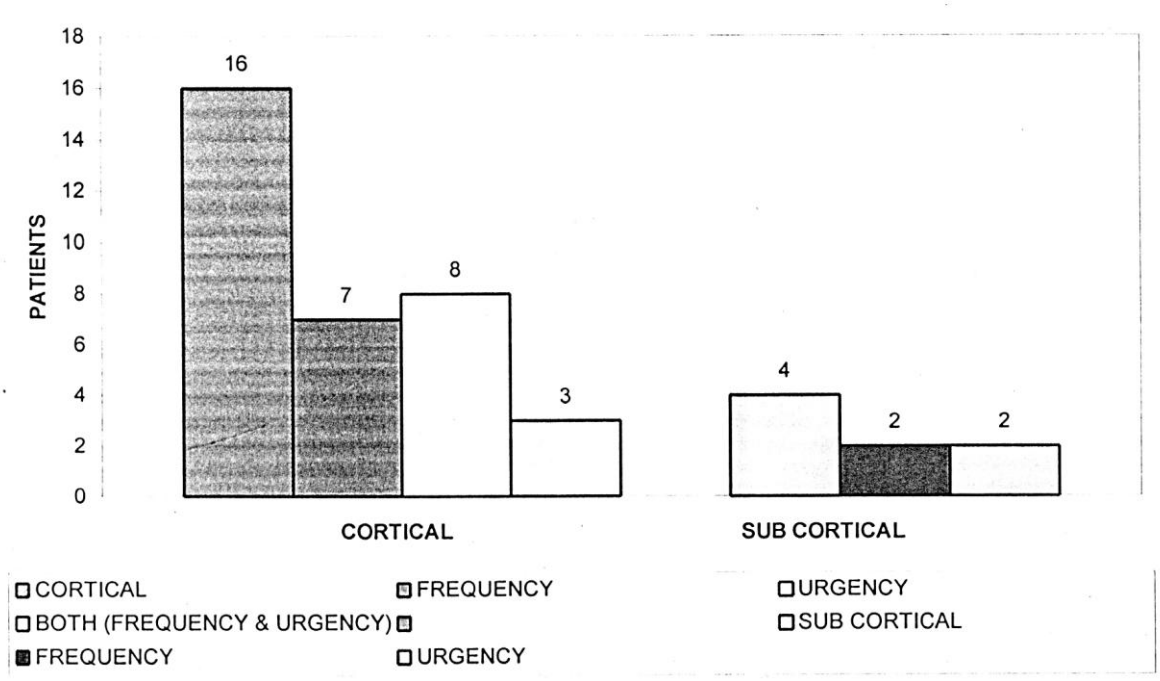
Of the 20 patients, 16 had cortical lesions and 4 patients had sub cortical lesions. Incontinence was present in 7 out of 16 patients with cortical lesions (43.75%) and 1 out of 4 patients with sub cortical lesions (25%)

BAR DIAGRAM SHOWING INCIDENCE OF INCONTINENCE IN CORTICAL VS SUB CORTICAL LESIONS



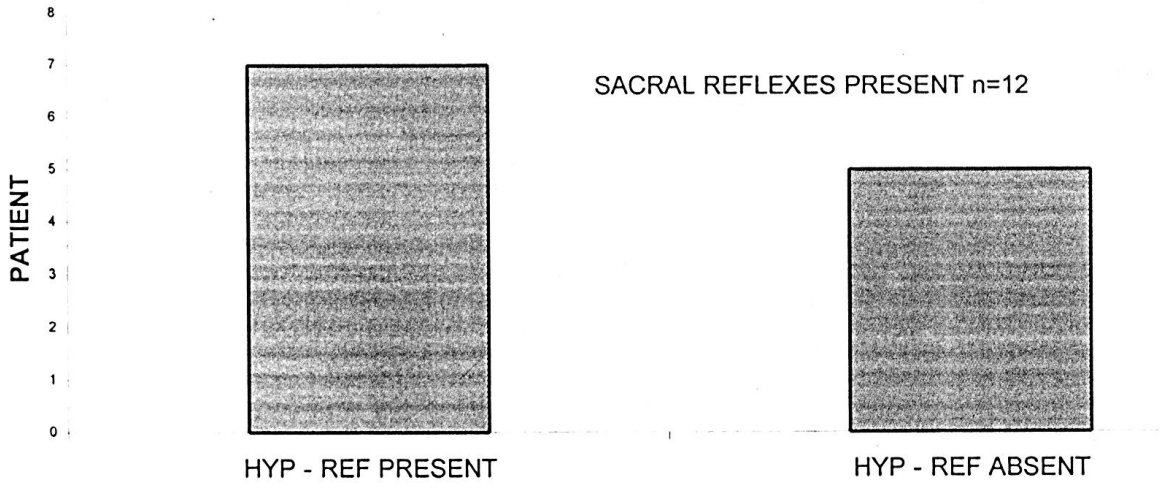
Among the 16 patients with cortical lesions, frequency was observed in 7 patients 8 patients has urgency and 3 patients has both urgency and frequency. 4 patients did not show any frequency or urgency. Among the 4 patients with sub cortical lesions frequency and urgency was present in 2 patients and the other 2 patients was no frequency or urgency.

INCIDENCE OF IRRITATIVE BLADDER SYMPTOMS IN CORTICAL VS SUB CORTICAL LESIONS



Sacral reflexes were present in 12 patients of whom 7 patients (58%) showed hyperreflexia in CMG. However, 5 patients (42%) did not show hyperreflexia.

CORRELATION BETWEEN SACRAL REFLEXES AND PRESENCE OF HYPERREFLEXIA

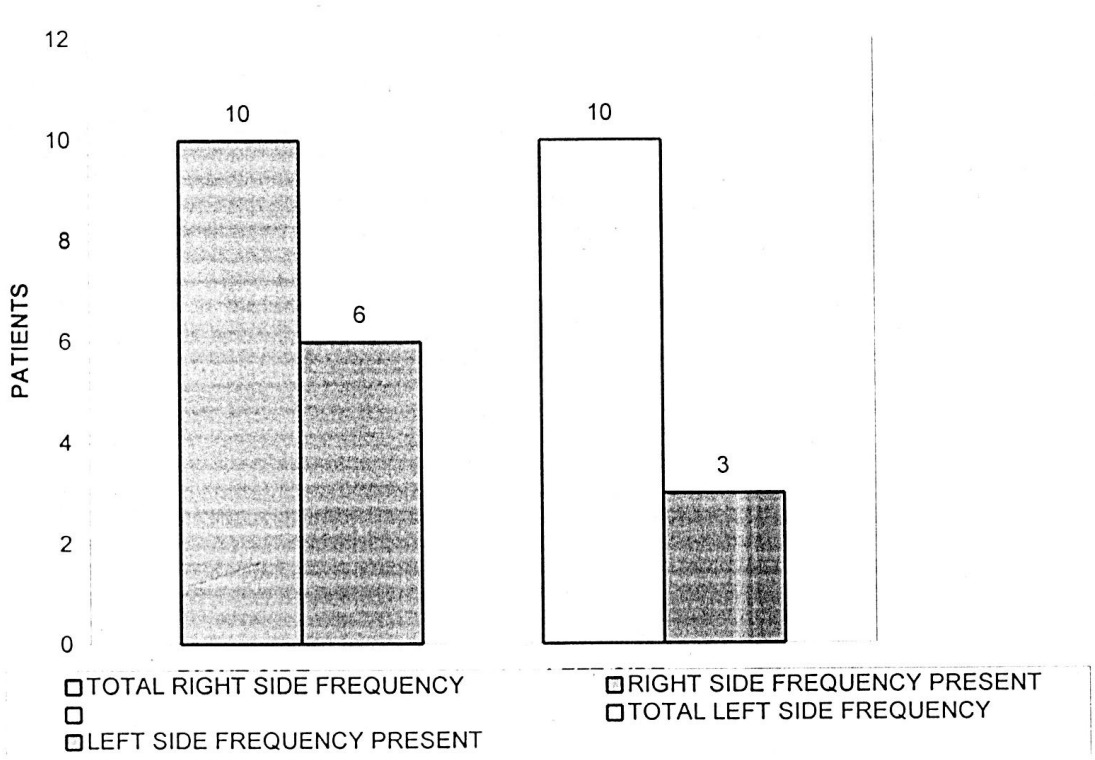


Frequency and side of brain lesion:

There were equal number of patients with right hemispheric lesion (10) and left hemisphere lesion (10). All these patients had left hemisphere dominance. Among the patients with left hemisphere lesion only 3 (30%) showed frequency.

Of the 10 patients with right hemisphere lesions 6 patients (60%) had frequency.

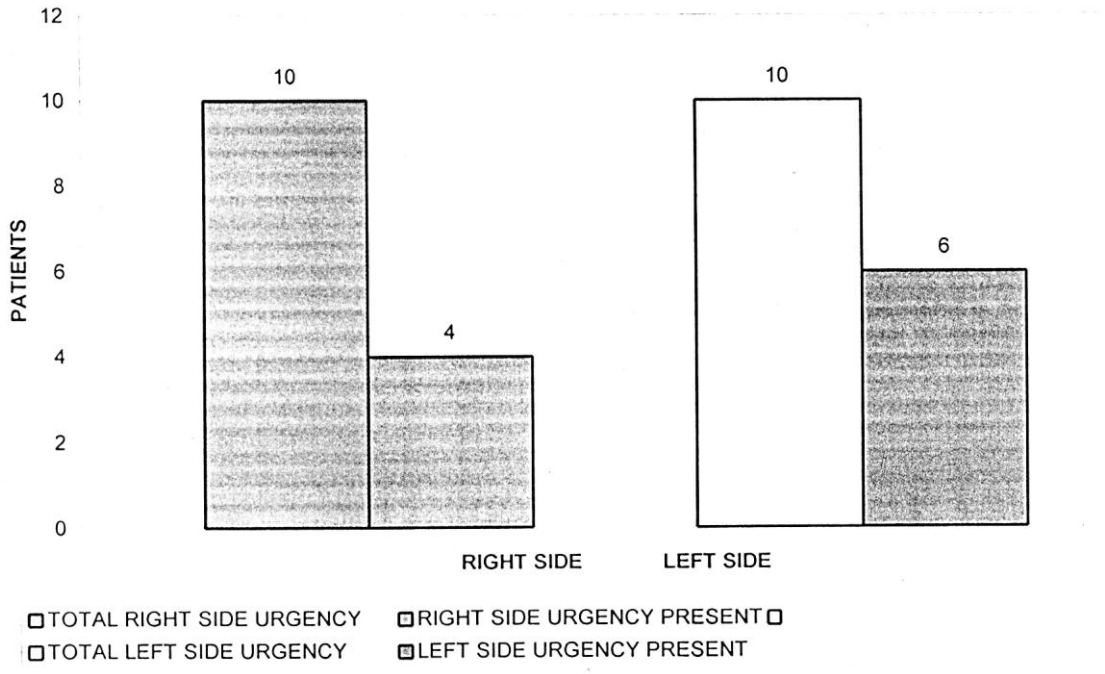
SIDE OF LESION VS FREQUENCY



Urgency and side of the brain lesion

Among the 10 patients with left hemisphere lesions 6 patients (60%) showed presence of urgency. Of the 10 patients with right hemisphere lesion 4 patients (40%) showed urgency. All patients were left hemisphere dominance.

SIDE OF LESION VS URGENCY



INCIDENCE OF VOIDING DYSFUNCTION SYMPTOMS

Patient No.	AGE	SEX	Obstructive symptoms	Urgency	Frequency	Incontinence	Voluntary voiding	Leak
1.	45	M	A	A	P	P	N	P
2.	41	M	A	A	A	A	N	A
3.	58	F	A	A	A	A	N	A
4.	48	F	A	A	A	A	N	A
5.	54	M	A	P	P	P	UNABLE	P
6.	57	M	A	A	A	A	N	A
7.	50	M	P	A	A	A	N	A
8.	63	M	A	P	A	P	N	P
9.	50	M	A	P	A	P	N	P
10.	60	M	A	P	P	A	N	A
11.	60	M	A	P	A	A	N	A
12.	63	M	A	A	A	A	N	A
13.	56	F	A	P	A	A	N	P
14.	65	M	A	P	P	P	N	P
15.	34	M	A	P	A	A	N	A
16.	56	M	A	P	P	P	N	P
17.	47	M	P	A	P	P	N	P
18.	52	M	P	A	P	A	N	A
19.	50	M	P	A	P	A	N	A
20.	40	M	A	P	P	P	N	P
			4/20	10/20	9/20		8/20	

A = Absent P = Present N = Normal M = Male F = Female

DISCUSSION

Stroke is classically described as, acute onset of a focal neurologic deficit, due to ischaemia or infarction, resulting from a vascular accident of the brain.

The cause of the ischemia leading to stroke, may be either an Occlusive process or Cerebral haemorrhage. The occlusive/embolic variety, accounts for roughly 80% of all strokes.

The risk factors for developing a stroke are:

Age

Hypertension

Cardiac disease

Diabetes Mellitus

Cigarette smoking

Alcohol abuse

Hyperlipidemia

Of the above, the single biggest risk factor is age, 70% of all strokes occur in individuals aged 65 and above.[-14]

The effects of a CVA on the genito urinary system can be devastating both directly and indirectly to the patient.[15,16] Voiding dysfunction as the result of a CVA, when superimposed on pre-existing voiding difficulties, causes a challenging diagnostic & therapeutic dilemma. Following an acute stroke urine retention commonly occurs. With time this resolves, and subsequently incontinence results in a significant number of patients, varying between 47 – 83% [15,25,29]

The present study was done to describe the voiding dysfunctions following stroke as demonstrated by urodynamic studies. Patients undergoing rehabilitation following stroke, between three weeks and 3 months after the CVA participated in the study.

All patients had undergone imaging of the brain, during the acute phase, to determine the site of brain lesion.

Patients beyond the age of 65 were not included because of the high incidence of obstructive lesions in the urethra. Patients with a past history of incontinence due to any cause, were also excluded. The other exclusion creiteria were, patients having previous history of stroke, patients with past history of epilepsy or brain surgery, patients having abnormal renal function as shown by elevated serum creatinine, patients with aphasia and cognitive deficits.

Patients satisfying the above criteria were physically examined regarding their neurological and cognitive status. This was followed by estimation of serum creatinine and doing urine culture. In those patients where urine culture showed microorganisms appropriate antibiotics were given, based on their sensitivity pattern. It was found that 12 patients did not have any growth on culture of urine. The remaining 8 had showed bacterial growth, commonest being E.coli (3 patients) followed by Enterococcus, proteus, and pseudomonous (1 each) (Diagram 4)

Following the acute stroke, urinary retention occurs commonly in a significant percentage of post CVA patients,. Normal voiding is a brain stem reflex, with input from suprapontine centers. As the detrusor contracts the striated sphincter relaxes and voiding occurs. In suprapontine CVA, this coordination may be affected causing unhibited detrusor contractions.

Urodynamics studies were performed on these 20 patients. CMG in 12 patients (60%) showed hyperreflexia, (Diagram 5) which compares near to figures reported in literature (77%). 11 patients did not develop any leak during the CMG. Of the 9 patients who showed leak, the leak point pressures varied between 28 cms to 106 cms of water. (Distribution curve shown, under observations & results) (Graph 4)

In this study, it was found that totally 8 patients (40%) had incontinence (n=20) (Graph 5) which is slightly lower than that reported in Western studies. This possibly could be having some correlation with the site of the brain lesion. The presence of urinary incontinence in the first seven days after the stroke is considered a poor prognosticator for survivor (Wade and Hewer). However, this could not be tested in this study due to absence of long-term follow up.

Dominant vs non dominant hemisphere lesion

On analysing the site of lesion in this study, 10 patients had infarct on the left cerebral hemisphere (dominant side) and 10 in the non dominant hemisphere (All patients were right hand dominant) (distribution shown under Diagram 2)

Unstable detrusor contractions were seen in 5 out of 10 dominant side lesions & 7 out of 10 non dominant side lesions.

7 of 10 (70%) dominant side cases, had absent sacral reflexes at time of doing CMG while it was present in 9 out of the 10 non dominant side lesions.

Among the patients with left hemisphere lesion only 3 (30%) showed frequency. Of the 10 patients with right hemisphere lesions 6 patients (60%) had frequency.

Among the 10 patients with left hemisphere lesions 6 patients (60%) showed presence of urgency. Of the 10 patients with right hemisphere lesion 4 patients (40%) showed urgency.

Sub cortical vs cortical lesion:

Considering the side of lesion along the neuraxis, 16 (80%) had cortical and 4 (20%) sub-cortical lesions.

On comparison of the 4 sub cortical vs 16 cortical lesions:

When compared with the incidence of incontinence after CVA none of the 4 sub cortical lesions showed incontinence while 7 out of 16 cases showed incontinence, among the cortical cases. (Graph 5)

2 out of 4 sub cortical cases (50%) showed hyperreflexia in CMG against 10 out of 16 (62.5%) cases demonstrating hyperreflexia in CMG.

Frequency as a symptom was seen in 2 out of 4 (50%) sub cortical cases, against 7 out of 16 (43%) cortical lesions. (Diagram 6)

Urgency was seen in 2 out of 4 (50%) subcortical cases, against 8 of 16 (50%) cortical lesions.

In this study 19 patients had cerebral thrombo embolism, and 1 patient had cerebral hemorrhage. Although a few studies in literature suggest higher incidence of areflexia in hemorrhagic intracranial lesions, this could not be commented upon since there was only one hemorrhage patient in this study.

In this study the age of the patients ranged from 34 - 63 years. The average time interval between the acute stroke and date of cystometric assessment was 6.47 weeks (range 3-12 weeks)

There was history of obstructive symptoms in 4 patients preceding the CVA; (Graph 2) these tended to occur in patients above 50 years (mean age 52 years) but was not significantly different from the mean age of the total group.

Urgency symptoms was present in 9 cases. 6 cases (66%) below 6 weeks from acute CVA 3 (33%) cases above 6 weeks from CVA.

Hyperreflexia in CMG, was seen in 12 patients of which 9 (75%) was below 6 weeks from acute stroke 3 (25%) was above 6 weeks from CVA.

Frequency was seen in 10 cases, 7 cases (70%) below 6 weeks, 3 cases (30%) above 6 weeks

Urine culture showed no growth in 12 cases (60%) and significant growth in 8 (40%) (Diagram 4)

Of the 12 no growth cases 8 (66%) had hyperreflexia in CMG.

Of the 8 cases with bacterial growth, 5 (62.5%) showed hyperreflexia in CMG.

Leak occurred in 9 cases (45%)

The leak point pressures ranged between 28 cm to 106 cm of H_20 (mean 62 cm of H_20) (Graph 4)

Of these 9 cases 6 cases (66%) had frequency as an associated symptoms

6 cases had cortical lesions & 3 had sub cortical lesions.

Of 9 cases with leak, 6 had no growth in urine culture, 3 had organisational growth in culture. So infection per se does not seem to contribute to causing leak.

Although there were equal number of patients with right and left hemisphere brain lesions, and all patients had left hemisphere dominance.

It was seen that urgency tended to occur in higher percentage (60%) of patients with dominant hemisphere stroke, compared to 40% of patients with non dominant hemisphere stroke.

Frequency as a symptom was reported by lesser number (30%) of patients with dominant hemisphere stroke, compared to 60% of patients with non-dominant hemisphere stroke.

CONCLUSIONS

Voiding dysfunction following cerebro vascular accident is certainly multi factorial. The symptomatology may be same for different types of dysfunctions.

Proper diagnosis of the cause and appropriate management of voiding dysfunction in the post stroke patient before the patient leaves the acute care facility is desirable because urinary dysfunction may complicate patient rehabilitation. In this institution we find that the average duration of acute care admission for strokes, is about a week.

Our study, although performed on a select population reveals that detrusor hyperreflexia accounts for the large percentage of urinary bladder dysfunction in post stroke patients.

The incidence of incontinence, which is supposed to be a bad prognosticator, was 40%.

Detrusor hyperreflexia is the major factor to cause incontinence, if not suppressed. Anti cholinergic agents for the treatment of detrusor, hyperreflexia have the potential to cause urinary retention.

- Detrusor hyperreflexia is the most common lower urinary tract dysfunction, associated with CVA.

- 60% of the CVA patients had hyperreflexia demonstrated in CMG.

- Unstable contractions, appear in a higher percentage (70%) of cases with non dominant hemisphere lesion.

- Incontinence symptoms was more prone to occur in patients with cortical lesions (43.75%) than in non cortical lesions (25%).

- The frequency & urgency symptoms showed no correlation between the brain hemisphere lesion.

- Urgency is not seen more frequently in cases below 6 weeks from CVA compared to cases above 6 weeks from CVA.

- Detrusor hyperreflexia occurred in 75% of the patients within 6 weeks of CVA, whereas only 25% patients with CVA had hyperreflexia after 6 weeks.

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