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DISCUSSION

SESSION ON URODYNAMICS

Discussion of papers by Wyndaele, Madersbacher, Gardner, Thomas, Cardozo, Hohlbrugger and Iwatsubo, on **Urodynamics and its Effects on the Treatment of the Neuropathic Bladder**.

Dr Key. (*Cape Town*). Mr Gardner, what does a residual urine of less than 100 mls of urine indicate? If the patient has a bladder capacity of only 110 mls and a residual urine of only 90 mls then, according to Mr Gardner, he will not have urodynamics performed.

Mr Gardner. (*Southport, Gt Britain*). I agree with this point. We take 100 mls as being the usual demarcation point between large and small residuals but clearly it is important to relate any residual to the bladder capacity.

Chairman (*Dr Hachen, Geneva*). Most spinal injury centres would consider 100 mls to be chronic retention. We usually aim for below 50 mls.

Mr Thomas. (*Sheffield*). Can we please get away from this idea of residual urine being very important? You can have a patient with complete paraplegia with reflex micturition who empties his bladder completely with no residual urine but with grossly high-pressure voiding. He can develop upper tract dilatation within two years of injury. I think it is vitally important that we look at the urodynamics of such a patient as well as his residual urine.

Professor Rossier. (*Boston*). I agree with Mr Thomas. You may have a patient with a very low residual, a normal IVP but grossly hypertrophied detrusor. He may have a reflux even in the absence of any changes of the IVP. I think it is important to get away from this idea of residual urine being important.

Dr Silver. (*Stoke Manderville*). When I went to Southport, rotating antibiotics were being used and I continued this practice. I then stopped it when I was told it was out of date. Does its present use in Southport reflect a change in policy? Secondly, I agree with your criticism of retrospective studies. This is going to give a tremendous slant. When I was in Southport one patient in three with complete tetraplegia died. With the Intensive Care Unit there now I am sure that many more tetraplegics are surviving and that therefore the latter part of the series will contain more tetraplegics than the early part.

Dr Linden. (*Cleveland, Ohio*). Question addressed to Mr Gardner: Firstly, Mr Gardner what do you mean by rotating antibacterials? This sounds to me like a rather empirical sort of procedure which horrifies a microbiologist. Secondly, is there any difference between the urine samples taken from the lower, mid and upper ureter from a bacteriological point of view? Thirdly, we find that a renal scan is more preferable as a method of determining differential renal function than bilateral ureteric catheterisation, firstly because it gives very good information on the relative function of the two sides and secondly there is no irradiation, and thirdly there are no dangers of introducing infection by inserting catheters into the ureters of infected patients.

Mr Gardner. Firstly, as regards rotating antibacterials, I agree that the treatment is empirical but if you have a patient with recurrent urinary infections whom you

cannot treat in any other way, then this is the only practical approach to his problem. Secondly, as regards the specimens taken from the lower, mid and upper ureter, we presume that if there is reflux present then the specimen in the lower ureter will reflect what is going on in the bladder, whereas that in the pelvis of the kidney is more likely to reflect what is going on in the renal parenchyma. Thirdly, the major reason for doing bilateral ureteric catheterisation is not to differentiate renal function but to perform bacteriological analysis on the two separate sides. If one contemplates performing a nephrouterectomy on a patient it is essential to make sure that it has no useful function for the patient. This will have been previously established by a renogram but the renal function tests using a ureteric catheter act as a belt and braces measure. Fourthly, there is no need for any irradiation with bilateral ureteric catheterisation. Fifthly, as regards rotating antibacterials, we tend to use antibiotics such as Nitrofurantoin for one month followed by Ampicillin for a month followed by Trimethoprim for another month and so on. I agree that the treatment is empirical but we are faced with a patient with a problem who wants to get home, and this is one solution, although it is somewhat imprecise. I emphasise that our presentation is a clinical one.

Dr Hachen. Professor Buzelin came to exactly the same conclusions two and a half weeks ago in Montpelier. This corroberates your findings.

Dr Hachen. It is important to use drugs carefully and appropriately in combination with urodynamics. This may gain time so that we do not operate on patients at too early a stage. The aim is to gradually reduce the dosage of the drugs towards the end of rehabilitation so that patients are not on any medication when they leave hospital.

Dr Madersbacher. (Bad Haring, Austria). I was very pleased to hear Mr Thomas's paper because it shows that Phenoxybenzamine is of no use in this group of patients. However, Edwards showed in studies on experimental animals that the duration of spinal shock could be shortened by giving them Phenoxybenzamine. Also, there are still some Centres where patients receive Phenoxybenzamine in the early stages following spinal cord injury. Do your findings suggest that this is nonsense?

Mr Thomas. No; I was careful to say that patients with well-established reflex micturition with obstruction from detrusor sphincter dyssynergia do not benefit. It may be of value in somebody who has low-pressure detrusor contractions for opening of the bladder neck in the very early phase of recovery of detrusor activity.

Professor Rossier. With regard to spinal shock, we have used Phenoxybenzamine for many months in acute cases without ever seeing any recovery from spinal shock. I therefore think that it is of no use. Secondly, I think we should be careful with Phenoxybenzamine. It was reported last year in Las Vegas that Phenoxybenzamine may be withdrawn very soon by the FDA because pleural and peritoneal cancer has been detected in animals on long-term Phenoxybenzamine.

Dr Madersbacher. Professor Rossier says that Phenoxybenzamine is of no benefit in the acute phase. Mr Thomas states that Phenoxybenzamine is of no use in the bladder with established reflex micturition and detrusor sphincter dyssynergia. It follows then that the only period during which Phenoxybenzamine is of benefit would be the short interval between these two periods when weak detrusor contractions are commencing and bladder outlet resistance is increased. Do you agree with this?

Mr Thomas. Yes.

Dr Meinecke. (*Hamburg*). We have the impression over the last two years that retraining bladder function in spinal cord injured patients by intermittent catheterisation is now taking much longer than it did in the past. I cannot find any explanation for this. In former years we needed about 6 to 8 weeks. Now, possibly because of the addition of modern drugs for spasticity, etc, it is taking much longer. Is this observation shared by other Centres?

Dr Wilmot. (*Santa Clara, USA*). I agree with this observation. We are now finding 6 to 9 months with no detrusor contraction. ICP is the treatment of choice with us and is almost a permanent solution. By using Clean-Cath and Macrodantin 50 mg b.d. they are doing very well. Thus we are using intermittent catheterisation almost totally now.

Dr Madersbacher. We have some experience with transurethral electrostimulation in an attempt to obtain detrusor contractions, and have demonstrated that slowly returning detrusor contractions are extremely sensitive to many drugs, even Valium and Lioresal. These drugs can immediately abolish weak detrusor contractions. I think that the increased use of all these drugs may be one of the reasons why the duration of catheterisation is prolonged.

Dr Wilmot. We are finding that more and more patients are preferring the method of intermittent catheterisation to the permanent use of a leg bag.

Dr Key. Our period of intermittent catheterisation has not altered. We only intermittently catheterise incomplete lesions. We do not intermittently catheterise complete lesions because we have 210 patients. However, we have found that Lioresal, Valium and Probanthine all influence detrusor contractions.

Dr Light. (*Houston*). One of the reasons why our patients may continue with prolonged catheterisation is that they may have a double lesion. We have been looking at patients who still have detrusor arreflexia after 6 to 8 months. All had cervical lesions. Using EMGs and lumbo-sacral evoked potentials we have found that the somatic sacral reflex arc is implicated.

Dr Hachen. How many members of the audience are still using the ice-water test to initiate detrusor contractions?

Dr Key. We do. We obtain a voiding picture on the cystourethrogram. We do not use it therapeutically.

Dr Wyndaele. (*Ghent, Belgium*). We use the ice-water test therapeutically when we have detrusor contractions which are rather weak in an attempt to increase them. It is sometimes beneficial.

Professor Rossier. I am biased by my past training with stimulators, in particular the bladder and conus medullaris stimulators. Your results are very good. What is your shortest follow-up and longest follow-up?

Dr Cardozo. (*London, Gt Britain*). In this series the shortest follow-up is eight months and the longest follow-up is over five years.

Professor Rossier. The general feeling of all those present here is that, when a stimulator is required to improve the efficiency of bladder voiding, the stimulator you have described is far superior to either the detrusor stimulator pacemaker or the conus medullaris stimulator.

Dr Kuhn. (*Switzerland*). We do not have any experience with the sacral root stimulators but we do have experience with other stimulators. We have noticed that they cause an increase in spasticity and subsequent increased difficulty in voiding. What is the experience in these patients with spasticity both pre- and post-operatively? Secondly, did the patients have drug medications pre- or post-operatively?

Professor Brindley. (*London, England*). Approximately half of our patients said that their pattern of leg spasms was altered after the operation. Most of them thought the alteration was for the worse either because of increased spasticity or because the spasms had changed from being flexor to being extensor, which they did not like so well. However, all patients who have had their implants in for more than a year have had their spasticity return to its initial state. Secondly, regarding drugs, many of them were taking drugs for spasticity. However, this is irrelevant because all the patients voided excellently whatever drugs they were on for their spasticity. None of the drugs used had any interaction with the micturition induced by the sacral anterior root stimulator. I would not have expected this on theoretical grounds and we have not observed it either.

Professor Rossier. Do you mean that the patients were taking the same drugs pre- and post-operatively?

Professor Brindley. If we are talking about drugs like Lioresal and Diazepam to reduce leg spasticity, these drugs have absolutely no effect whatever on the sacral anterior root stimulator and its use. There was some alteration in medication for spasticity post-operatively, but this was not under my control.

Mr Krishnan. (*London, Gt Britain*). As regards drug medication, with our four patients who have had this stimulator, although there was some initial increase in the prescriptions of Valium of Lioresal, the prescriptions of these all returned to the pre-operative base line levels later.

Professor Rossier. Though most of us are conservative in approach, I think we will find some definite indications for this technique in the future. For example, the tetraplegic female who is unable to transfer herself to the toilet and is unable to intermittently catheterise herself and with an inactive bladder, this technique might be the method of choice.

Professor Rossier. Dr Madersbacher, you mentioned that infection can greatly alter the characteristics of the urothelium. Did all these patients have sterile urine at the time of the different studies?

Dr Madersbacher. Yes.

Dr Wyndaele. Did you distinguish between patients with upper and lower motor neurone lesions?

Dr Madesbacher. You will see from one of the slides that our results were not altered by whether the patient had a reflex or an arreflexic bladder. When the lesion is complete the PD values fall, regardless of the level of lesion.

Mr Fellows. (*Stoke Mandeville, Gt Britain*). Ten to 15 years ago it was thought that there was no active transport of ions across mammalian urothelium. Clearly you have shown that this is out of date. Can you give some idea of the magnitude of this active transport across bladder urothelium compared with other epithelial surfaces elsewhere which are more considered to be surfaces at which active transport takes place, such as kidney, gall bladder and gut?

Dr Madersbacher. I and other investigators have shown that this active transport exists across human urothelium. However, I do not know its magnitude.

Dr Silver. We practice self-catheterisation at Stoke Mandeville. What worries us is over-distention. Autonomic hyperreflexia may result and I have seen this complication in one patient in whom the over-distention occurred at seven days and another in whom it occurred at ten days. Many of Dr Iwatsubo's patients had upper thoracic or cervical lesions but I do not see autonomic dysreflexia recorded as a complication of over-distention. Did you ever see this complication?

Dr Iwatsubo. (Iizuka, Japan). No, though I did look for it. I think autonomic dysreflexia occurs only after the spinal shock has worn off.

Dr Silver. Autonomic dysreflexia may occur when spinal shock is still present. This was so in my patients in whom the dysreflexia was shown at 7 and 10 days from injury and Dr Frankel and Dr Mathias have shown that tapping the bladder of patients with spinal shock may give rise to early changes of dysreflexia. So I think that the possibilities of intracerebral haemorrhage and death should be conveyed to the patients before they are given this therapy.

Professor Rossier. Firstly, Dr Iwatsubo has been extremely well trained, so that we can assume that he has not missed this complication. Secondly, what you describe, Dr Silver, is the exceptional situation in that autonomic dysreflexia does not usually arise in spinal shock. Furthermore, spinal shock requires definition and we also know that it can disappear within 24 hours. Hence, Dr Silver, it is possible that the cases you describe at seven and ten days were out of spinal shock. I personally have never seen dysreflexia in true spinal shock.