MODELLING OF THE SACRAL MICTURITION CENTRE USING A DELIBERATIVE INTELLIGENT AGENT

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Abstract: **This article presents a model of the sacral micturition centre, which belongs to the modelling of the lower urinary tract, based on a multiagent system. Results obtained in tests generate several urodynamic curves whose behaviour is closed to the one that can be obtained from a human being, studying both healthy individuals and people with dysfunctions due to neuronal causes.**
Keywords : **Modelling, multi-agent systems, lower urinary tract**

I. INTRODUCTION

The lower urinary tract (LUT) carries out two main functions, storage of urine in the bladder and the expulsion of urine via the urethra (micturition process). The LUT can be divided into two parts: the mechanical system and neuronal regulator [1]. The first part describes the LUT's biomechanics and the anatomy and physiology of the muscles and tissues that make it up. The second part refers to the anatomy and physiology of the neuronal control pathways, retransmission centres and exciter and inhibitory areas associated with micturition.

Different LUT models have been presented in several publications [2-5]. Most of these papers deal with simplifications and assumptions. Furthermore, they focus on solving the problem from a global approach. In this study, we deal with the problem from a distributed viewpoint, with emergent characteristics.

Our study consists of investigating and developing a model of the sacral micturition centre based on recently published qualitative studies. It has been developed from a formal multiagent-system-based frame which enables processes and partially well-known biological systems to be modelled [6]. We have used a computer simulation that permits model evaluation and the study of the behaviour of healthy individuals or with dysfunctions due to neuronal causes.

II. METHODS

The sacral micturition centre (SM) is situated in the intermediolateral portion medullary located in the sacral segment S2-S4. It is a neuronal centre related to the regulating biological function of urine elimination [1, 7].

Afferent pelvic signals from the detrusor and urethral musculature cross the intramural ganglia and pass along the pelvic nerves to the SM [1]. Depending on the intensity of the impulses registered by the receivers in the bladder and urethra, the centre can send impulses to certain supraspinal areas that intervene in the mictional process, such as the centre of the grey periaqueductal area, the preoptic area and other cortico-diencefalic areas that are micturition exciters or inhibitors [1, 8].

The parasympathetic efferent fibers are originated in the centre and are driven by the pelvic nerve to the wall of the bladder, the urethra and the pelvic plexus. Depending on the stimulation level from the inputs to the centre, efferent or motor impulses will be generated, causing contraction of the bladder and relaxation of the vesical neck and the striated intrinsic urethral muscle [9].

Anatomically and functionally, two involuntary reflexes have been identified: the vesicoparasympathetic one and the uretrovesical-parasympathetic one [1].

Based on the previous analysis, several inputs and outputs are identified in the centre. Regarding inputs, it presents one coming from the pontine micturition centre that indicates if it is necessary to empty the bladder or not ($^{PM}I_{SM}$), another one that provides detrusor pressure ($^{D}A_{SM}$), a third one that indicates urine flow through the urethra ($^{U}A_{SM}$) and a final input that comes from the thoracolumbar storage centre ($^{TS}I_{SM}$). Related to outputs, it presents one that goes to the detrusor muscle to contract it ($^{SM}E_{D}$), another one to the internal sphincter to relax it ($^{SM}E_{IS}$) and a third one that goes to the striated intrinsic urethral musculature to relax it ($^{SM}E_{IUS}$).

The SM centre will be inactive when the thoracolumbar storage centre has activated its output $^{TS}I_{SM}$. This happens during the storage phase.

Associated to the SM centre, two involuntary facilitator loops are identified at sacral level: the vesicoparasympathetic loop that is activated when the signal $^{D}A_{SM}$ goes beyond the threshold $^{D}H_{SM}$ and the uretrovesicalparasympathetic loop that is activated when the signal $^{U}A_{SM}$ goes beyond the threshold $^{U}H_{SM}$.

Bearing in mind that the neuronal centres are constantly registering information from the mechanical system and from other centres, and that they act as autonomous entities by means of efferent signals on the mechanical system and by means of internal neuronal

signals on other centres, we model the SM centre as an agent that presents a PDE (Perception-Deliberation-Execution) architecture [10] to which the capacity to memorize has been incorporated in order to obtain a richer and more powerful deliberation. With this consideration, the centre is made up of the following structure:

$$SM = \langle \Phi_{SM}, S_{SM}, Percept_{SM}, Mem_{SM}, Decision_{SM}, Exec_{SM} \rangle \quad (1)$$

where Φ_{SM} corresponds to the set of perceptions, S_{SM} corresponds to the set of internal status, $Percept_{SM}$ provides the centre with information about the state of the system, Mem_{SM} allows the centre to show awareness of the state, $Decision_{SM}$ selects the following influence and $Exec_{SM}$ represents the centre's intention of acting on the system. These functions present a general structure that depends on each centre's specific sets and functions [6]. We can appreciate the internal structure of a centre in figure 1.
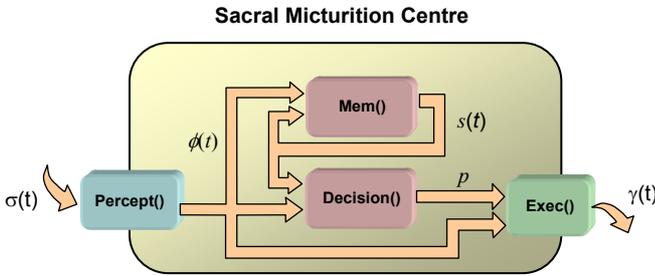
Sacral Micturition Centre

Fig. 1 Internal structure of the neuronal centre.

The perception function associated with the SM centre provides the group of signals of the state of the world whose origin or destination is this neuronal centre, and which depends on the group of afferent *(ANS)* and efferent *(ENS)* neuronal signals related to the centre:

$$ANS_{SM} = \left\{ {}^{D}A_{SM}, {}^{U}A_{SM} \right\}$$
$$ENS_{SM} = \left\{ {}^{SM}E_{D}, {}^{SM}E_{IS}, {}^{SM}E_{IUS} \right\} \quad (2)$$

The internal state of a centre is formed by the internal neuronal signals of origin or destination, that is to say, the input (INS_{SM}^{I}) and the output neuronal signals (INS_{SM}^{O}). In the case of the centre modelled in this article, the sacral micturition centre, only input signals are presented:

$$INS_{SM}^{I} = \left\{ {}^{PM}I_{SM}, {}^{TS}I_{SM} \right\} \quad (3)$$

The SM centre's associations shown in table 1 belong together to a state in which the centre does not provide any signal (I); a state in which the centre has been stimulated by vesical afferent signals, providing a low-level micturition exciter efferent intensity (MB); another condition would be given when the centre is stimulated by urethral afferent signals, a fact that facilitates a

medium-level micturition exciter efferent intensity (MM); a fourth situation would be when the centre is stimulated by vesical and urethral afferent signals providing a high-level micturition exciter efferent intensity (MA); the following state would correspond to the situation in which the centre is in the storage phase and, therefore, does not provide any signal (R); the last one would be the state in which the centre is stimulated by its afferent

Table 1 Definition of the translation function. A - association; ϕ - perception; **s** - internal state.

Condition	A
$(\phi.{}^{D}A_{SM} < {}^{D}H_{SM}) \wedge (\phi.{}^{U}A_{SM} < {}^{U}H_{SM}) \wedge \neg \mathbf{s}.{}^{PM}I_{SM} \wedge \neg \mathbf{s}.{}^{TS}I_{SM}$	I
$(\phi.{}^{D}A_{SM} \geq {}^{D}H_{SM}) \wedge (\phi.{}^{U}A_{SM} < {}^{U}H_{SM}) \wedge \neg \mathbf{s}.{}^{PM}I_{SM} \wedge \neg \mathbf{s}.{}^{TS}I_{SM}$	MB
$(\phi.{}^{D}A_{SM} < {}^{D}H_{SM}) \wedge (\phi.{}^{U}A_{SM} \geq {}^{U}H_{SM}) \wedge \neg \mathbf{s}.{}^{PM}I_{SM} \wedge \neg \mathbf{s}.{}^{TS}I_{SM}$	MM
$(\phi.{}^{D}A_{SM} \geq {}^{D}H_{SM}) \wedge (\phi.{}^{U}A_{SM} \geq {}^{U}H_{SM}) \wedge \neg \mathbf{s}.{}^{PM}I_{SM} \wedge \neg \mathbf{s}.{}^{TS}I_{SM}$	MA
$\neg \mathbf{s}.{}^{PM}I_{SM} \wedge \mathbf{s}.{}^{TS}I_{SM}$	R
$\mathbf{s}.{}^{PM}I_{SM}$	M

inputs and by the signal coming from the pontine micturition centre, which provides a complete efferent intensity (M).

By means of a translation function, ts(t), the centre's internal states are associated with the different segments of the vesical pressure curve, identified by the different phases of the system [11] (I corresponds to a inactive state; MB, MM, MA and M to a micturition state; R is a retention state), thus simplifying its current state. The definition of this function is shown in table 1.

According to its current state and what it perceives, the centre will be able to change to a new state and decide what action to take. The decision function ($Decision_{SM}$) presents a general structure that depends on its internal functions ($PreD_{SM}()$ and $FunD_{SM}()$) [6].

The same as the decision function, the memorization function (Mem_{SM}) also presents a general structure dependent on its internal functions ($PreM_{SM}()$ and $FunM_{SM}()$) [6]. These functions are defined in table 2.

III. RESULTS

To validate the model of the SM centre we have carried out different LUT simulations with data regarding both healthy individuals and those with dysfunctions due to neuronal causes.
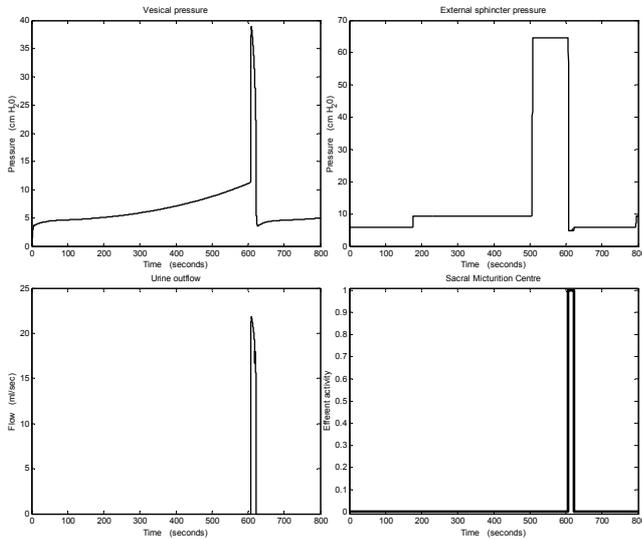
A. Situation without dysfunctions

The result of the tests with the centre working normally, without dysfunctions, can be observed in figure 2. In the storage phase, the centre remains inhibited,

Table 2 Internal functions of decision and memorization. ϕ - perception; s - internal state; $ts()$ - translation function.

$\phi.^D A_{SM}$	$\phi.^U A_{SM}$	$s.^{PM} I_{SM}$	$s.^{TS} I_{SM}$	$ts(t)$	$PreD_{SM}()$	$FunD_{SM}()$	$PreM_{SM}()$	$FunM_{SM}()$	$ts(t+1)$
$< {}^D H_{SM}$	$< {}^U H_{SM}$	0	0	I,M	False	$<(\phi.^{SM}E_D,0),(\phi.^{SM}E_{IS},0),(\phi.^{SM}E_{IUS},0)>$	False	$\langle\ \rangle$	I
$\geq {}^D H_{SM}$	$< {}^U H_{SM}$	0	0	I,MB,MA	True	$<(\phi.^{SM}E_D,0.2),(\phi.^{SM}E_{IS},0.2),(\phi.^{SM}E_{IUS},0.2)>$	True	$\langle\ \rangle$	MB
$< {}^D H_{SM}$	$\geq {}^U H_{SM}$	0	0	I,MM,MA	True	$<(\phi.^{SM}E_D,0.25),(\phi.^{SM}E_{IS},0.25),(\phi.^{SM}E_{IUS},0.25)>$	True	$\langle\ \rangle$	MM
$\geq {}^D H_{SM}$	$\geq {}^U H_{SM}$	0	0	I,MA,MM	True	$<(\phi.^{SM}E_D,0.45),(\phi.^{SM}E_{IS},0.45),(\phi.^{SM}E_{IUS},0.45)>$	True	$\langle\ \rangle$	MA
X	X	0	1	MB,R	True	$<(\phi.^{SM}E_D,0),(\phi.^{SM}E_{IS},0),(\phi.^{SM}E_{IUS},0)>$	True	$\langle\ \rangle$	R
X	X	1	X	R,M	True	$<(\phi.^{SM}E_D,1),(\phi.^{SM}E_{IS},1),(\phi.^{SM}E_{IUS},1)>$	True	$\langle\ \rangle$	M

allowing the bladder to be filled. During the emptying phase, we can observe how the person activates the SM centre to contract the detrusor and thus causing expulsion of urine to the exterior.

Fig. 2 Urodynamic data in a situation without dysfunctions.

During the first part of the storage phase, we can observe that an increase in urine generates exponential increases in pressure due to the initial stretching of the muscle. As it becomes full, pressure does not increase so much due to the adaptation of the muscle. When the micturition begins, a contraction of the detrusor takes place generating a great increase in vesical pressure, reaching values of 40 cm of H_2O. During the emptying phase, pressure decreases according to the quantity of urine in the bladder. At the end of the micturitional process, the bladder will have practically emptied, maintaining a basal pressure.

During the rest of the storage phase, external sphincter pressure increases. Initially, the external sphincter presents a basal pressure of 6 cm of H_2O. When the vesicosomatic guarding loop is activated, the pressure will increase to 9 cm of H_2O. At the end of the filling phase, the person voluntarily wants to retain urine and the external sphincter contracts and is able to increase its pressure to 65 cm of H_2O. When the emptying phase begins, the guarding centres are inhibited and the generated pressures decrease, letting the urine pass from the bladder to the urethra and from there to the exterior.

The urine flows to the exterior when the external sphincter is opened. It can be seen how the output flow of urine increases to 23 ml/seg.

B. Situation with neuronal dysfunctions

When there is a lesion that affects interaction among the sacral centres and the rest of the neuronal centres (thoracolumbar centre, pontine centre and suprapontine centres), interaction ceases. The LUT no longer controls voluntary and involuntary areas, but the vesicosomatic guarding reflex and the vesicosimpatic and urethralparasympathetic reflexes of micturition remain.
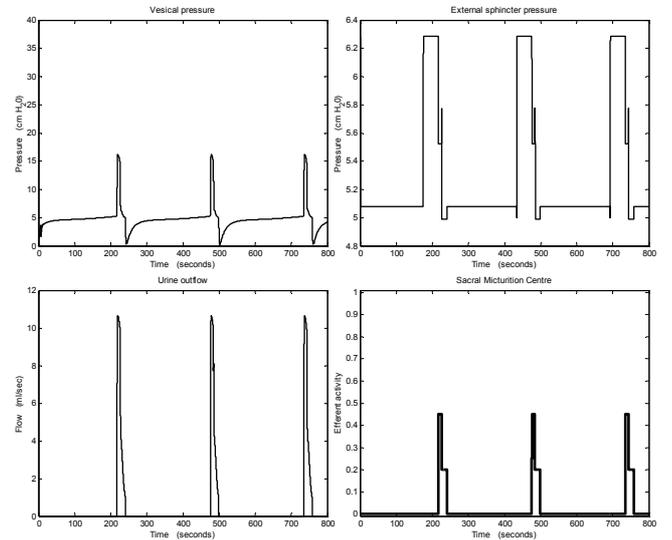
Fig. 3 Urodynamic data in a suprasacral dysfunction.

Figure 3 shows the urodynamic curves of a suprasacral lesion. In it, detrusor-sphincter disinergy can be observed: at the same time as the detrusor is

contracted, the external sphincter is also contracted. When detrusor pressure is greater than sphincter pressure, urine loss takes place. The SM centre is activated involuntarily due to the reflexes.

IV. DISCUSSION

In this work we investigate the dynamic behaviour of the sacral micturition centre as an autonomous entity that perceives, deliberates and executes actions to carry out the micturitional process in an appropriate way. Different situations of the model have been studied using computer simulation.

On observing the data results of the simulations (vesical pressure curves, output flow, electromyography of the external sphincter), adaptation during the storage phase and the electromyographic silence of the external sphincter in the emptying phase can be appreciated. We can also see that vesical pressure, the maximum flow rate and maximum urethral pressure fall within the permitted ranges for the International Continence Society [12].

With a suprasacral lesion, we have observed that a person does not retain urine voluntarily and begins the emptying process in the presence of a small quantity of urine in the bladder. These data are similar to real data and usually generate a detrusor-sphincter disinergy [13].

Other published works study the sacral micturition centre as a reactive system in which its emergent behaviour is not taken into account [3]. Highlighting the centre as an agent not only allows the emergent behaviour to be studied, but it also incorporates the modular characteristic, enabling possible improvements to be made on the system when we have new knowledge about the operation of the LUT, and I also allows the individualized study of each centre that belongs to the LUT.

V. CONCLUSION

We have proposed a model of the sacral micturition centre that presents a behaviour comparable to the one obtained with healthy people or individuals with dysfunctions due to neuronal causes.

The use of the intelligent agents paradigm as a tool for modelling permits the incorporation of cognitive aspects that act as base elements for the development of artificial LUT control.

This study can be used as a basis to obtain finer and more rigorous models and to develop a control system for incontinence due to neuronal causes.

We are currently investigating the development of other centres belonging to the LUT, as well as their integration and control using a multiagent system, which will allow monitoring systems and diagnostic help to be carried out.

REFERENCES

[1] M. V. Kinder, E. H. C. Bastiaanssen, R. A. Janknegt and E. Marani, "The Neuronal Control of the Lower Urinary Tract: A Model of Architecture and Control Mechanisms", *Arch Physiol Biochem*, vol. 107, pp. 203-222, 1999.

[2] E. H. C Bastiaanssen EHC, J. L. van Leeuwen, J. Vanderschoot and P.A. Redert, "A myocybernetic model of the lower urinary tract", *J Theor Biol*, vol. 178, pp.113-133, 1996.

[3] F. van Duin, P. F. Rosier, B. L. Bemelmans, H. Wijkstra, F. M. Debruyne and A. van Oosterom, "Comparison of different computer models of the neural control system of the lower urinary tract", *Neurourol Urodyn.*, vol. 19, pp. 289-310, 2000.

[4] J. M. García, A. Soriano, , F. Ibarra and F. Maciá F, "Urodynamic model of the lower urinary tract", *Proc. of CIMCA'99*, pp. 123-128, 1999.

[5] F. A. Valentini, G. R. Besson, P. P. Nelson and P. E. Zimmern, "A mathematical micturition model to restore simple flow recordings in healthy and symptomatic individuals and enhance uroflow interpretation", *Neurourol Urodyn*, vol. 19, pp. 153-176, 2000.

[6] A. Soriano, *Modelado y Simulación del Regulador Neuronal del Tracto Urinario Inferior*, Thesis, University of Alicante, 2001.

[7] J. Gallego and E. Martínez, "Farmacoterapia en la disfunción vesicouretral", in *Urodinámica Clínica*, J. Salinas and J. Romero, Eds. Merck Sharp & Dohme, 1995, pp. 537-560.

[8] B. F. M. Blok and G. Holstege, "The central control of micturition and continence: implications for urology", *Brit. J. Urol.*, vol. 83, pp. 1-6, 1999.

[9] N. Yoshimura, "Bladder afferent pathway and spinal cord injury: possible mechanisms inducing hyperreflexia of the urinary bladder", *Progress in Neurobiology*, vol. 57, pp. 583-606, 1999.

[10] J. Ferber, Multi-Agent Systems. An Introduction to Distributed Artificial Intelligence, Addison-Wesley, 1999.

[11] J. M. García, F. Maciá , A. Soriano and F. Flórez, "A Multi-Agent System uses Artificial Neural Networks to Model the Biological Regulation of the Lower Urinary Tract", *Proc. of WSES NNA 2002*, Accepted 2002.

[12] D. Castro, "Urodinámica. Generalidades y terminología de la función del tracto urinario inferior", in *Urodinámica Clínica*, J. Salinas and J. Romero, Eds. Merck Sharp & Dohme, 1995, pp. 61-78.

[13] J. R. Sotolongo, "Causes and treatment of neurogenic bladder dysfunction", in *Clinical Urology*, R. J. Krane et al., Eds. J.B. Lippincott Company, 1994, pp. 558-568.