

A Multi-Agent System uses Artificial Neural Networks to Model the Biological Regulation of the Lower Urinary Tract

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Abstract: - The robustness that shows the biological regulation of the human lower urinary tract provides a suggestive paradigm for the artificial control. The biological regulator consists of a heterogeneous group of nervous centres that act cooperatively, in a distributed way. That regulation conforms a behaviour of several types (autonomous work or conscious one) and it reduces the consequences in situations of bad operation. Related to that system, we propose a model of the paradigm of heterogeneous and distributed control that can be found in biological systems. The objective is to artificially reproduce the benefits of robustness in order to use it in the control of natural systems and artificial devices. The distributed aspects have been obtained using multiple intelligent agents, each one of which represents one of the biological centres. The interaction pattern among agents provides a heuristic based on the OAM neural network (Orthogonal Associative Memory). The knowledge has been added to the system by training, using correct patterns of behaviour of the urinary tract and wrong behaviour patterns due to the inoperability in up to two of the agents (representing deficiencies in up to two nervous centres at the same time). The experiments show that the model is robust and it satisfies the expectations of providing a model of the regulator system that allows to break into fragments the problem, in simple modules with own entity each.

Key-Words: - Multi-Agent Systems, Distributed Artificial Intelligence, Neural Networks, Lower Urinary Tract.

1 Introduction

The lower urinary tract (LUT) carries out two main functions, the storage of urine in the bladder and the expulsion of urine through the urethra (micturition process). Micturition consists of the expulsion of urine stored in the bladder through the urethra to the exterior and this depends mainly on co-ordination between the detrusor and the external sphincter [1]. This coordination is carried out by neural control which involves a complex system of interconnections and nerve centres. The LUT can be divided into two parts; Mechanical System of Lower Urinary Tract (MSLUT) and Neural Regulator of the Lower Urinary Tract (RLUT). The first part describes the LUT's biomechanics and has to do with the anatomy and physiology of the muscles and tissue that make it up. The second part is referred to the anatomy and physiology of the neural control pathways, retransmission centres and exciting and inhibitory areas associated with micturition [2].

Different LUT models have been presented in various publications [3][4][5][6]. Most of these papers accomplish simplifications and assumptions. Furthermore, they focus on solving the problem from a global approach. In this paper, we solve the problem from a distributed viewpoint.

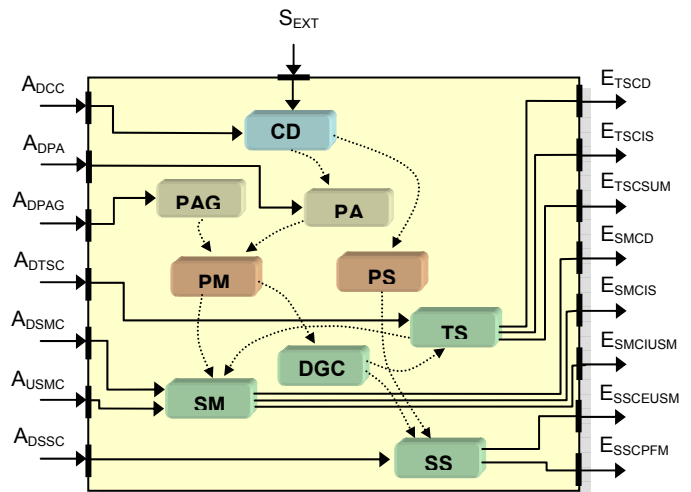


Fig.1. Structure of RLUT.

This paper presents a multi-agent framework for the neural regulator of the lower urinary tract (RLUT) model proposed in [7] that incorporates a heuristic that makes it more robust in presence of possible inconsistencies. The agents which constitute the Multi-agent System (MAS) correspond to the neural centres of RLUT. These agents collect information generated by the MSLUT and process/transmit it back towards the mechanical part. Each agent makes

a contribution to the system, called an influence, in such a way that the total count of the different influences will determine the overall state of the system and the activation or non-activation of the different involved signals.

However, this type of systems should face continuous inconsistencies caused so much for the own nature of the problem, like for the fact of working with a system that is not still known in all its dimension.

In our investigation we have identified several centres: Cortical-Diencephalic (CD), Preoptic Area (PA), Periaqueductal Grey (PAG), Pontine Micturition (PM), Pontine Storage (PS), Sacral Micturition (SM), Dorsal Grey Commissure (DGC), Sacral Storage (SS), Thoracolumbar Storage (TS). Figure 1 shows the model inputs and outputs, the agents of which it is comprised and their interrelations. In table 1 is shown the model inputs and outputs.

Signal	Description
S_{EXT} :	Signal that indicates whether the patient is in an appropriate environment to initiate micturition.
A_{DCC} :	Afferent Nervous Signal (ANS) generated by the Detrusor tension and arrives at the CC.
A_{DPA} :	ANS generated by the Detrusor tension and arrives at the PA.
A_{DPAG} :	ANS generated by the Detrusor tension and arrives at the PAG.
A_{DTSC} :	ANS generated by the Detrusor tension and arrives at the TSC.
A_{DSMC} :	ANS generated by the Detrusor tension and arrives at the SMC.
A_{DSSC} :	ANS generated by the Detrusor tension and arrives at the SSC.
A_{USMC} :	ANS generated by the urine flow through the Urethra and arrives at the SMC.
E_{SMCD} :	Efferent Nervous Signal (ENS) generated by the SMC and arrives at the Detrusor to contract it.
E_{TSCD} :	ENS generated by the TSC and arrives at the Detrusor to relax it.
E_{SMCIS} :	ENS generated by the SMC and arrives at the Internal Sphincter (IS) to relax it.
E_{TSCIS} :	ENS generated by the TSC and arrives at the IS to contract it.
E_{TSCSUM} :	ENS generated by the TSC and arrives at the Smooth Urethral Muscle to contract it.
$E_{SMCIUSM}$:	ENS generated by the SMC and arrives at the Intrinsic Urethral Striated Muscle to relax it.
$E_{SSCEUSM}$:	ENS generated by the SSC and arrives at the Extrinsic Urethral Striated Muscle to contract it.
E_{SSCPFM} :	ENS generated by the SSC and arrives at the Pelvic Floor Muscle to contract it.

Table 1. Correct training pattern pairs.

The input and output signals of the model, as well as the interconnections between agents, change with time. In the storage phase, as urine enters into the bladder, intravesical pressure increases and pressure on the bladder wall is heightened. In order to retain

urine in this phase, as detrusor pressure increases so do the disabling micturition nerve signals. When the micturition process begins, clear co-ordination between the retaining elements (internal and external sphincter) and the emptying elements (detrusor) can be seen. This happens in such a way that the internal and external sphincter nerve signals are reduced almost to nil while the detrusor nerve signal increases considerably. This is the cause of the changes in pressure which lead to the expulsion of urine from the bladder to the urethra and from there to the outer.

The evolution of the main variables during the filling and emptying phases can be seen in figure 2. These phases allow us to determine five different states in the system, which are characterized by the fact that they are sequential and periodical.

State s'_0 represents a state of transition randomly produced by a disruption, such as a cough, sneeze, blow to the abdomen, etc.

States s_4 and s_5 are related, voluntary (and therefore, optional) and can be produced more than once. These states represent a refining of the model, for this reason, we will not deal with them in this paper.

In the following sections, we present a formal framework in which we analytically specify our agent-based model. In section 3, we add a heuristic to the model. In section 4, we present the results obtained from the experiments. Finally, in section 5, we offer conclusions and outline different areas of research in which we are currently involved.

2 Model

We formally define the neural regulator of the lower urinary tract using the tuple:

$$RLUT = \langle {}^{MS}I_R, NC \rangle \quad (1)$$

where ${}^{MS}I_R$ is the regulator interface with the MSLUT and NC is the set of neural centres.

Since we consider that the interface contemplates to the LUT like an actions and reactions system [8], we define it by:

$${}^{MS}I_R = \langle \Sigma, \Gamma, P, React \rangle \quad (2)$$

where Σ is the set of the possible system status, P is the set of possible actions that the different centres can perform on the system, Γ is the set of influences or action attempts and $React$ is the reaction function that carries out the different contributions that perform the neural centres like influences, expressing them in terms of the new state of the LUT.

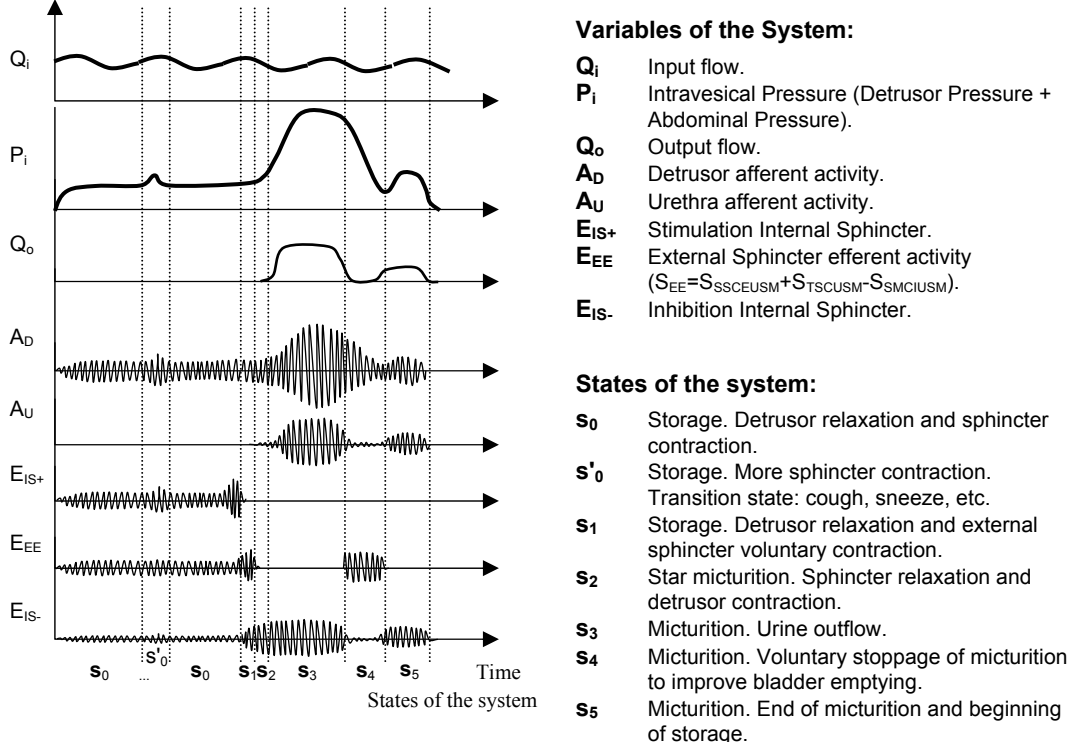


Fig. 2. Variables main, signals and states of the model.

The neural centres are modelling as PDE agents with memorizing and decision capacity [9]. In this way, we define a centre $\alpha \in NC$ by the tuple:

$$\alpha = \langle \Phi_\alpha, S_\alpha, Percept_\alpha, Mem_\alpha, Decision_\alpha, Exec_\alpha \rangle \quad (3)$$

where Φ_α corresponds to the set of perceptions, S_α corresponds to the set of internal status, $Percept_\alpha$ represents the perception function, Mem_α represents the memorizing function, $Decision_\alpha$ represents the decision function and $Exec_\alpha$ represents the execution function. We can appreciate the internal structure of a centre in figure 3.

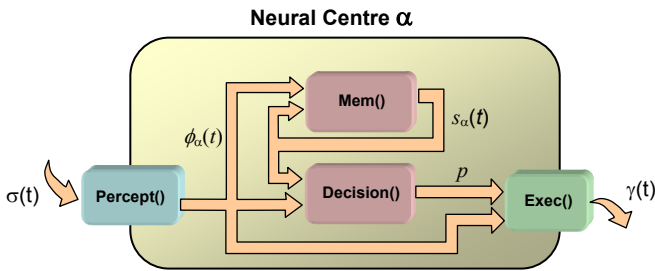


Fig. 3. Internal structure of the neural centre.

The execution of a particular task by a specific agent produces an influence on the overall state of the system. A change in the state of the system is determined by the combination of influences contributed by the simultaneous and concurrent execution carried out by the various agents of a set of

tasks. This combination is defined by means of the function

$$\bigcup^\Gamma: \Gamma^n \rightarrow \Gamma \quad (4)$$

Function React provides the current state of the system from the influences of the different agents:

$$React: \Sigma \times \Gamma \rightarrow \Sigma \quad (5)$$

The new state of the system is the result of having evaluated the influences contributed by different agents when concurrently performing their tasks:

$$\sigma(t+1) = React\left(\sigma(t), \bigcup^\Gamma(\gamma_1, \gamma_2, \dots, \gamma_n)\right) \quad (6)$$

The problem that may be encountered is that a particular component may be unable to contribute its influence or, because of a dysfunction in one of the nerve centres, the influence contributed is not correct. In order to solve this problem, we introduce a heuristic which is capable of rebuilding the appropriate influence vector from a vector without faults.

3 Heuristic

If we suppose that the heuristic function capable of contributing the correct influences is obtained from a set of influences contributed by the different agents:

$$H^\Gamma: \Sigma \times \Gamma \rightarrow \Gamma \quad (7)$$

Then, we will obtain the new state as:

$$\sigma(t+1)=\text{React}(\sigma(t),H^\Gamma(\sigma(t),\gamma(t))) \quad (8)$$

Heuristic H^Γ will allow the correct influence vector to be deduced from the influences contributed by the various nerve centres, whether they are correct or not. Although any classifier could be used as a heuristic, we decided to use a neural network because of the high degree of dispersion among the values of the various vectors and their corresponding states. This tool adapts well to the characteristics of this type of problem because of its high tolerance to faults and its great ease in modelling non-linear functions [10][11][12]. Of particular interest is the fact that the neural network used in our model is an Orthogonal Associative Memory (OAM) [13], whose main advantages are its great capacity for storage and its high noise tolerance.

The function to be evaluated by the neural network has as its input the vector of influences contributed by the agents together with the current state of the system. The output is the estimate of the correct influence vector:

$$\gamma' = H^\Gamma(\sigma, \gamma) \quad (9)$$

where σ represents the current state of the system, γ the input (influence vector) of the system agents corresponding to the various nerve centres and γ' the output (influence vector) which the system's output agents should have contributed, if all the nerve centres had functioned correctly.

The main function of the OAM is to establish associations between input patterns and intermediate auxiliary orthogonal vectors, with respect to their input patterns, and the associations of those with the system's output patterns. The OAM training phase involves obtaining two matrices of synaptic weights

$$\mathbf{W} = f_1(\mathbf{A}) \cdot f_2(\mathbf{Q}^T) \quad (10)$$

$$\mathbf{V} = f_2(\mathbf{Q}) \cdot f_1(\mathbf{B}^T) \quad (11)$$

where the matrix \mathbf{A} is the input patterns, matrix \mathbf{B} is the output patterns, the intermediate matrix \mathbf{Q} is an orthogonal Householder matrix [14], function $f_1()$ is a classic bipolar filter and function $f_2()$ is another bipolar filter which is dependent on the orthogonal matrix used. Recall of an unknown pattern a_i is obtained directly from the equation

$$b_i = f_1(f_2(f_1(a_i^T) \cdot \mathbf{W}) \cdot \mathbf{V}) \quad (12)$$

Two types of pattern were used to train the network. The first type is shown in table 2 and corresponds to all patterns composed of a state of the system plus a non-dysfunctioning influence vector which is valid for the said state of the system. That is, if, for example, we are in a current state of retention (s_0), the initiation of micturition (s_2) cannot be generated directly without having previously undergone a state of retention (s_1). The second type of patterns used in the training phase correspond to variations in the correct patterns to which various types of dysfunctions have been introduced, randomly altering the influence vector components. We have tested different patterns from training with several types of dysfunctions. In all the tests, the results are similar and even worse as we increased the number of dysfunctions.

	Input (a_i)			Output (b_i)				Input (a_i)			Output (b_i)		
1	s_0	γ_{s0}	γ_{s0}	6	s_0	γ_{s0}	γ_{s0}	11	s_1	γ_{s2}	γ_{s2}		
2	s_3	γ_{s0}	γ_{s0}	7	s_1	γ_{s1}	γ_{s1}	12	s_3	γ_{s2}	γ_{s2}		
3	s'_0	γ_{s0}	γ_{s0}	8	s_0	γ_{s1}	γ_{s1}	13	s_3	γ_{s3}	γ_{s3}		
4	s_1	γ_{s0}	γ_{s0}	9	s_2	γ_{s1}	γ_{s1}	14	s_2	γ_{s3}	γ_{s3}		
5	s'_0	γ_{s0}	γ_{s0}	10	s_2	γ_{s2}	γ_{s2}	15	s_0	γ_{s3}	γ_{s3}		

Table 2. Correct training pattern pairs.

In the next section, we present the results for the different training values and their validation obtained from the various unknown patterns bearing a number of dysfunctions.

4 Experiments

Two types of pattern were used to train the network. The first type corresponds to all patterns composed of a state of the system plus a non-dysfunctioning influence vector which is valid for the said state of the system. The second type of patterns used in the training phase correspond to patterns with several types of dysfunctions.

Before carrying out the experiments, we have performed a preliminary study of the influence of the dysfunctions number in the training patterns. We have obtained that the learning of patterns with more than two dysfunctions presents similar or even worse results.

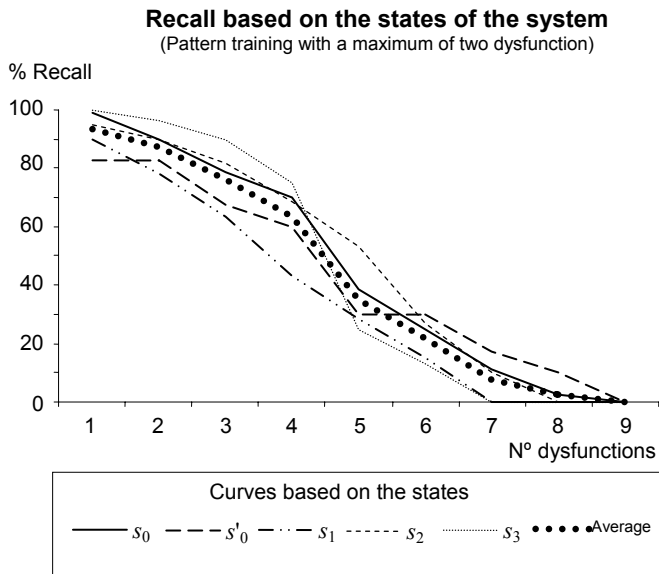


Fig. 4. Recall percentage based on the states of the system.

The following figures present a graph of the results corresponding to the network trained with patterns with a maximum of two dysfunctions. For this case, between a hundred and a thousand series of tests were performed on patterns with any type and number of dysfunctions, producing average values which are approximately the same for all of them.

Figure 4 shows the curves of evolution for the recall of patterns belonging to a certain state of the system as dysfunctions are introduced. It can be seen that all of these curves follow the same dynamic, with quite valid results even for patterns with four dysfunctions.

Figure 5 offers the same data but presented as dysfunction curves in the different possible states of the system. This time, although the results are perfectly acceptable, we can see a couple of alterations which should be commented on from a more qualitative approach.

In the graph in figure 5, it can be clearly seen that the curve corresponding to patterns with one dysfunction has a 70% success rate in the case 5 ($s_0\gamma s'_0$), where it had been expected that the network would recognize the vector $\gamma s'_0$. This case corresponds to a state of retention with an increase in pressure on the sphincter to absorb a disruption (cough, sneeze, blow to the abdomen, etc). However, the system originates from a state of retention and it is vital that, at least, it retains that state. When closely analyzing the vectors returned by the network at the said point when no recognition has occurred, we can see that for each case it was γs_0 . That is, the retention state is maintained and, therefore, it can qualitatively be considered as correct and perfectly viable.

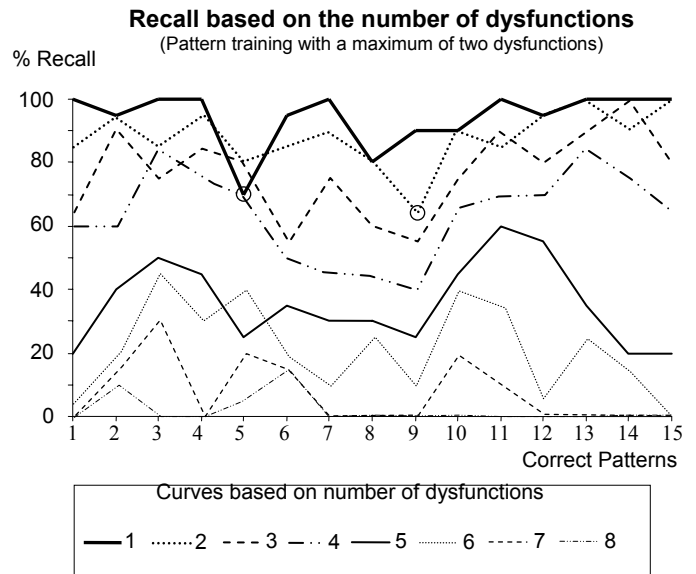


Fig. 5. Recall percentage based on the number of dysfunction.

In reference to figure 5 once again, it can be observed that the curve corresponding to two dysfunctions also presents a very low success rate, 65% for the case 9 ($s_2\gamma s_1$). When analyzing the said point, we can see that it corresponds to state s_2 , where an initiation of micturition process was underway. However, the system turns back to a voluntary retention state s_1 . Upon closely examining the values returned by the network, we can see that in most cases where the pattern γs_1 is not recognized, the vector γs_2 is returned. This implies that the system will find it difficult to detain the micturition process once it has begun, and this circumstance will follow the biological process. We can therefore consider these results as valid.

4 Conclusion

Our model approaches the analysis of the RLUT as a distributed system. This has allowed the general problem to be divided into much smaller and easier to approach sub-problems, a task which had been too complex to carry out until now. This achievement is especially valid if we take into account the fact that they are not highly connected modules. Furthermore, basic interaction between them is quite basic and tasks are very specific because they correspond directly to the various nerve centres.

This distributed approach to the problem has allowed us to define a model which is capable of individually controlling each of the RLUT nerve centres. This feature, together with the possibility of dynamically correcting dysfunctions in up to even four nerve centres at once, allows the system to take

dynamic control of those dysfunctioning centres, temporarily correcting and performing its activity. Although at first it may seem that the ability to detect only four dysfunctions is a restriction, in practice it is unlikely that so many dysfunctions would appear at once. Therefore, the system can gradually absorb them and even take total control of the RLUT if such a high number of dysfunctions were detected that the system could not differentiate them.

This paper has showed how the paradigm of agents adapt to the design of our system and opens the way towards a generalization of the model for use in other biological or non-linear control processes and how OAM supplies a better model performance.

The next stage will be to enable the different agents to dynamically learn the constants of the system from their experience with the patient, and subsequently use them to rectify more complex dysfunctions, improve the heuristic or predict the way in which each patient's particular system functions.

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