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APPLICATION OF CONTROL FAULT DETECTION IDEAS TO ARTERIAL HYPERTENSION DIAGNOSIS

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ABSTRACT

In this paper, the feasibility of transferring control engineering fault detection ideas to the problem of cause identification of arterial hypertension in humans,is investigated.Specifically,two methods are considered: a model-based parameter estimation technique and an artificial neural network approach (ANN).

The mathematical model used for the simulation of the human arterial pressure control system,the proposed malfunction detection and identification methods,together with their merits and shortcomings, are described in details.

1. Introduction

The fields of engineering and medicine have been traditionally involved in exchanging ideas and knowledge information aiming at improving the effectiveness of methods in both areas. Some late results from the field of artificial neural networks in control engineering are based on findings from the molecular workings of human neurons while progress in microelectronics and computing science have greatly enhance the diagnostic power of contemporary medicine. These are only two of the many examples that can be cited in order to substantiate our opening statement.

In this work we are investigating the feasibility of transferring control engineering fault detection ideas to the problem of cause identification of arterial hypertension in humans. Specifically, we are considering two such methods: a model-based parameter estimation technique and an artificial neural network approach (ANN).

The idea of applying control fault detection ideas to the identification of malfunctions of the human system is not new. Gustafson and his research team (1978a, b) have investigated the detection and classification of cardiac arrythmias using generalised likelihood ratio (GLR) techniques. In this approach a set of phenomenological models for both persistent and transient rhythms was developed, in order to match observed statistical variations. Arrythmias were identified by calculating statistical probabilities and likelihoods associated with these models, based on R-R interval data. Doerschuk and co-workers (1986) have extended this approach by implementing a distributed, hybrid model, i.e. one involving both continuous and discrete phenomena. In this framework, formulation of models and estimation problems that capture all aspects of rhythm analysis was possible.

More recently Frangakis et. al (1990) have applied an expert system approach to the problem of blood pressure (BP) signal diagnosis. In this method, rules that correlate the BP pattern shape with diseases in the circulation system were formulated in terms of a set of primitive patterns which constituted the expert system knowledge base. The expert system module was interfaced with a signal analysis module, responsible for BP signal acquisition and pattern recognition. Apart from the "normal" state, three abnormal conditions were detected in this manner: aortic stenosis, arteriosclerosis and aortic insufficiency.

The desire to understand more fully the mechanisms of human arterial pressure regulation so as to aid the diagnosis of its malfunctions, mainly chronic hypertension, has stimulated this work. In the next sections we describe the mathematical model used for the simulation of the human arterial pressure control system, the proposed malfunction detection and identification methods, together with their merits and shortcomings.

2. Mathematical model of arterial pressure regulation.

The development of a precise mathematical model of the monitored process, is more or less required for either of the proposed fault detection and identification (FDI) methods described later.

The systems analysis of circulatory regulation adopted in this work is based on the research of the team around Guyton (1980) and later Coleman (1985). This work has led to the development by Montani et al. (1989) of MODSIM, a simulation tool for studying very large dynamic models of the human system. Version 1.0 of this system, written in C, is currently under evaluation for the needs of present research.

To effectively model the human circulatory system, one must know the various parts of the body that affect arterial pressure and their interrelations. This would in fact lead to a model of the whole human organism. Therefore different levels of complexity must be examined, starting from the very simple and going to the more difficult. Figure 1 shows such a simple hemodynamic model while Figure 2 is its block diagram equivalent. All the pressures are represented by the letter *P*; the capacitances, *C*; volumes, *V*; resistances, *R*; flows, *F* and heart strength, *Hs*. Subscripts denote arteries, *a*; capillaries, *c*, veins, *v* and right atrium *ra*.

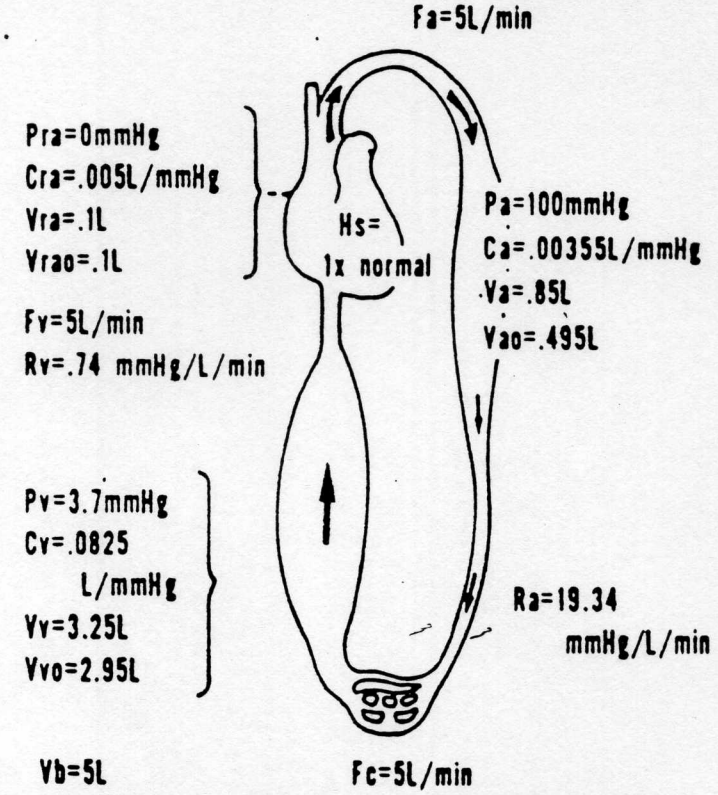


Figure 1. A simple hemodynamic model of the circulation

In this model, heart pumping effectiveness is measured by the cardiac output curve as illustrated in Figure 3 for normal heart strength (*Hs*=1). The basic physical equations used to build this model are the following:

$$\text{flow} = \frac{\text{pressure difference}}{\text{resistance}}$$

$$\text{rate of change of blood volume} = \text{flow difference}$$

$$\text{pressure} = \frac{\text{excess blood volume}}{\text{capacitance}}$$

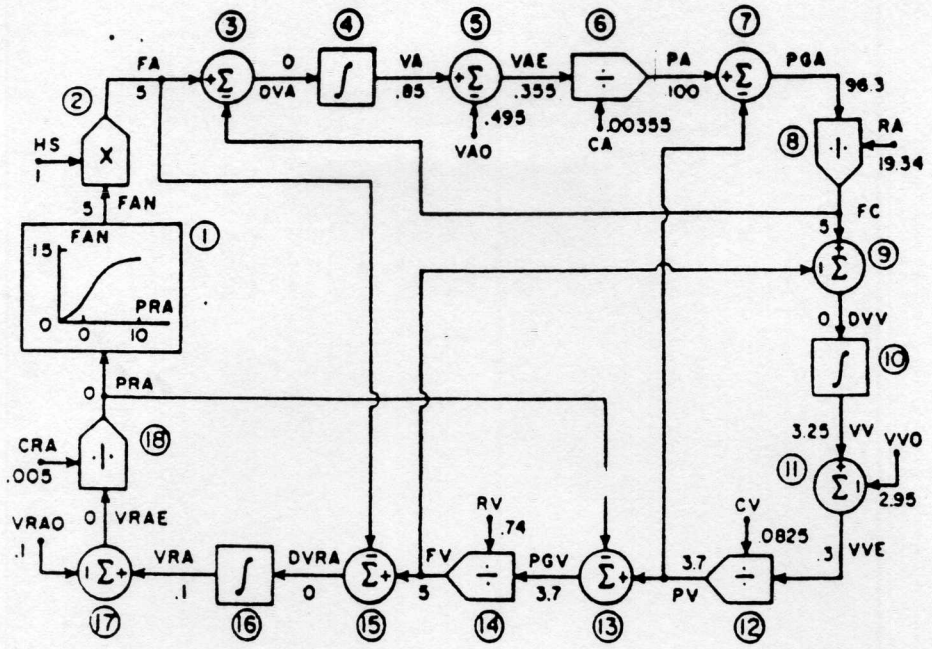


Figure 2. Block diagram of the simple hemodynamic model

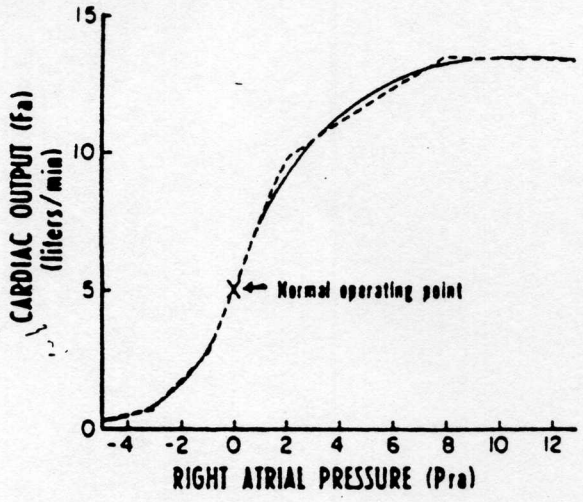
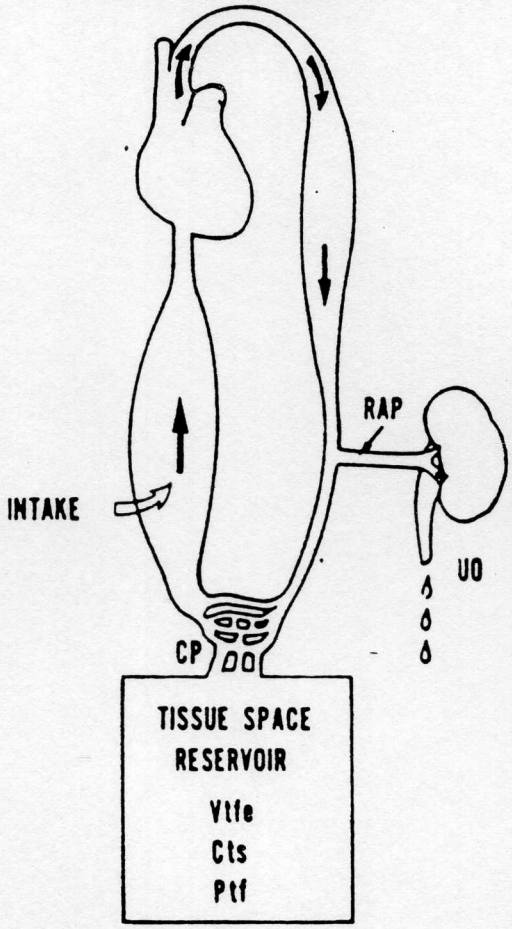


Figure 3. The normal cardiac output curve (*Hs*=1)

This simple model is in fact a closed hydrodynamic system that has no means for regulating the blood volume, which remains constant. Therefore, regulation of arterial pressure is limited. However, it can be used to test the applicability of the proposed FDI methods.

In a second level of difficulty, the model of Figure 4 is used (Figure 5 is its block diagram equivalent, only relevant blocks used). In this model, two essentials for blood volume control have been added: (1) the fluid intake-renal output system, and (2) the tissue space fluid reservoir system, which can interchange interstitial fluid with the circulating blood.

The Intake-Output system: This is composed of the daily intake of water and electrolytes on one hand, and the daily output of water and electrolytes on the other. Only losses through the kidneys are considered, since the rest do not vary with arterial pressure changes. The kidney portion of the intake-output system however, is highly responsive to the arterial pressure as illustrated in Figure 6.



RAP: renal arterial pressure, UO: urinary output, Pc: capillary pressure, Vtfe: excess fluid in interstitial space, Cts: capacitance of tissue space, Ptf:tissue and colloid osmotic pressures of capillary blood and tissue fluids

Figure 4. The basic circulation with intake-output and tissue space reservoir systems.

The marked increase in urinary output as the arterial pressure rises is the basis of a negative feedback mechanism for control of arterial pressure. This mechanism is extremely powerful for long-term control of arterial pressure. Another important characteristic of the renal function curve is the way by which its characteristics change. This can be done in either of two ways:

1. If the intake level is changed, thus changing the set-point of the feedback control system to a new point defined by the intersection of the renal function curve and the intake level line.
2. If the chronic renal function curve itself is changed, which can be the result of many different mechanisms. The curve may be shifted to the left or right of the normal position or can change its slope or shape.

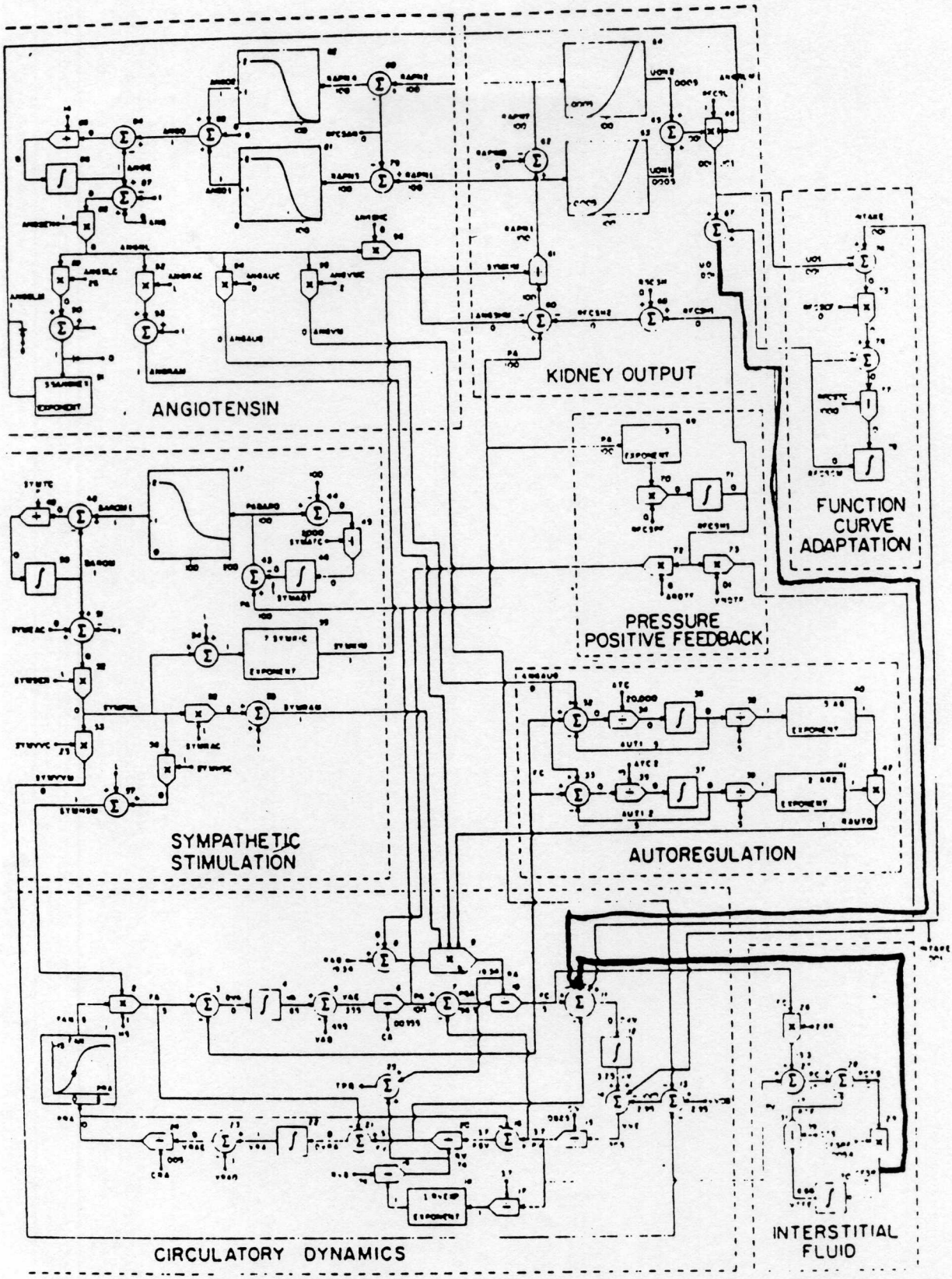


Figure 5. Block diagram of basic circulation with intake-output and tissue space reservoir systems.

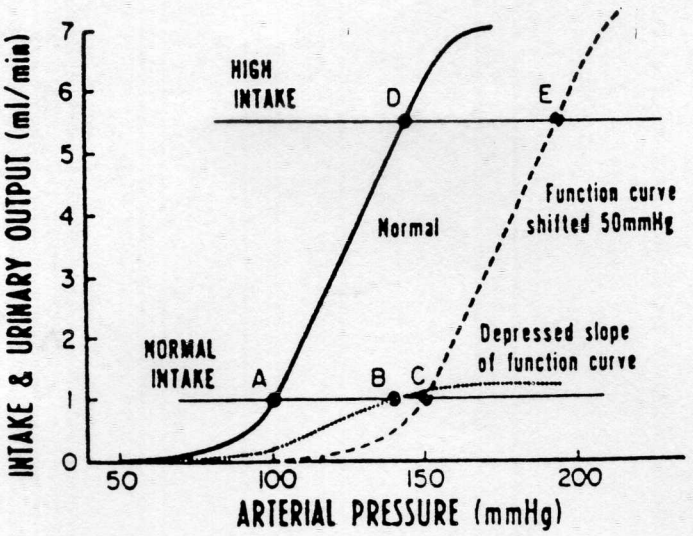


Figure 6. A pressure analysis diagram showing the different arterial pressure levels that are predicted for different combinations of intake level and renal function curves.

The tissue space fluid reservoir system: The filling of this reservoir is in balance with the filling of the circulatory system. When the capillary pressure becomes too high, fluid transudes into the tissue space and vice versa. However, the tissue space reservoir does not have infinite capacity: is capable of "buffering" the blood volume by a factor of 5 to 10. That is, if blood is lost from the system, the tissue space reservoir will, within a few miniutes to few hours, automatically replenish between 5/6 and 10/11 of the lost blood volume.

Analysis of this system shows that changes in the total peripheral resistance, the circulatory capacitance or the heart strength are all unable to change the long-term level of arterial pressure. This is only possible if either the intake or the quantitative characteristics of the renal function curve are changed.

If further complexity is required, the following blocks can be added to the basic system:

Autoregulation: Autoregulation is the process whereby each local tissue of the body controls its own blood flow according to the needs of that tissue. The simulated mechanism of the autoregulation process is roughly the following: whenever the cardiac output becomes different from the normal level, it sets into motion a progreesive change in total peripheral resistance, until cardiac output reapproaches normal level. The degree to which it reapproaches the normal level is expressed by the gain of the autoregulatory feedback mechanism, and the time required for it to approach the new steady-state level is expressed by its time constant. One of the very important factors of the autoregulatory mehanism is to compensate for the arterial pressure-dilatation phenomenon.

The renin-angiotensin system: The basic actions of angiotensin that are known to be important in blood pressure control are the following:

1. Angiotensin has a potent direct effect to increase arteriolar resistance in all or almost all areas of the body, thus increasing total peripheral resistance.
2. Angiotensin has a moderate effect to decrease the overall vascular capacitance. Though angiotensin mainly causes arteriolar constriction, it does cause a mild to moderate degree of constriction of the veins as well.
3. Angiotensin causes the kidneys to retain both salt and water.

Studies have shown that only the third type of effect causes chronic hypertension.

Nervous mechanisms: The nervous mechanisms of pressure control fall into two separate categories:

1. Pressure controlling reflexes, and
2. Non-reflex signals that are transmitted from the central nervous system to the vasculature and other organs and that eventually affect blood pressure regulation.

The most important pressure controlling reflex is the *baroreceptor reflex.* The baroreceptor reflex damps acute changes in arterial pressure. However it does not affect the long-term level to which the arterial pressure is controlled because the baroreceptors adapt after several days to the new pressure level. There are reasons to believe that all or most of the other blood pressure controlling reflexes (peripheral chemoreceptors, receptors of the vasomotor center, abdominal compression reflex, volume reflex, cardiopulmonary pressure reflex) also adapt and therefore do not affect the long-term arterial pressure level.

The previous paragraphs outline the basic factors the affect arterial pressure control. Yet many other factors also have various degrees of effects on arterial pressure control. These include the aldosterone system, the ADH system, the thirst mechanism, the salt appetite mechanism, the concentrations of sodium and potassium in the circulating blood, the vascular stress-relaxation system, the red blood cell production system, capillary membrane dynamics, the compliance of the interstitial space, the composition of the interstitial fluid, the long-term control of heart muscle mass and other factors. A block diagram incorporating all those factors is depicted in Figure 7.

3. Fault detection via parameter estimation

Fault detection via parameter estimation relies on the principle that possible faults in the monitored process can be associated with specific parameters and states of a mathematical model of a process given in general by an input-output relation,

$$y(t) = f(u, e, \theta, x) \tag{1}$$

where $y(t)$ represents the vector output of the process, $u(t)$ the vector input, $x(t)$ the partially measurable state variables, θ the nonmeasurable process parameters likely to change and $e(t)$ unmodeled or noise terms affecting the process. It is obvious therefore, that it is necessary to have a good theoretical dynamic model of the process in order to apply parameter estimation methods. This is usually derived from the basic balance equations for mass, energy, and momentum, the physico-chemical state equations and the phenomenological laws for any irreversible phenomena.

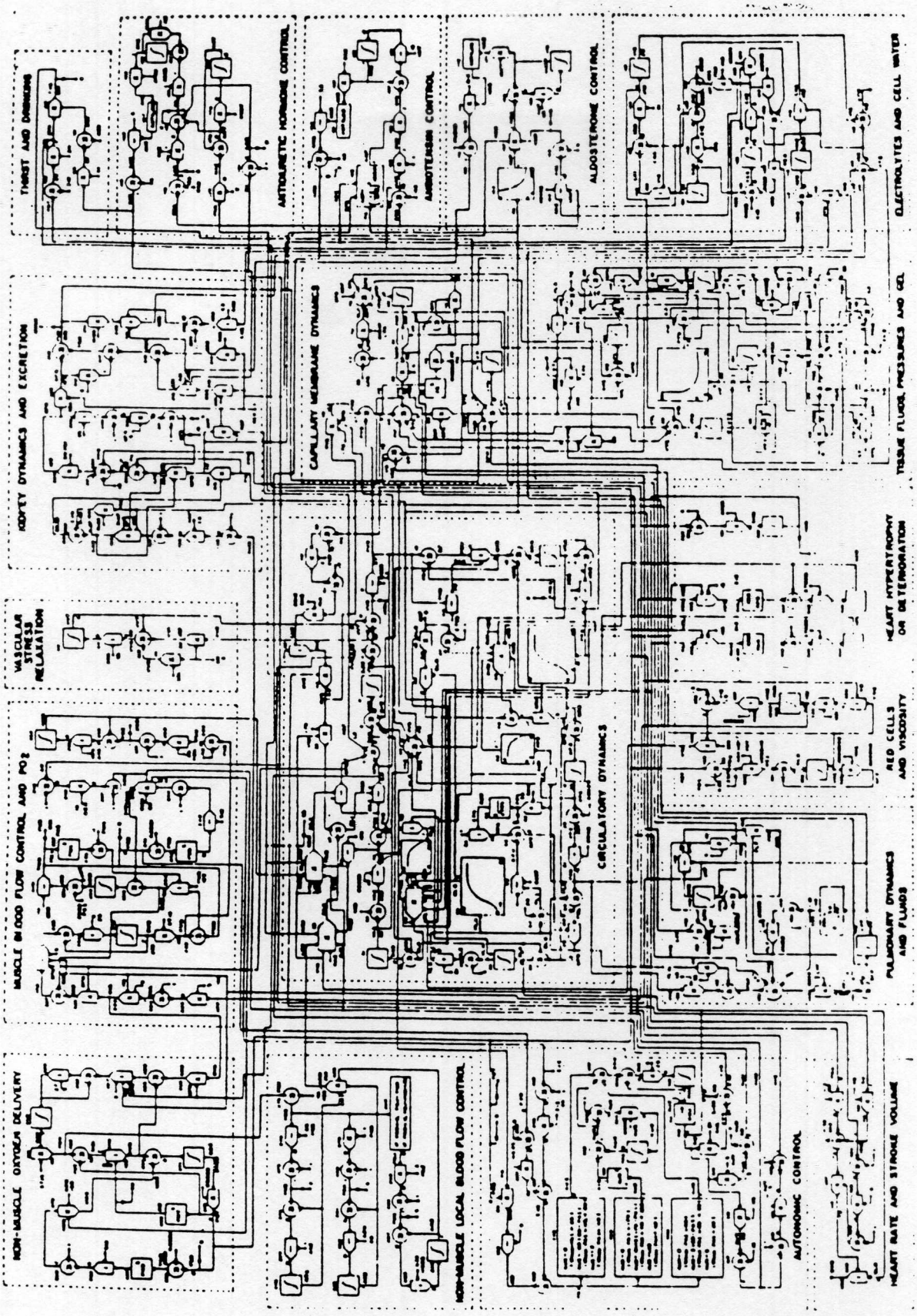


Figure 7. A complex, integrated system for arterial pressure control.

The models will then appear in the continuous or discrete time domain, in the form of ordinary or partial differential or difference equations. Their parameters θ_i are expressed in dependence on process coefficients p_j, like storage or resistance quantities, whose changes indicate a process fault. Hence, the parameters θ_i of continuous time models have to be estimated. As an example consider a simple dynamic process model with lumped parameters, linearized about an operating point, which may be described by the differential equation,

$$y(t) + \ldots + a_n y^{(n)}(t) = b_0 u(t) + b_1 u^{(1)}(t) + \ldots + b_m u^{(m)}(t) \qquad (2)$$

The process model parameters,

$$\theta^T = [\, a_1, \ldots, a_n \mid b_0, \ldots, b_m \,] \qquad (3)$$

are defined as relationships of several physical process coefficients, e.g. length, mass, speed, drag coefficient, viscosity, resistances, capacities. Faults which become noticeable in these physical process constants are therefore also expressed in the process model parameters. If the physical process coefficients, indicative of process faults, are not directly measurable, an attempt can be made to detect their changes via the changes in the process model parameters θ. The following procedure is therefore applicable in general:

(1) Establishment of the mathematical model of the normal process,

$$y(t) = f(u(t), \theta) \qquad (4)$$

mainly from theoretical considerations. At this stage allowable tolerances for process coefficient values are also defined.

(2) Determination of the relationship between the model parameters θ_i and the physical process coefficients p_j,

$$\theta = f(p) \qquad (5)$$

(3) Estimation of the model parameters θ_i from measurements of $y(t)$, $u(t)$, by a suitable estimation procedure,

$$\hat{\theta}(t) = g(y(1),\ldots,y(t),u(1),\ldots,u(t)) \qquad (6)$$

(4) Calculation of process coefficients, via the inverse relationship,

$$\hat{p}(t) = f^{-1}(\hat{\theta}(t)) \qquad (7)$$

(5) Decision on whether a fault has occurred, based either on the changes Δp_j calculated in step 4 or on the changes $\Delta\theta_i$ and tolerance limits from step 1. If decisions are made based on the $\Delta\theta_i$ the affected p_i's can be easily determined from (5). This may be achieved with the aid of a fault catalogue in which the relationship between process faults and changes in the coefficients Δp_j has been established. Decisions can be made either by simply checking against the predetermined threshold levels, or by using more sophisticated methods from the fields of statistical decision theory. A fault decision should include the fault location, fault size and time of occurrence.

The basis of this class of methods is the combination of theoretical modeling and parameter estimation of continuous time models. Since, however a requirement of

this procedure is the existence of the inverse relationship (7) it may be restricted to well-defined process.

The implementation of the full procedure requires considerable effort in modeling the process, more sophisticated and fault-sensitive identification methods and fast processing hardware suitable for on-line operation.

In order to apply the aforementioned ideas to the problem of malfunction detection and diagnosis of arterial hypertension, a mathematical model of the arterial control system in the form of equation (2) must be built. Considering the analysis of the previous section, this should not prove to be a difficult task. For example, the system of figure 2 is described by the following system of first order non-homogeneous differential equations,

$$\begin{bmatrix}\dot{V}_A\\ \dot{V}_V\end{bmatrix}=\begin{bmatrix}-\frac{1}{C_AR_A} & \frac{1}{C_VR_A}\\ \frac{1}{C_AR_A}-\frac{1}{C_{RA}R_V} & -\frac{1}{C_VR_A}-\frac{1}{C_VR_V}-\frac{1}{C_{RA}R_V}\end{bmatrix}\begin{bmatrix}V_A\\ V_V\end{bmatrix}+\begin{bmatrix}-\frac{V_{V0}}{C_VR_A}+\frac{V_{A0}}{C_AR_A}+f_A(t)\\ \frac{1}{R_A}\left(\frac{V_{V0}}{C_V}-\frac{V_{A0}}{C_A}\right)+\frac{1}{R_V}\left(\frac{V_{V0}}{C_V}-\frac{V_{RA0}}{C_{RA}}+\frac{V}{C_{RA}}\right)\end{bmatrix}$$

where $f_{A(t)}$ is the cardiac output function of figure 3. Zero subscripts denote unstressed volumes, while V is the total instantaneous volume of the circulatory system. This system can be put in the form of equation (2), if it is rewritten as,

$$\dot{V}_A=\begin{bmatrix}V_A & V_V & 1\end{bmatrix}\begin{bmatrix}-\frac{1}{C_AR_A}\\ \frac{1}{C_VR_A}\\ -\frac{V_{V0}}{C_VR_A}+\frac{V_{A0}}{C_AR_A}+f_A(t)\end{bmatrix}$$

$$\dot{V}_V=\begin{bmatrix}V_A & V_V & 1\end{bmatrix}\begin{bmatrix}\frac{1}{C_AR_A}-\frac{1}{C_{RA}R_V} & -\frac{1}{C_VR_A}-\frac{1}{C_VR_V}-\frac{1}{C_{RA}R_V}\\ \frac{1}{R_A}\left(\frac{V_{V0}}{C_V}-\frac{V_{A0}}{C_A}\right)+\frac{1}{R_V}\left(\frac{V_{V0}}{C_V}-\frac{V_{RA0}}{C_{RA}}+\frac{V}{C_{RA}}\right)\end{bmatrix}$$

Hence, the following parameters can be estimated by observing V_A, V_V:

$$\begin{bmatrix}\theta_1\\ \theta_2\\ \theta_3\end{bmatrix} = \begin{bmatrix} -\dfrac{1}{C_A R_A} \\ \dfrac{1}{C_V R_A} \\ -\dfrac{V_{V0}}{C_V R_A} + \dfrac{V_{A0}}{C_A R_A} + f_A(t)\end{bmatrix}$$

$$\begin{bmatrix}\theta_4\\ \theta_5\\ \theta_6\end{bmatrix} = \begin{bmatrix} \dfrac{1}{C_A R_A} - \dfrac{1}{C_{RA} R_V} \\ -\dfrac{1}{C_V R_A} - \dfrac{1}{C_V R_V} - \dfrac{1}{C_{RA} R_V} \\ \dfrac{1}{R_A}\left(\dfrac{V_{V0}}{C_V} - \dfrac{V_{A0}}{C_A}\right) + \dfrac{1}{R_V}\left(\dfrac{V_{V0}}{C_V} - \dfrac{V_{RA0}}{C_{RA}} + \dfrac{V}{C_{RA}}\right)\end{bmatrix}$$

Note, that even though volumes are used in the above equations, they can be easily calculated from measured pressures.

The equations for the more complicated versions can be obtained in a similar manner, if one works with the block diagram of figure 4.

It can be seen therefore, that, at least in theory, faults in the circulatory system can be detected if an adequate history of pressures is available. A difficulty that may arise is the requirement of many identification methods, ie. that the system be persistently excited.

4. Neural network approach to hypertension diagnosis

Artificial Neural Networks (ANN) are a kind of nonlinear net systems composed of a large number of interconnected simple computing elements. Neural networks can autonomously store knowledge by learning from historical information and have the associative memory ability. Various researchers have demonstrated the ability of neural networks to diagnose faults in dynamical processes. Hoskins et al. (1988) applied ANN's to the diagnosis of a simple chemical processes composed of three continuous stirred tank reactors in series. Watanabe et al. (1989) presented a two-stage multilayer neural network to diagnose faults in a PI-controlled process. Venkatasubramanian and Chan (1989) developed an ANN to diagnose the faults of a fluidised catalytic cracking process.

In this section we propose the use of the perceptron network and the back-propagation algorithm for the diagnosis of hypertension conditions. The proposed methodology treats fault diagnosis as a classification problem. Similar to classification theory, neural networks perform the classification by creating decision boundaries to separate the different pattern classes. However, unlike traditional classifiers, when a classification is realised with neural networks, the whole mapping from sample space into decision space is done at the same time. The knowledge of the fault diagnosis is stored distributely in the highly interconnected neuron-like elements. Morover, it is these internal representations

which lead to the associative memory and generalisation abilities exhibited by these structures.

The single-layer perceptron network: The *perceptron* was first presented by F. Rosenblatt in 1957. It is a simple single computation layer neural network with an input and output layer, as shown in figure 8.

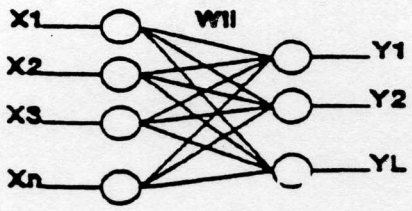


Figure 8. Single-layer perceptron network

The outputs of the network are calculated from the formula:

$$Y_j = \mathrm{f}(\sum_{i=1}^{N} W_{ji} X_i - \theta_j),\ \mathrm{j} = 1,\ 2,\ \ldots,\ 1$$

where X_i is the input to the input layer node i, Y_j the output of the output layer node j, W_{ji} the weight of the connection from the output node j to the input node i, θ_j the bias of the output node j and f is a nonlinear function, usually a hard limiter, threshold logic or sigmoid function.

For a group of M training input-output pattern samples X_{p1}, X_{p2}, ..., X_{pN}, T_{p1}, T_{p2}, ..., T_{pl}, p=1, .,, M, the mean square error over all training patterns between the actual outputs Y_{pj} and the desired ones is,

$$E = \frac{1}{2M} \sum_{p=1}^{M} \sum_{j=1}^{L} (T_{pj} - Y_{pj})^2$$

The process of network training or learning is nothing more than a process of minimising the error E by adjusting the weights by a suitable algorithm such as the *delta rule* [Pao, 1989]. This rule adjusts the weights according to,

$$W_{ji}(k+1) = W_{ji}(k) + \eta \delta_j X_i + \alpha [W_{ji}(k) - W_{ji}(k-1)]$$

$$\delta_j = (T_j - Y_j) f'(\sum_{i=1}^{N} (W_{ji} X_j + \theta_j)$$

where k denotes the iteration step during training, η ($0<\eta<1$) is the learning rate or the gain coefficient which provides the step size during weight adjustment and α ($0<\alpha<1$) is the coefficient of the momentum term, which assures rapid convergence with large step sizes without oscillations.

In a similar way multilayer perceptrons are trained using the *generalised delta rule* of Rumelhart et al. (1986).

The process of fault diagnosis is also a process of pattern recognition. In the traditional pattern recognition technique, the pattern classification is carried out through a series of decision functions. For a classification of N-dimension pattern space with M clusters, it may be posed as a problem of how to define hyperplanes to divide the N-dimension Euclidean space into M decision regions. In a similar fashion, neural networks can perform the classification by creating decision boundaries to separate the different pattern classes. In this way, the neural network performs fault detection and isolation in a transparent way. In a highly nonlinear system, such as the human circulation, analytic development of relevant algorithms is almost impossible. Neural networks however are capable of performing these functions. The whole procedure is as follows:

1. Development of circulation system simulator. This is essential, since it is not possible or sometimes ethical, to simulate faults on a living organism. Results of the previous section can be used here.
2. Selection of representative fault situations for study. These may be increased arterial resistance, renal function abnormalities, angiotensin secretion irregularities etc. Let these faults denoted by f_i. For each of these faults an output pattern vector is selected usually an ith unit vector as shown in Table 1.

FAULTS	OUTPUT VECTORS
f_1	1 0 0 0 ... 0
f_2	0 1 0 0 ... 0
.....	.....
f_N	0 0 0 0 ... 1

Table 1. Output training vectors

3. Specification of training parameters, η, α, E_{min} and structure of network (number of layers, number of nodes per layer). Usually a single-layer perceptron is used for a start.
4. Training of the network using input test patterns and output test patterns until $E<E_{min}$.
5. Following the training, verification of the proposed structure is carried out by presenting the network with samples not contained in the training. Results are used to decide whether fault classisification is correct in all cases.

6. Conclusions

We have presented two methodologies for malfunction diagnosis in human arterial pressure regulation. This is a very difficult problem since on one hand this a very complicated non-linear dynamical system and on the other the causes of chronic hyperension are not agreed upon by all researchers in the field. We believe that the merging of well established control fault detection ideas in this field, will

produce good results and will help in the understanding of hypertension development.

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