Role of Coronary Endoarterial Blocking Devices in Myocardial Preconditioning

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Abstract

Introduction

Healthy physiologic function is characterized by a complex interaction of multiple control mechanisms that enable an individual to adapt to the exigencies and unpredictable changes of everyday life [15]. As implied by its name, non-linear dynamics studies systems, such those in physiology, in which output is not proportional to input [6]. Physiologic processes show highly variable fluctuations resembling "chaos" [24, 31]. The term "chaos" describes an apparently unpredictable behaviour that may arise from the internal feed-back loops of certain non-linear systems [13, 16].

This healthy variability is not just random, uncontrolled fluctuation, however. Although the precise mathematical definition of chaos is some-what complicated, its presence in the body can be characterized by two things: first, it is there by design, for instance it is not caused by the random firing of neurons; second, the behaviour of a chaotic system is complicated and unpredictable [22].

As a matter of facts, vasomotility and vasomotion of every tissue-microvascular-unit physiologically show an highly complex type of variability, "constrained randomness", reminescent of chaos [12,17], which may be fortunately evaluated nowadays at the bed-side with the aid of biophysical-semiotics, as I demonstrated previously, for the the first time clinically [24, 31]. In addition, ischaemic preconditioning [17, 31, 35-39] indicates that during ischaemic episode healthy myocardium, as well as other organs, such as kidney, liver, lung, brain, pancreas, skeletal muscle, a.s.o., can develop a protective adaptation towards successive ischaemic insults by means of favorable microcirculatory modifications and complex biochemical processes, resulting in ventricular size changes, evaluated with the aid of biophysical semiotics in reliable and refined manners, some of them easy to perform. Interestingly, from the etiopathogenetic viewpoint, alterations of EBD are strictly related to those of preconditioning (See later on).

Finally, myocardial oxygen supply can be assessed clinically in a precise way, illustrated in following, as I referred exhaustively for the first time in a previous article [25].

The aim of this conference is to illustrate some original methods of Biophysical Semeiotics [25], utilizing the above-mentioned parameters, useful and reliable in bedside detecting coronary artery ischaemic disorder, even clinically silent, since its very initial stage, i.e., CAD real risk.

Methods

Biophysical Semeiotics and its practical applications have been elsewhere fully described [24, 25, 26, 33, 35-39]. See also http://www.semeioticabiofisica.it. As regards the evaluation of biophysical-semeiotic signs, used in my research, i.e. aspecific gastric-, caecal-, and ureteral-reflexes, beside some technical knowledges are indeed necessary (Figures 1, 2, 3, 4, 5).
Figure 1: Figure clearly shows the right position of the conical bell of a stethoscope and lines (arrows), along which finger percussion must be applied, directly and gently, carrying out auscultatory percussion of the stomach and caecum. Moreover, aspecific gastric- and caecum- reflex are characterized by stomach and respectively caecum dilation.

Figure 2: Upper, middle, and lower ureteral reflexes are shown in the figure.
Figure 3: Auscultatory Percussion of the heart

Figure 4: Physiology Fluctuations of upper and lower ureteral reflexes, caused by digital pressure of different (low, mean, moderate, intense, s.o.) intensity applied upon cutaneous projection area of healthy subject’s biological systems, psycho-physically relaxed with open eyes (= melatonin secretion inhibition), in supine position, evaluated in a synergetic model, parallel those of diverse microvessel structures: upper ureteral reflex is related to small arteries and arterioles, according to Hammersen; middle ureteral reflexes are correlated with Endoarterial Blocking Devices both physiological type II (low-intense stimulation), type I moderate-intense stimulation), and newborn-pathological (moderate-intense – type I, subtype b) – and intense – type I, subtype a) – stimulation; finally low ureteral reflex oscillations give information on nutritional capillaries. Interestingly, mean digital pressure upon Th-1 – Th-2 dermatomeres stimulates cardiac β-adreno-receptors. Physicians assess the capillary diameter as intensity of low ureteral reflex. Highest spike (HS) intensity divided for minimal oscillation is ratio 3/1 under physiological condition. This value is unavoidable in calculating biophysical-semiotic fractal Dimension (fD) of microvascular deterministic chaotic system. It is perfectly identical to the value of differential latency time of heart-aspecific gastric and –caecum-reflex, surely easier to be evaluated. Interestingly, the period of oscillations is not fixed or constant: under physiological condition, it varies from 9 sec. to 12 sec. showing 6 cicles per minute. The average duration of fluctuations is, therefore, 10.5, i.e., a fractal number. Finally, the intensity of “normal” oscillation is variable in an unpredictable manner, varying in health from 0.5 cm. to 1.5 cm (HS).
In the present conference, a lot of biophysical-semeiotic parameters have been evaluated, which demonstrate the strict relation between normal and pathological EBD and myocardial preconditioning. First of all, it is important to underscore that, in health, coronary microvessels are only equipped with physiological, type II, EBD. Therefore, the simple presence of newborn-pathological type I EBD, not dependent of their precise, real nature, indicates coronary disease real risk.

1. Deterministic chaos of myocardial vasomotility and vasomotion

In a supine healthy subject, psycho-physically relaxed, with his (her) open eyes, aiming to inhibit melatonin secretion, digital pressure of "low-mean" intensity, applied upon the skin projection area of heart, brings about upper, middle, low-ureteral-, gastric aspecific-, caecal-, and choledocic- reflexes, i.e., upper-, mean, low-ureter as well as stomach, caecum, and choledocus dilate, the latter three after a latency time of 8 sec.

In health, the dilation of upper and low ureteral reflexes, happen after 6 sec. and lasts 6 sec., while all other reflex duration is less than 4 sec. The latter parameter value proved to be of paramount importance, from diagnostic viewpoint, informing precisely about local microvascular structures and function, as well as microvessel remodelling (Figures 3, 4).

In fact, such as digital pressure brings about "low-mean" stimulation of coronary trigger-points, inducing "rapidly" oscillations of upper and choledocic reflexes (= small arteries, according to Hammersen) and subsequently those of lower ureteral (= nutritional capillaries), which parallel fluctuations of the related microvessel structure, according to synergetic model. In addition, more intense stimulation provokes numerous, pressure-dependent, middle ureteral reflexes, informing respectively on various type EBD, type A, group I, and II AVA, and type B, group I and group II AVA, according to Bucciante (Table 1).
Interestingly, in health, the intensity of these reflexes – that’s their diameter – appears oscillating
from 0.5 cm. to 1.5 cm. at rest, in unpredictable manner, showing periods, which last about 10.5 sec.
(fractal number), varying from 9 sec. to 12 sec., (6 cycles per minute). Physiologically, after two
normal, different in intensity, unpredictable fluctuations, we observe an highest oscillation - highest
spike (HS) – that corresponds to “quantic”, maximal, periodic adrenaline and nor-adrenaline discharge
from autonomic nervous system endings, which occurs exactly every 25 sec., as I demonstrated
earlier [31, 24, 25] (Figures 4, 5). Finally, these signs can usefully be evaluated under stress tests.

To summarize, Biophysical-Semeiotics allows doctor to detect the chaotic behaviour of both intensity
and period of ureteral (and choledocic) oscillations, i.e. vasomotility (= upper ureteral reflex: small
arteries) and vasomotion (= low ureteral reflex: nutritional capillaries) of the microcirculatory bed of
all organ and tissue, including the heart (Figures 3, 4, 5).

From biophysical semeiotic point of view, it is useful and easy to calculate the so-called fractal
dimension (D) of microvascular chaotic system: in 120 sec. we observe 4 HS, that divide the space in
4 segments; each segment is subdivided in 3 tracts by two normal oscillations. It is, therefore,
possible to calculate the fractal dimension, which, roughly speaking, indicates how much space a
figure takes up, i.e. the degree of chaos, and is a measure of the complexity of the figure [16]:

\[ r = N - (1/D) \quad \text{when} \quad r = 3 \quad N = 4 \]

\[ \log_4 n \cdot 4 / \log_4 3 \quad [14] \times F', \text{fractal factor} \]

From the biophysical semeiotics viewpoint, fractal factor – f – corresponds to the ratio HS/minimal
oscillation. In health, for example, D is > 3 < 4 (precisely 3.81); in case of metabolic, classical and
variant, syndrome evolving to diabetes mellitus (27), D is > 2 ≥ 3 (i.e. 2.4); finally, in type I and type
2 diabetes, D is 1, a topological dimension [31, 24].

Assessed in the phase space, the trajectories of a deterministic chaotic system of D 3, present as a
strange attractor; in case of D > 1 < 3 the trajectories correspond to a closed loop attractor. Finally,
when D is 1, the attractor is at fixed point (Figure 5).

In clinical practice, it is sufficient to assess the fluctuations intensity of low ureteral reflex
(= nutritional capillaries), caused by digital pressure of mean intensity, applied upon the skin
projection area of the heart, and evaluate the ratio HS/minimal oscillation, i.e., “F”, fractal factor.

Interestingly, fractal dimension D is directly related to the value, calculated in seconds, of the
differential latency time (= disappearing time) of caecum- and/or aspecific gastric-reflex [8, 32]: in
health, during digital pressure of “mean” intensity upon heart projection area, as above-referred, both

### Table 1

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
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<tbody>
<tr>
<td>Low intense stimulation</td>
<td>1 cm; 7 sec. duration; 6 sec. disappearing time = type II EBD.</td>
</tr>
<tr>
<td>Mean-moderate intense stimulation</td>
<td>1.5 cm; 15 sec. duration; 6 sec. disappearing time = type I, AVA.</td>
</tr>
<tr>
<td>Moderate intense stimulation</td>
<td>2 cm; 20 sec. duration; 6 sec. disappearing time = type I normal and newborn-pathological, subtype b) EBD.</td>
</tr>
<tr>
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<td>1.5 cm; 15 sec. duration; 6 sec. disappearing time = type II, AVA.</td>
</tr>
<tr>
<td>Intense stimulation</td>
<td>2.5 cm; 20 sec. duration; 6 sec. disappearing time = type I, newborn-pathological, subtype a) EBD.</td>
</tr>
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differential latency time (= disappearing time) of caecum- and/or aspecific gastric-reflex [8, 32]: in
health, during digital pressure of “mean” intensity upon heart projection area, as above-referred, both
caecum- and aspecific gastric-reflex appear physiologically after 8 sec. latency time, then persists for less than 4 sec. (parameter value of paramount importance from diagnostic view-point), before disappearing.

After > 3 < 4 sec. (= “differential latency time”, identical to the related fractal dimension), caecum- and aspecific gastric-reflex occur again: such as parameter value, positively related to coronary microvascular functional reserve (MFR), proved to be the same to fractal dimension, informing about myocardial oxygenation, myocardial pH, microcirculatory bed structure/function, local metabolic situation, and then myocardial preconditioning.

2. Myocardial biophysical-semeiotic preconditioning

It is well known that exists a precise sympatethic nervous correlation between dermatomeres and related visceromeres, I fully corroborated with the aid of Biophysical Semeiotics [24, 25, 28, 30]. Due to this fact, ischaemic coronary diseases bring about alteration of the corresponding dermatomeres, Th 1 - Th 8, easy detectable by means of palpation [19, 32]. With the condition reversed, Th 1- Th 2 dermatomere stimulation of diverse intensity brings about sympathetic-dependent coronary tone modifications.

In the day-to-day practice, myocardial ischaemic preconditioning can be evaluated bedside in a rapid, easy, and reliable way: in health, in supine position and psycho-physically relaxed, digital pressure of “mean” intensity, applied upon skin projection area of the heart (Figure 3), and then exactly upon ventricular and/or atrial projection areas, induces the aspecific gastric- and/or caecal-reflex (i.e., their dilation) after latency time (lt) of 8 sec.: normal tissue acidosis, as clinical and experimental evidence suggests (Figures 1, 2).

After exact 5 sec. interruption, the newly applied digital pressure, as mentioned above, causes the caecal-, and aspecific gastric-reflex after lt of 16 sec.; latency time raises significantly to the doubly value: type I, physiological preconditioning (Table 2).

### Various Types of Biophysical-Semeiotic Preconditioning

- **Physiological, type I Preconditioning** → Tissue-microvascular unit type I, associated activation → MFR normal → outcome +
  (normal EBD Physiological Function)

- **Intermediate, type II Preconditioning** → Tissue-microvascular unit type II, dissociated activation → MFR compromised → outcome ±
  (normal, slightly modified EBD function, and small number of pathological EBD)

- **Pathological, type III Preconditioning** → Tissue-microvascular unit type III, dissociated activation → MFR absent → outcome –
  (normal EBD function pathological, and large number of pathological EBD)

Table 2: The table shows clearly the diverse types of preconditioning, as well as the close relation between preconditioning types on the one hand, and the function of different forms of EBD and tissue microvascular unit activation, on the other hand.

EBD: Endoarteriolar Blocking Devices; MFR: Microcirculatory Functional Reserve.

A more refined way is the following: after delimiting left and right atra and ventricles, the “mean” pressure of belt piece of a stethoscope, applied upon cutaneous projection area of the heart, enhances the coronary vessel sympathetic tone, correlated with the intensity of pressure stimulating the relate dermatomeres, in perfect accordance with sympathetic regulation, generally admitted [20]. Therefore, in health, the “mean” pressure brings about only the contraction of corresponding coronary arteries and, as a consequence, after latency time (lt) of 3,5 ± 0,5 sec., ventricle quick dilation (NN = 2 sec.), that lasts 7 sec. Both left and right ventricle dilation, as well as their return to normal basal size occurs rapidly in two seconds, due to the persisting mean pressure, giving useful information on ventricle function.

Moreover, after lt of about 4 sec. ventricle dilates again, but for 6 sec., returning thereafter quickly to basal size, indicating a physiological ejection fraction.
Finally, after about 4 sec It, under above-mentioned condition, we observe the third ventricular dilation, which lasts 5 sec. (minimal time).

From the above remarks, it appears that in health the sympathetic stimulation induces cardiac preconditioning, detectable in a few seconds with the aid of Biophysical Semeiotics: sympathetic stimulation of the heart activates physiologically local tissue-microvascular-units, wherein EBD are functioning normally.

In practice, it is sufficient to assess the duration shortening of left (or right) ventricle dilation, from the starting value of 7 sec to 5 sec., correlated perfectly with both Endoarterial Blocking Devices function and tissue microvascular unit activation (Figure 2).

Interestingly, in individual at real risk of CAD, heart-gastric aspecific reflex basal latency time is still 8 sec. (NN = 8sec.), but reflex duration is 4 sec. or more (NN less than 4 sec.), in relation to underlying risk seriousness. In addition, in the second evaluation, it persists identical: type II, intermediate preconditioning (See Table 2).

Finally, in overt CAD, even symptomless or in initial stage, we observe type III preconditioning pattern. Obviously, among these three type of preconditioning there are a "continuum" of patterns. The above remarks underline clearly the internal and external consistency of the theory of myocardial preconditioning evaluation by means of Biophysical Semeiotics: the It of cardiac-caecal and -aspecific gastric reflex gives information about myocardial oxygen supply, related directly with local coronary microcircular function, ruled principally by EBD [24, 25].


In health, digital pressure of “mean” intensity, applied upon cutaneous projection area of the heart, brings about aspecific gastric- and caecal-reflexes after a It of 8 sec., informing on myocardial oxygenation at rest, as well under stress situations, such as Valsalva’s Manoeuvre, lasting about 7 seconds [24, 25]. In addition, the latency time of both caecal- and aspecific gastric-reflexes (i.e. caecal and gastric dilation) increases significantly, raising to 16 sec., when digital pressure becomes "intense", because it stimulates coronary vessels and myocardial fibers, hence inducing local metabolic regulation of tissue-microvascular-units, i.e. activating microvascular functional reserve [11].

In my clinical, long experience, I evaluated successfully the same parameters, mentioned above, helpfuly soon thereafter Valsalva's Manoeuvre, since it allows doctor to assess bedside endothelial function [28, 30, 35-39]. In fact, primary reduction in myocardial blood flow rather than increase in demand seems to be responsible for many angina episodes, even clinically silent. These results, although interesting, have not been referred due to their exact correlation with those of cardiac-caecal and -gastric reflex, under different pressures.


Endoarterial Blocking Devices (EBD), derived from arteriolar medial layer, and located in a single point of vascular wall with two (= arterioles) or more (= small arteries, according to Hammersen) layers of smooth muscle cells, protruding to the lumen, show very different structure and form, under physiological and pathological conditions (Table 1, 3, 4): small cushions with wide base, polyloid formations, generally pedunculated [1, 2, 35-39], sphincteric formations, intimal contractile architectures. They are ubiquitous since they are located in all biological systems; more precisely speaking, only type II, normal, EBD, localized in arterioles, according to Hammersen, are ubiquitous. EBD are playing a primary role in the regulation of local microcirculatory flow-motion, as the following clinical evidence demonstrates: when abnormal, at least from functional biophysical-semeiotic viewpoint, EBD bring about impairment of Functional Microcirculatory Reserve (FMR), which contribute to conditioning the “real risk” of disorders, like CAD, whose onset will possibly occur after years or decades, as allows me to state a 50-year-long clinical experience with the original physical semeiotics.
In addition, in my opinion, it is very likely that both type I and type II, physiological EBD are contemporaneously present exclusively in those biological systems which need high blood supply only temporarily, i.e., sometimes in the day, as skeletal muscle, right cerebral hemisphere of individuals CAEMH-positive, and conjunctival mucosa, emphasizing their pivotal role in the regulation of flow-motion (Table 4).

Doctors must know that, beside normal, either inherited or newborn, physiological, type I (i.e., localized in small arteries) and type II (i.e., localized in arterioles) EBD, according to S.B.Curri from the conceptual viewpoint [41, 42], do really exist type I, newborn-pathological EBD, until now overlooked by physicians all around the world: a) subtype, characteristic of oncological real risk, and b) subtype, typical of all other disorder “real risk” and present in the different biological systems, I recently discovered and described in earlier papers, particularly from diagnostic and differential
diagnostic viewpoint (See http://www.semeioticabiofisica.it/microangiology, Physiology, and Pathology) [35-39] (Table 1, 3). These microcirculatory structures play a pivotal role in physio-pathology of the most common and serious human diseases, including diabetes, hypertension, ATS, CVD, and cancer, permitting, for the first time "clinically", to define the link existing between genetic factor and phenotype, according to my theory of Angiobiopathy [35-39].

In fact, the clinical study of Endoarterial Blocking Devices has allowed to discover and assess "quantitively" the genetic abnormalities of all biological systems, which play a paramount role in preconditioning outcome.

As a consequence, EBD clinical evaluation allowed me to recognize individuals at "real risk" of the more frequent and dangerous human disorders, as well as to comprehend fully the underlying different biophysical-semeiotic constitutions, I described earlier [35-39].

Due to these reasons, I claim the essential value of knowing both anatomy and physiology of such microcirculatory structures, i.e., EBD, both physiological and pathological, at the present time unfortunately overlooked by all clinicians around the world (unpublished personal data), in order to understand the importance of studying Clinical Microangiology, and particularly the branch, I suggested to term Clinical Microangiology of Endoarterial Blocking Devices [28, 35-40].

Furthermore, Biophysical Semeiotics allows doctor to detect at the bed-side the persistent opening (i.e., hyperstomy) of all arterio-venous anastomoses (AVA), etymologically speaking, as clinical and experimental evidence suggests: in arteriosclerotic patients, digital pressure of "mean" intensity upon the microcirculatory bed, e.g. on the microvessels of a finger pulp, scars, great or little joints, of individuals lying down in the supine position, psycho-physically relaxed with open eyes (= melatonin secretion inhibition) brings about upper ureteral reflex (= upper ureteral tracts dilate about 2,5 cm.), lasting characteristically "stiff" also during "extreme-intense" pressure [24-28] (Figure 1).

On the contrary, in healthy and young controls, the reflex, that shows an intensity smaller than 2 cm, disappears rapidly if digital pressure becomes "extreme-intense". In addition, if the subject hand is raised to 10-15 cm. above the heart level, "mean" intense digital pressure applied on the pulp of a finger does not cause upper ureteral reflex. In other words, in health, under the latter condition, AVA are closed, and simultaneously type I and II EBD contract, facilitating the blood-flow through nutritional capillaries (= activation of Microcirculatory Functional Reserve).

In addition, since the very early stage of arteriosclerosis, such as reflex persists "stiff" also under the latter conditions, hindering blood supply to local parenchyma.

Interestingly, this biophysical-semeiotic sign increases suddenly when the patient moves the other, vertically raised hand as waving good-bye - "slightest effort test" – because of the increasing of blood viscosity, detected with the aid of Biophysical Semeiotics.

Analogously, during the "simulated cold test" (= patient is thinking to dip his hands or a single finger in eis-cold water), arteriovenous anastomoses result slightly opened in healthy subjects.

On the contrary, under identical conditions, AVA opening appears to be particularly increased in patients involved by arteriosclerosis: 1,5 cm. vs 2,8 cm., respectively: p<0,001, Student's test.

Finally, middle ureteral reflex, different in type, size, duration, nature, induced by digital pressure of different intensity, applied, e.g., on tissue-micro-vascular-units of finger tip, gives useful information about the diverse endoarterial blocking devices, type I AVA as well as type II, group I and group II AVA, when present as in the foot-sole (26) (Table 1).

As a consequence, doctor may nowadays assess the diverse EBD at the bedside in easiest way, calculating the duration of heart-aspecific gastric and/or caecal reflex duration: in presence of "normal" EBD alteration and type I newborn-pathological EBD, the reflex lasts 4 sec. or more (NN = less than 4 sec.), correlated with the seriousness of underlying disorder. Moreover, the final tonic Gastric Contraction indicates the presence of newborn-pathological, subtype a), oncological, EBD.

Certainly, who knows the "direct" evaluation of middle ureteral reflexes can utilize a very refined, exhaustive, and reliable method [39] (See above-cited website).

Finally, knowing the precise location of physiological, type I, EBD (i.e., skeletal muscle, conjunctival mucosa, and right emisphere of individuals CAEMH-positive), doctor recognizes more quickly the type I, subtype a) and b), newborn-pathological EBD (Table 4).

To summarize, examining carefully the relation between EBD and myocardial preconditioning, the following parameters unavoidably must be evaluated:
Theoretical and practical Aspects of bedside Evaluating EBD and Preconditioning

It is well known for many years that patients with coronary heart disease may have no symptoms [20, 34], and that the electrocardiographic feature of ischaemia may be induced by exercise without accompanying angina [34]. Nevertheless, such "silent ischaemia" has only recently been recognized to be an important feature of ischaemic heart disease [7, 18]. The silent ischaemia prevalence is unknown, although over a quarter of myocardial infarctions are unrecognized and half of them cause no symptoms at all [14]. According to Cohn, there are three categories of people with silent ischaemia, who may be at such risk [5]. People of type 1° have no symptoms and no history of myocardial infarction or angina; those of type 2° are symptomless survivors of myocardial infarction; finally, patients of type 3° have angina together with episodes of silent ischaemia, whose mechanisms in most cases are obscure.

My data suggest that biophysical-semeiologic methods, illustrated above, are reliable, helpful, and then advisable in bed-side detecting individuals, even asymptomatic, who have to undergo, promptly and rationally, whatever stress testing, such as electrocardiographic exercise test, atrial pacing, thallium stress redistribution scintigraphy, exercise radionuclide ventriculography, and spiral CT, a.s.o., during which silent ischaemia usually may be elicited, corroborating bedside diagnosis [1, 2, 21, 29].

Furthermore, the clinical, biophysical-semeiotic selection of symptomless patients is interesting, because it can be applied on very large scale, helping doctors in actively searching for ischaemic heart disease, particularly serious when silent, from the clinical viewpoint. As a matter of facts, a lot of data suggest that episodic silent ischaemia carries a poor prognosis in stable coronary artery disease [3, 23, 35-39].

Given the accumulating evidence that ischaemia, whether silent or not, carries a poor prognosis in patients with known coronary artery disease, it is justified to follow an active policy even in patients who are totally free of symptoms [4, 33]. Essentially, the rationale for the use of histangioprotective drugs (like L-Carnitine, Co Q10, Conjugated-Melatonine, a.s.o.) in patients with ischaemic heart disease clinically silent, relates three premises.

Firstly, the favourable effects of these products on lipid and glucose metabolism, illustrated previously [27, 35-39].

Secondly, the positive influence of these drugs on angina pectoris as well as on myocardial ischaemic preconditioning, because they improve blood flow in cardiac tissue microcirculatory units [8, 9, 35-39].

Thirdly, when utilized in early stage, histangioprotective drugs can ameliorate coronary microcirculatory remodelling, e.g., lowering the number of newborn-pathological type I, subtype b) EBD: the intensity of specific middle ureteral reflex significantly decreases under such treatment [39].

Practically, in order to ascertain clinically silent ischaemia it is advisable to assess shape and intensity of low ureteral reflex oscillations, i.e. vasomotion, as illustrated above, which permits doctor to calculate the fractal dimension of myocardial microvessels deterministic chaos (NN > 3 < 4; ratio HS/Minimal oscillation = 3/1 cm.), which corresponds perfectly to the duration of heart-gastric aspecific reflex disappearing time, or differential latency time, as well as the duration of this reflex (NN < 4 sec.); the latter parameter value can be easier evaluated (Figure 4, 5).

As far as myocardial ischaemic preconditioning is concerned, it is sufficient and hence advisable in day-to-day practice to assess the latency time of the second heart-gastric aspecific reflex, i.e., in the second evaluation, performed exactly after 5 sec. interruption, namely soon after 5 sec. from the end of basal evaluation: in health, latency time raises in a significant manner from 8 sec. (basal value) to 16 sec., i.e., to doubly value.

Another refined, elegant method proved to be reliable, i.e., the assessment of shortening of left ventricle enlargement duration during the above-described test (NN = from 7 sec. to 5 sec.) and/or conversely the prolonged latency time from 3 sec. to 5 sec. or more, preceding another ventricle dilatation. This latter evaluation, however, may be a little more difficult to ascertain by doctors not experienced and skilled in the field of the original semeiotics.

From the practical viewpoint, both duration (NN < 4 sec.) and differential latency time, i.e., disappearing time (NN > 3 sec. < 4 sec.), of cardiac-caecum and/or -aspecific gastric reflex, i.e. fractal dimension D, gives exhaustive information about coronary vessels morphological and structural situation, according to my Angeobiopathy theory [35-39].
Actually, important data are easily obtained also by means of the latency time of heart-caecum and/or -aspecific gastric reflex, which informs about myocardial oxygen supply: in health, during digital "mean" pressure upon cutaneous projection area of the heart, basal latency time value is 8 sec. However, doctor must remember that in case of CAD "real risk" and CAD initial stage, such as parameter value is still normal (8 sec.), but reflex lasts 4 sec. or more (NN < 4 sec.), indicating coronary pathological condition.

Furthermore, in health, during "intense" digital pressure upon cutaneous projection area of the heart, as above described, and immediately after about 7 sec. apnea test or Valsalva's manoeuvre, the basal latency time of cardiac-gastric aspecific reflex (basal value = 8 sec.) raises significantly to 16 sec., as well as after preconditioning (i.e., doubly value) (p<0,02), showing a physiological coronary artery dilation and consequently normal endothelial function, physiological Microcirculatory Functional Reserve, type I physiological Preconditioning, and absence of newborn-pathological, type I subtype b) EBD (Table 2).

In conclusion, in a long, well-established, clinical experience, the above-described biophysical-semeiotic methods proved to be reliable, easy to performe on very large scale, useful, and suitable for detecting ischaemic coronary disease, even clinically silent or really initial, i.e. since CAD "real risk" (39).

Abstract

In the paper clinical methods, which allow doctor to assess bedside various types of biophysical-semeiotic myocardial preconditioning, and to recognize promptly coronary artery disease, even clinically silent, as well as inherited CAD "real risk", characterized by the presence of newborn-pathological, type I subtype b), Endoarterial Blocking Devices (EBD) in coronary small arteries, according to Hammersen, are fully described. These methods are based upon the "quantitative" evaluation of the following biophysical-semeiotic parameters: deterministic chaos of heart tissue-microvascular units, myocardial oxygenation, myocardial ischaemic preconditioning, and especially assessing both physiological (in health, coronary microvessels are only equipped with type II, EBD) and newborn-pathological type I, subtype b) EBD, which play a pivotal role in preconditioning. Biophysical-semeiotics procedures, easily and promptly applicable on very large scale since birth, proved to be reliable and useful in primary prevention, bedside diagnosing, and therapeutic monitoring of ischaemic heart disease, even clinically silent, since its very initial stage, i.e., CAD "real risk".

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Publication: September 2007

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