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CAD Inherited Real Risk, Based on Newborn-Pathological, Type I, Subtype B, Aspecific, Coronary Endoarteriolar Blocking Devices. Diagnostic Role of Myocardial Oxigenation and Biophysical-Semeiotic Preconditioning

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In the following, I illustrate some original methods of biophysical semeiotics (www.semeioticabiofisica.it) [1], utilizing bedside biophysical-semeiotic reflex parameters, useful and reliable in detecting coronary artery ischemic disease, even clinically silent, from its very initial stage, i.e. CAD inherited real risk, characterized by microcirculatory remodeling, wherein newborn-pathological, type I, subtype b, aspecific, endoarteriolar blocking devices play a central role [2-7]. To easily evaluate all these events it is sufficient to know stomach auscultatory percussion [See: Practical Applications, Technical Page 1, and Bibliography in above-cited website]. With regard to bedside evaluation of biophysical-semeiotic ureteral-reflexes, unavoidable in directly assessing coronary vasomotion and vasomotility, it is necessary for the doctor to have further technical knowledge.

Myocardial Vasomotility and Vasomotion Deterministic Chaos

In health, in the supine position and psycho-physically relaxed, with his/her eyes opened, aiming to inhibit melatonin secretion, digital pressure of "low-mean" intensity, applied on the heart cutaneous projection area, brings about aspecific gastric reflex (a.g. R.), as well as upper, middle, low-ureteral, caecal-, and choledocic- reflexes, i.e. upper-, mean, low-ureter as well as stomach, caecum, and choledocus dilation, the latter three after a latency time of 8 seconds exactly.

In health, at rest, the dilation of upper and low ureteral reflexes appears after 6 seconds, lasting 6 seconds, while all other reflex duration is less than 4 seconds. Such a parameter value proved to be of paramount importance, from a diagnostic viewpoint, identifying precisely the local microvascular structures and function, i.e. local microcirculatory functional reserve (MFR), hence indicating microvessel remodeling. Importantly, when a.g.R. duration is less than 4 seconds, the doctor may exclude whatever coronary disorder!

In fact, "light-mean" digital pressure, applied upon coronary trigger-points, provokes rapid oscillations of upper and choledocic reflexes (= small arteries, according to Hammersen) and subsequently those of lower ureter (= nutritional capillaries), which parallel fluctuations of the related microvessel structure, according to synergistic model. In addition, "more intense" digital stimulation causes 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 [8,9].

Interestingly, in health, the intensity of these reflexes – their diameter – appears to oscillate from 0.5 cm. to 1.5 cm. at rest, in an unpredictable manner, which last about 10.5 seconds (fractal number) and vary from 9 seconds to 12 seconds (6 cycles per minute). Physiologically, after two normal, different in intensity, unpredictable fluctuations, we observe the highest oscillation - highest spike (HS) – that corresponds to "quantic," maximal, periodic adrenalin and nor-adrenalin discharge from autonomic nervous system endings, which occurs exactly every 25 seconds, as I demonstrated earlier [1,8-15]. Finally, these signs must be evaluated also under stress tests.

I emphasize that the duration of a.g.R. disappearing, before the subsequent reflex, is physiologically > 3 seconds < 4 seconds, average value 3.81, paralleling the fractal dimension of these microvessel deterministic-chaotic dynamics, evaluated in a more difficult, refined way. To summarize, biophysical-semeiotics allows doctor to detect the chaotic behavior of both the intensity and period of ureteral (and choledocic) oscillations. However, doctors can gather at the bedside the same data – vasomotility (= upper ureteral reflex: small arteries) and vasomotion (= low ureteral reflex: nutritional capillaries) of the microcirculatory bed of all organs and tissues, including the heart – in an indirect and easier way, by means of aspecific gastric reflex duration (NN < 4 seconds).

From the biophysical-semeiotic point of view, it is useful and easy to calculate the so-called fractal dimension (D) of microvascular chaotic system: in 120 seconds 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]:

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

$$\log.n\ 4/\log.n\ 3\ [14]\ X\ \text{"f", fractal factor}$$

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 the case of the metabolic syndrome, both classical and variant, evolving to diabetes mellitus, D is > 2 ≥ 3 (i.e. 2.4); and in type I and type 2 diabetes, D is 1, a topological dimension [3,11-27].

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.

In day-to-day practice, it is sufficient to assess the fluctuation intensity of low ureteral reflex (= nutritional capillaries), caused by the 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. However, as I noted above, the fractal dimension D is directly related to the value, calculated easily in seconds, of the differential latency time (= disappearing time) of caecum- and/or aspecific gastric-reflex [1,12]: in health, during digital pressure of "mean" intensity upon heart projection area, as above-illustrated, both caecum- and aspecific gastric-reflex appear physiologically after an 8-second latency time, then persist for less than 4 seconds (the parameter value is of paramount importance from a diagnostic view-point), before disappearing. After > 3 < 4 seconds (= "differential latency time," identical to the related fractal dimension), caecum- and aspecific gastric-reflex occur again; as such, the parameter value, positively related to coronary microvascular functional reserve (MFR), proved to be the same to fractal dimension, indicating myocardial oxygenation, myocardial pH, microcirculatory bed structure/function, local metabolic situation, and then myocardial preconditioning. Under such conditions, the doctor can exclude the presence of CAD inherited real risk [28-33].

Myocardial Biophysical-semeiotic Preconditioning

It is well known that a precise sympathetic nervous correlation exists between dermatomeres and related visceromeres, which I fully corroborated with the aid of biophysical semeiotics [1,3,11,12,14,15]. Due to this fact, ischemic coronary diseases bring about an alteration of the corresponding dermatomeres, Th 1 - Th 8, easily detectable by means of palpation [16,17]. 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 ischemic preconditioning can be evaluated at the bedside in a rapid, easy, and reliable way: in health, in the supine position and psycho-physically relaxed, with open eyes, digital pressure of "mean" intensity, applied upon skin projection area of the heart, and than exactly upon ventricular and/or atrial, as well as valvular projection areas, induces the aspecific gastric- and/or caecal-reflex (i.e. their dilation) after

latency time (lt) of 8 seconds; reflex duration of less than 4 seconds gives information on MFR (this value is inversely correlated with disappearing time of reflex, i.e. fractal dimension: NN = 3.81), indicating normal tissue acidosis, as clinical and experimental evidence suggests [1,3,11,12,14,15]. After exactly a 5-second interruption, the newly applied digital pressure, as mentioned above, causes the caecal-, and aspecific gastric-reflex after doubled latency time, i.e. 16 seconds: type I, physiological preconditioning.

On the contrary, in the case of CAD congenital real risk, the first value at base line may be normal (i.e. 8 seconds), but reflex duration is 4 seconds or more. In addition, after preconditioning, latency time raising is impaired, namely less than 16 seconds, in inverse relation to the seriousness of underlying real risk: type II, intermediate, preconditioning.

Finally, in overt CAD, since its initial stage, basal l.t. is already altered, resulting less than 8 seconds, and the gastric aspecific reflex is clearly pathologically prolonged, i.e. more than 4 seconds, paralleling disorder seriousness: type III, pathological preconditioning.

In conclusion, 53 years of clinical experience allows me to state that a new era in the war against CAD has been initiated [18,33]. In fact, we are now able to recognize from birth all individuals at real risk of CAD at bedside, treat them successfully with the Mediterranean diet, conjugated melatonin, according to Di Bella-Ferrari, associated with personalized NIR-LED (LLLT) application, under clinical monitoring.

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