Quantum Biophysical Semeiotics of Oncological Inherited Real Risk of Myelopathy: The diagnostic role of glycocalyx.

*Sergio Stagnaro**
*Simone Caramel***

Introduction

The bone marrow is the charge of hematopoietic function, which governs the production of figured elements of the blood, red blood cells, platelets, monocytes, granulocytes. Neoplastic bone marrow diseases have different levels of severity, from the Inherited Real Risk in Congenital for those subjects affected by Oncological Terrain: for example, who fell ill with myeloid leukemia and myeloma from the moment of birth is carrying the Inherited neoplastic Real Risk of myelopathy (1).

The purpose of this article is to illustrate in detail Quantum Biophysical Semeiotics – QBS - diagnosis and treatment of this pathology of the bone marrow, which was described in a non-detailed way in a previous paper (1).

State of the art

The most serious disease of the bone marrow occur when the disease interferes directly with the hematopoietic function with the consequence that the production of blood cells decreases and the supply of matter-information-energy to the parenchyma is compromised. Among the most common tumors of the bone marrow, there is myeloma and some forms of leukemia.

Leukemia causes an abnormal production of white blood cells, which are transformed in tumor cells in individuals who are affected by Inherited Real Risk of Cancer dependent on Oncological Terrain (RRC) from the moment of birth (1-16). These cells invade the bone marrow and enter in the bloodstream attacking even other organs normally not involved in blood production. The decreased production of blood can result in serious harm: anemia, infections due to lack of effective white blood cells in the organism’s defense, bleeding, but this happens only after decades and decades from the onset of the RRC.

Multiple myeloma is a malignant tumor which affects the bone marrow through the excessive production of plasma cells, which in turn creates antibodies. It is a disease that occurs "clinical" in old age and is often discovered accidentally, although present at birth as RRC. Today the doctor, who knows Quantum Biophysical Semeiotics, can recognize the different inherited real risks (1-16), including the neo-plastic Inherited Real Risk of myelopathy as described below in detail (1).

Oncological Real Risk: Pathogenetic and Diagnostic Role of Endoarteriolar Blocking Device Neoformed-Pathological, Type I, Subtype a), neo-plastics

The congenital real risk, clinically recognized by the QBS, plays a major role in primary prevention of most common and serious diseases such as T2DM, CAD and cancer (16). Therefore, the physician should know about it, worthy of a nosographic clarification not restricted just to oncology, but extending it to more serious diseases in humans, because fatal too often.

In fact, anyone who knows this new physical semeiotics had the opportunity to recognize the inherited real risk of cancer: in a subject with Oncological Terrain is possible to highlight a limited area of a biological system, where there are typical microcirculatory (microcirculatory remodeling) and parenchymal alterations (genetic mutations of both n-DNA and mit-DNA), according with the theory of Angiobiopathy (1-10, 16, 17-30).

Endoarteriolar Blocking Devices (EBD) are present in the precise site where the cancer may arise, in this case, in the bone marrow. These EBD are newborn-pathological, type I, subtype a) tumoral, even in tissues where only EBD type II, ubiquitous, are physiologically present. This fact represents a primary importance's diagnostic element, which explains the unusual centralization of local microcirculatory flow (2, 16, 31-33).

Interestingly, in the presence of any QBS constitution (2-7), there are the characteristic pathological EBD type I: newborn-pathological, subtype a) in the Oncological Terrain and subtype b) in any other susceptibility disease (1-10, 16).

The pathological EBDs have different structure and function in relation to those of physiological nature. In fact, the "intense" stimulation of their trigger-point provokes a middle ureteral reflex with a reduced intensity (1 cm), while the stimulation of natural contractile structures is "mean-intense" and provokes a middle ureteral reflex of 2 cm. The duration of the reflex is the same, both of 20 seconds.

In terms of differential diagnostic we remember that only the reflex from EDB, type I, physiological, disappears during the "intense" stimulation, while it remains unchanged that from neoplastic EBDs, i.e., EBD subtype a). EBDs, newborn-pathological, type b) show an intermediate behavior: if the stimulation is maximally "intense", the relative middle ureteral reflex is reduced by one third of its initial value.

As a result, the physician should determine the possible presence of "congenital real risk", depending on the altered mitochondrial condition, inherited from the mother, monitoring the likely future trend towards the pre-morbid, pre-metabolic syndrome, which is the first step in ontogenesis, firstly of metabolic syndrome, and then of the different and respective diseases (8, 11, 16).

In other words, QBS "real risk" represents the scientific and modern version of the "metaphysics" term of traditional medicine, respecting the *adequatio rei et intellectus*: *locus minoris resistaentiae* (see below) (12).

In this regard, i.e., we can think of the precise point of the arterial wall where the endothelium HP are located, which later become the initial site of the arteriosclerotic process; we consider the location of β-cells of Langherans islets, which are predicted to run out functionally after a period of different duration of hyperactivity with increased hormone secretion (18-20).

We could present a lot of examples, all based on the same microcirculatory alterations, and on the parenchymal one, in turn based on CAEMH, particularly intense at these sites, according to the Unified Pathogenetic Theory (12).

EBD, are of vital importance, as shown previously (3-7), among all the microcirculatory structures, both functionally and structurally altered.

At this point it should be noted that the EBD are located in small arteries, according to Hammersen (blocking station, type I, by Curri) and in arterioles (blocking stations, type II). Their opening-contraction allows of course the passage of blood, while their closing-dilatation hinders the flow-motion, more or less intensively (2, 16).

In small arteries, EBD, type I, can have a pedunculated form, i.e., like proboscis (Fig. 1). If blood flow is sufficiently fast, they fall back on their stems during arteriolar dilatation, positively influencing on intake the nutritional blood capillaries, which otherwise would be significantly reduced.

In healthy, the EBD type I stay open for 20 seconds. During this time they show two emphases of the intensity of ureteral reflex "in toto" in relation to the two "normal" waves of vasomotility.

After the opening time, they close for 6 seconds with a simultaneous contraction of the arterioles, before an “highest spike”. In other words, the stimulation of the opening-contraction of EBD Type I occurs simultaneously with the maximum highest wave of arteriolar sphygmicity, according to Hammersen, below and above two "normal" arterial fluctuations.

This detailed description of micro-vascular events of Clinical Microangiology aims to highlight the harmony of the correlations of the activities of the single microcirculatory structures, whose main purpose is to ensure the control of blood flow in vessels less than 100 microns, whose role for the supply of material-energy-information relating to the parenchyma is of essential importance.

At this point, the reader understands how a change of this “thin microcirculatory game” adversely affects “tissue economy” whose pH is lowered, and of course the hydrogen-ions concentration (H^2) rises, the typical expression of an impaired mitochondrial respiration, cause of histangic or tissue acidosis.

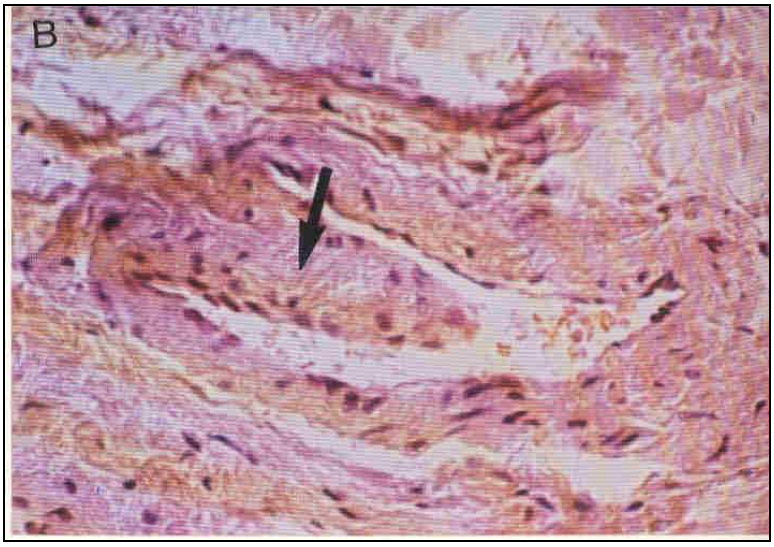

Figure 1

The arrow indicates a EBD in a small artery, according to Hammersen: its contraction (dilatation of middle ureteral reflex) increases the flow-motion in nutritional capillaries, while its de-contraction (disappearance of middle ureteral reflex) reduces the blood supply to tissue. Courtesy of Prof ..SB Curri (Le Microangiopatie. Ed. Inverni della Beffa, Arte Grafica S.p.A. Verona,1986).

EBD type II are ubiquitous and located in one of the two arterioles of the divided small one, according to Hammersen. In pancreas, mammary adipose tissue, breast, stomach, bone, lung, prostate, etc. there are only EBD type II. Their structure (longitudinal fibers within and the circular ones outside) encourage the microcirculatory flow even when they are only slightly relaxed.

In fact, QBS allows us to affirm that their sole presence testifies to the need for an abundant blood supply to the active pulsed parenchyma, as in the pancreas and other glands of internal secretion.

On the contrary, EBD type I, newborn-pathological, are present even in biological systems, in which physiologically they lack, expression of a particular QBS constitution, as well as of an inherited real risk. This fact is of extreme diagnostic importance, as mentioned above.

In the latter case, i.e., inherited real risk, this is limited only in this location, with the sole exception of the inherited real risk of "liquid" tumors.

It follows that such pathological distribution of pedunculated EBD, type I, subtype a), facilitates the diagnosis of inherited real risk of cancer and of cancer in progress: in our healthy case, bone marrow contains only physiologically EBD, type II, which are ubiquitous. On the contrary, in the inherited real risk of cancer are also seen EBD type I, with more intense blocking device, which explains the lack of local tissue oxygenation, according to the behavior of the sternal-gastric aspecific reflex.

From a technical point of view, the seat of the "inherited real risk" is characterized by:

1) a basal latency time of the gastric aspecific reflex, at rest, below normal value or even normal, but with a pathological duration of 4 seconds or more. (basal value = NN > 3 seconds < 4 seconds) impaired expression of Microcirculatory Functional Reserve (MFR);
2) the preconditioning of bone marrow, type II or intermediate, precisely because of impaired activation of the RFM (latency time does not double);
3) the microcirculatory activation, type II, intermediate, in which the increase of the sphygmicity of the arterioles (vasomotility) maintains in the standard that of capillary-venous (vasomotion).

The diagnosis of "inherited real risk" cancer of the bone marrow is done in the most simple way, based on the parametric values provided by the basic gastric aspecific reflex and the same one after QBS preconditioning (31, 32).

For example, in a healthy individual (Table 1):

- the sternal bone-gastric aspecific reflex shows a latency time of 10 seconds (NN = 10 sec.);
- the duration is less than 4 seconds (NN> 3 sec. <4 sec.), indicating that the local Microcirculatory Functional Reserve is perfect;
- there is no “micro-circulatory remodeling”;
- there are not EBD newborn-pathological;
- the histangic oxygenation is physiological.

In the case of inherited real risk of cancer:

- the sternal bone-gastric aspecific reflex continue to show basal latency time (10 seconds);
- the duration is equal to 4 seconds, pathological, and is followed by the characteristic tonic gastric contraction (2).
- the spinal cord preconditioning could highlight the persistence of an increased latency time, even not significant from a statistical point of view, remaining well below the physiological doubling of the baseline value (NN: tl Basic = 10 seconds; after preconditioning = 20 seconds) (2, 30-33).

Sternal - Gastric Aspecific Reflex (Str. G. A. R.) mean-intense digital pressure on any point of the spleen projection area– (sternal trigger points)

Latency time (Lt) in seconds	Latency time after preconditioning (pause of 5 sec.)	MFR in seconds	fD & equilibria	EBD	Preconditioning	tCG	Diagnosis
Lt = 10	Lt = 20 [Lt = 20 if intense digital pressure on single sternal trigger point — absence of bone marrow disorder]	3< MFR <4 normal MFR, associated activation, outcome +	fD ≥ 3 (ideal value fD=3.81) stange attractor	Normal EBD phisiological function	Type I Physiological tissue microvascular unit	**Negative tonic Gastric Contraction - tCG**	Health
Lt = 10	Lt < 20 [Lt = 0 if intense digital pressure on single sternal trigger point – presence of bone marrow disorder]	MFR = 4 compromised MFR, dissociated activation, outcome ±	2<fD<3 limit cycle	Normal, slightly modified EBD function, small number of pathological EBD	Type II A Intermediate tissue microvascular unit	**tonic Gastric Contraction - tGC** - local autoimmune syndrome - accompanied by gallbladder - and splenic contraction - decongestion: **positive tCG**	Myeloid Leukemia Inherited Real Risk
7<Lt <10	14<Lt < 20 [Lt = 0 if intense digital pressure on single sternal trigger point – presence of bone marrow disorder]	4< MFR≤ 5 growing compromised MFR, dissociated activation, outcome ±	1<fD≤2 limit cycle	Modified EBD function, increasing number of pathological EBD	Type II B Intermediate tissue microvascular unit	**tonic Gastric Contraction - tGC** - local autoimmune syndrome - accompanied by gallbladder - and splenic contraction - decongestion: **positive tCG**	Myeloid Leukemia Inherited Real Risk in evolution
Lt≤7	Lt < 14 [Lt = 0 if intense digital pressure on single sternal trigger point – presence of bone marrow disorder]	MFR>5 absent MFR, dissociated activation, outcome –	fD=1 fix point	Normal EBD function pathological, large number of pathological EBD	Type III Pathological tissue microvascular unit	**tonic Gastric Contraction - tGC** - local autoimmune syndrome - accompanied by gallbladder - and splenic contraction - decongestion: **positive tCG**	Overt Myeloid Leukemia

Table 1. Legend: MFR (Microcirculatory Functional Reserve); EBD (Endoarteriolar Blocking Device); CAD (Coronary Artery Disease; fD (fractal Dimension); Lt (Latency time)
*PAY ATTENTION: In case of **lymphocytic leukemia**, the triggers point are those of spleen or lymph nodes, and the values of the above table are the same*

Oncological Real Risk and Primary Prevention of malignant tumor

According to the theory of Angiobiopathy - intimate correlation existing between the biological activity of the parenchyma and that of its tissue micro-vascular system in physiological and pathological conditions - which supplements the Tischendorf's theory of Angiobiotopy - structural relationship between parenchyma and related microvessels - the accurate micro-angiological clinic investigation allows the doctor to assess the activity of the correlated parenchyma with a stethoscope, albeit in limited areas of tissue (2-7, 12, 16, 30). To clarify the abstractness of this concept, as an example, we can think of the sternal marrow of a woman whose mother died of myeloid leukemia and his father is positive for Oncological Terrain, bearing in mind that what follows is true for all other types of malignancies and other serious and common human diseases.

The most serious and common genetic disease is transmitted by the mother because of the genetic alteration of mit-DNA and consequent cytopathy (CAEMH), with the possible exceptions reported and discussed elsewhere (2-7, 12).

For instance, in one quadrant of the breast (or, rarely, in most quadrants) the physician is now able to detect the possible presence bedside of a tiny area at "real risk", defined as a result of the typical local microcirculation, due to an altered material input of matter-energy-information to the related parenchyma, in a state of local acidosis, obviously of different severity.

This microcirculatory condition, expression of a related parenchymal changes "upstream", *conditio sine qua non* of oncogenesis, is defined in literature as a inherited real risk of cancer and is characterized by the subsequent behavior of the typical aspecific gastric reflex, lasting 4 seconds or more (NN < 4 seconds), followed by the tonic gastric contraction, always pathological, after an appropriate stimulation of their respective trigger-points (see below).

At this point we emphasize the importance of the early diagnosis of cancer, for example, the outcome of an oral cancer depends on the precise moment of his diagnosis. In fact, survival depends on the timing of its recognition. Therefore, it seems remarkable the diagnostic value of the Oncological "real risk", or of early cancer, in the oral cavity, where the relief is allowed quickly and easily by the "modified" simple Valsalva maneuver (the patient exhales slightly, with the mouth closed, thus stimulating the buccal trigger points).

In healthy, after a latency time of exactly 8 seconds, the gastric aspecific reflex appears, which lasts less than 4 seconds (parametric value of paramount diagnostic importance), never followed by the contraction of the stomach.

In contrast, in the presence of "real risk" of cancer on the spot, the latency time is normal or reduced to 7 seconds, depending on the severity of the condition below. A fact of high diagnostic value, is the duration of mouth-gastric aspecific reflex which is of 4 seconds or more (NN > 3 sec. < 4 sec.).

It follows, then, the characteristic tonic gastric contraction, pathological, which is absent in healthy, expression of strong reduction of histangic pH and autoimmune lesions.

Finally, in oral cancer in progress, the latency time is reduced to just 3-4 seconds, and the reflex is quickly interrupted and replaced by an intense tonic contraction of the stomach.

From the above remarks it follows that, in patients with Oncological Terrain and inherited real risk of cancer in defined areas of specific biological systems, the known environmental risk factors do not cause, but they can only promote, facilitate, stimulate oncogenesis (14, 30 34).

The clinical and "quantitative" recognition of this condition, which is essential for primary prevention, may be done by different ways, at different grade of refinement, elegance, style and difficulty of execution, but all reliable and rapid to do.

A) In healthy individuals, digital pressure of mean intensity, applied over the midline of the sternum or above the iliac crests, causes gastric aspecific reflex after a latency time of exactly 10

seconds, regardless of sex and age, expression of tissue oxygenation. The reflex duration is greater than 3 seconds, and less than 4 seconds, correlated with the local Microcirculatory Functional Reserve.

On the contrary, in case of inherited real risk of bone marrow cancer, the latency time is still normal, reduced or not significant (9 seconds).

However, the duration is clearly pathological: 4 seconds or more (NN> 3 seconds < 4 seconds). Interesting data is provided by preconditioning marrow: in healthy, in the second assessment, which must follow the above after an interval pause of exactly 5 seconds, the latency time improves and is statistically significant, doubling the base value: the second lag time is 20 seconds.

B) The microcirculation, assessed during digital pressure of "light-medium" intensity applied on the trigger points of the bone marrow, as outlined above, is physiological: AL + PL + DL (fluctuations of upper and lower ureteral reflexes) is of 6 seconds both in vasomotility and in vasomotion.

The intense stimulation causes an identical increase in the fluctuation of the upper and lower ureteral reflexes by implementing the activation of the associated microcirculation , type I, physiological.

QBS evaluation of Oncological Inherited Real Risk of neoplastic Myelopathy

The physician should know well QBS methods and tools to understand and enforce the original QBS assessment (17-30).

However, next to the description of the refined assessment of the inherited real risk of neoplastic myelopathy, we offer here an illustration of the argument easier to understand, for which is required just the simple knowledge of auscultatory percussion of the stomach (2-5).

A fundamental experimental experience is that during the observation of the normal activity (bone marrow tissue microvascular unit diagram) the doctor invites quickly the patient with a short conventional signal to perform the *apnea test*. In practice, after the signal from the doctor, the patient suddenly do not breath for a period of just two to three seconds.

In healthy, fluctuations of microvascular vasomotility "simultaneously" cease therefore causing a decrease in local histangic oxygenation.

More simply, the latency time of the sternum-gastric aspecific reflex (NN = 10 sec.) is basic evaluated. After a pause of at least 5 seconds, to avoid misinterpretation due to a "preconditioning bone marrow (2, 12, 31), together with the apnea test, as explained above, the doctor evaluates a second time the latency time of sternum-gastric aspecific reflex, which physiologically is reduced: 8-9 seconds.

In healthy, "simultaneously" to the resumption of breathing the oscillations appear in the maximum intensity. More simply, the latency time of the sternum-gastric aspecific reflex, measured "simultaneously" with the resumption of breathing, is doubled (NN = 20 seconds).

On the contrary, from the first decades of life, in the individual with positive Oncological Terrain and inherited Real Risk of bone marrow disease, i.e., which is predisposed to spinal cord diseases, both benign or malignant, the dynamics described above appear to be significantly compromised, and occur with significant delay, slow and less intense. The simultaneous reactions of tissue microvascular unit is absent and, importantly from the diagnostic point of view, only after a few seconds (6-8 seconds in relation to the severity of the inherited real risk), the doctor observes the cessation of the fluctuations, which is **significantly** impaired.

More simply, in the individual affected by the inherited real risk of neoplastic bone marrow disease, the latency time of the sternum-gastric aspecific reflex, "simultaneously" at the beginning of the apnea test is not reduced, remaining identical to the baseline: 10 seconds. Moreover, "while"

the rapid recovery of respiration, the latency time does not increase at all: only a few seconds later there is an increase that does not reach never the double of baseline value.

The pathogenic mechanisms underlying the behavior of the sternum-gastric aspecific reflex, in healthy and in subjects with inherited Oncological real risk of myelopathy, is understandable if you know the early alterations of the glycocalyx of cells carriers of genetic mutations, both of n-DNA and mit-DNA, for the first time described in our previous articles (17-21, 30).

The cellular glycocalyx, a structure virtually ignored today by the Medicine, consists mainly of glyco-lipids, glyco-proteins, and especially hyaluronic acid, synthesized by three different enzyme complexes within the cell itself, conveyed through vesicles to the outer surface of the membrane.

The different cell receptors, such as antennas, are moving in the glycocalyx , and through it is a change of matter-energy-information from the microvascular and interstitial connective tissue to the cell and vice versa is happening.

Therefore, an altered structure of the glycocalyx, which precedes that of amorphous interstitial matrix, if not treated with adequate therapy (18), impairs the normal function of receptors of different hormones, as explained above.

These facts were used for the first time in the diagnostic field (17-21, 30).

Conclusions

The neoplastic nature of myelopathy may occur only in individuals with positive Oncological Terrain and Inherited Real Risk of cancer localized in the bone marrow, and quickly ascertained clinically from the moment of birth in the individual, through numerous methods, which differ in sophistication and technical difficulty, but all reliable to recognize the typical microcirculatory remodeling, presented in this article.

The simplest method is based on the evaluation of the latency time and duration of the sternum-gastric aspecific reflex caused by the stimulation of sternal trigger points or of iliac crests, relative to the bone marrow, where there is the congenital neoplastic real risk, depending on the Oncological Terrain.

The duration of gastric aspecific reflex (NN: > 3 sec. <4 sec.) and the characteristic tonic Gastric Contraction, which immediately follows the reflex, is also an important parameter from the diagnostic point of view.

In fact, when in the presence of a normal latency time of Sternal-gastric aspecific reflex (NN = 10 sec.), the baseline value of the duration is pathological, i.e., 4 seconds or greater: impaired expression of Microcirculatory Functional Reserve. This allows itself to recognize the congenital real risk of cancer and, therefore, to rationally select individuals from enrolling in effective primary prevention of cancer, carried out on a massive scale, at no cost for the NHS (National Health Service).

At the beginning of the third millennium, are spreading, albeit slowly, further progress in clinical medicine, which, once known by doctors, surely exert positive influences in the field of primary prevention of most common and serious diseases, today by all authors considered rising epidemic.

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* **Sergio Stagnaro MD** Via Erasmo Piaggio 23/8, 16039 Riva Trigoso (Genoa) Italy Founder of Quantum Biophysical Semeiotics, Honorary President of International Society of Quantum Biophysical Semeiotics (SISBQ) Who's Who in the World (and America) since 1996 to 2010 Ph 0039-0185-42315 Cell. 3338631439 www.semeioticabiofisica.it www.sisbq.org dottsergio@semeioticabiofisica.it	** **Simone Caramel D** Via Doberdò 3 31020 Fontane di Villorba – TV – Italy President of International Society of Quantum Biophysical Semeiotics (SISBQ) Cell. +39 338 8129030 www.sisbq.org info.sisbq@gmail.com simonecaramel@yahoo.it