Unitary Approach of Mechanical Structures and Living Organisms Lifetime

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At present, the cumulation of stress effects that are similar in nature may be carried out for engineering structures and living organisms only when the behavior of matter is linear. In reality, matter behaves nonlinearly, stresses are often of a different nature (electrical, magnetic, chemical, mechanical, thermal, etc.). In essence, the lifetime of engineering structures and living organisms can be treated in the same way. We have put forth a system of assessing lifetime based on dimensionless concepts. There are shown, in charts based on dimensionless quantities, the factors that determine lifetime. A parallel is drawn between the process of deterioration and irreversibility. Reference is made to such facts as: - with the nonlinear behaviour of matter the total effect is different from the sum of partial effects; - the synergistic effect is determined by some behavioral particulars of matter; - the influence of matter deterioration on lifetime; - the possibility of an integrated treatment in calculating lifetime in engineering structures and living organisms based on the behaviour of matter when under load. The analysis made it possible to comprehend how - in general – lifetime is generated.

Keywords: lifetime of mechanical structures, lifetime of living organisms, deterioration, critical participation, synergy

Since time immemorial man has wondered about the duration of his life and the possibility of extending it. More recently, in the twentieth century, engineers have prescribed a certain lifetime when manufacturing equipment, devices or installations. At the end of their life they were replaced. Then the problem arose regarding the calculation of lifetime after repair in the case of chemical or petrochemical reactors, nuclear reactors, steam generators and turbines, gas turbines, ships, trains, bridges etc. Many industrial plants are expensive and their replacement after the expiry date of their prescribed lifetime is considered uneconomical. At present a major challenge will be extension of lifetime in conditions of safety for some industrial structures. For example, the lifetime of a steam turbine is 40 years; one faces the challenge of extending its lifetime to 60 years.

With living organisms we might ask how long is the lifetime resulting after a period of stress, disease and treatment? Is it possible to extend the life of a living organism?

The life of living organisms and engineering structures

The external aggressions represented by noise, pollutants, toxins, viruses, bacteria, stress factors etc. as well as the effects of medicinal products, treatments etc. are considered stresses on living organisms. The implication of the above statement is that the lifetime issue can be treated fundamentally the same, both for the living and the inanimate matter. Both the one and the other are characterized by critical values of the stress or the effects obtained. The critical stress is that value of stress which causes the death of the living organism, the decommissioning or destruction of an engineering structure. The critical state can be reached either by continuously increasing the stress value, or by cumulation the effects of different stresses (the value of each being less than its critical value), but it can also be reached by reducing in time the critical parameters correlated with lifetime.

An engineering structure is assigned, from the very design stage, a certain lifetime, t_{pr}, that is implemented in the manufacturing stage t_f, while throughout its operation, owing to the conditions of actual use, its lifetime becomes $t_r < t_f$. By repairing it, it is possible to extend the lifetime, t_{ex}, so that $t_{ex} > t_f$.

Any living organism has been ascribed with a certain lifetime in the genetic code of the species to which it belongs, t_{pr}. In the intrauterine phase of the developing organism (its *manufacturing* stage) there is implemented the lifetime inscribed in its own genetic code,t_f. All throughout its lifetime the body will deteriorate naturally as well as a result of overstress. The actual lifetime, t_r, becoomes shorter than the one implemented, $t_r < t_f$. Appropriate treatment may reset the state of the body and may extend its lifetime within certain limits.

One can note the analogy between the lifetime issues regarding engineering structures and living organisms. The difference consists in the methods and means used in each case.

At present, when assessing the result of the effects cumulation upon the human body, one assigns a number to each stress, N_i. The more dangerous the effect, the higher the assigned number. One establishes a critical value, N_{cr}, of the number that defines the maximum risk for a particular disease. If

$$\sum_i N_i < N_{cr} , \quad (1)$$

there is no lethal risk to the body. But if

$$\sum_i N_i \geq N_{cr} , \quad (2)$$

the body dies. Such assessments are made in surgery. Starting from value ΣN_i one determines the risk of a new surgical intervention, as ΣN_i also includes the intervention likely to be made. The same applies to the assessment of

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the influence of factors likely to have a stressful effect on the risk of falling ill. In this respect, for instance, the Holmes-Rahe scale allows the assessment of the possibility of social rehabilitation [1].

This approach is indicative of the problem of cumulated effects. It represents a qualitative assessment backed up by assigning, also qualitatively, a quantity (number). It is not based on the quantitative correlation between stress, its effect and some characteristics of the material involved. That is why the previous relationships allow for only an indicative approach to making the decision of continuing the treatment or surgical intervention.

In case the body undergoes the action of electric fields at different frequencies that act simultaneously, some constraining conditions have to be imposed. At frequencies up to 10 MHz it is required that [2]

$$\sum_i \frac{J_i}{J_{i,L}} \leq 1, \quad (3)$$

where J_i is the electrical current density in A . m^{-2}, while $J_{i,l}$ is the J_i limit corresponding to the maximum allowable effects.

If several polluting agents act simultaneously upon the environment or upon a body, their cumulated effect resulting in the critical state is calculated with the empirical relation,

$$\sum_i \frac{c_i}{c_{i,cr}} = 1, \quad (4)$$

where c_i is the concentration of the polluting agent i, while $c_{i,cr}$ - its critical value to the environment or the body.

In order to calculate the lifetime of an engineering structure undergoing fatigue it is recommendable to use from the very design stage the Palmgren-Miner relationship [3-6], despite its shortcomings highlighted experimentally [3,7]. For the calculation of lifetime, there have been derived empirical relationships in the case of monotonic loading under creep [8], theoretical relationships for fatigue loading under creep conditions [9], or in materials with cracks, fatigue loaded [10].

At present, the assessment of the lifetime of engineering structures or of the resistance of living organisms is done with different concepts, without considering the behavior of matter depending on such factors as stress, stress rate, the sense of the external action (for increasing or decreasing lifetime). The present paper raises the problem of unitary approach to the issue of calculating the lifetime of engineering structures and living organisms, first qualitatively then quantitatively.

A new philosophy of lifetime calculation

In relations (3) and (4) one uses dimensionless quantities. This leads to the idea of solving the problem of lifetime via dimensionless variables.

Consider a body subjected to stress Y_i = (i=1, 2,...i...n). In response, one obtains effects X_i = (j=1, 2,...i...n...p). Generally there are at least two effects (the desired effect and heat effect) corresponding to one load.

One considers that the simultaneous and / or successive action of stresses Y_i is equivalent to a dimensionless value P_T which can range between zero and 1.0. The critical value of P_T is written as P_{cr}. Its maximum value $P_{cr,max}$=1.0. The value of P_{cr} decreases in time due to the deterioration of matter, which is why one writes $P_{cr}(t)$. Let D(t)- material deterioration defined as a dimensionless value ranging between zero and 1.0; it is caused by aging, viruses, bacteria, fatigue, creep, the action of harmful factors, stress, corrosion, erosion etc. P_{cr} depends on time via D(t), namely,

$$P_{cr}(t) = 1 - D(t), \quad (5)$$

where [11],

$$D(t) = \begin{cases} 0, \text{ for virgin matter (unstressed) at } t = 0; \\ 1, \text{ for totally deteriorated (damaged) matter} \\ \text{(unusuable), or for the death of live structures.} \end{cases}$$

The value of $P_{cr}(t)$ is called the critical participation of specific energy [12]. $P_{cr}(t)$, a dimensionless value, expresses the resistance of matter to stresses.

As D(t) increases over time, $P_{cr}(t)$ decreases over time (fig. 1). Lifetime, t_l, is obtained at the intersection of curves $P_{cr}(t)$ and $P_T(t)$or by solving the equation,

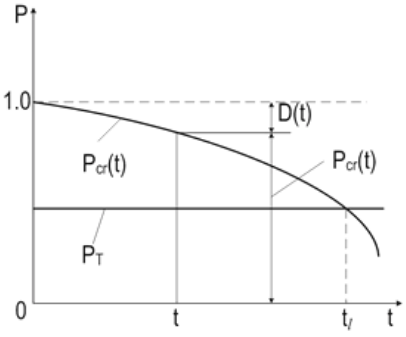

Fig. 1. Lifetime t_l at the intersection of curves $P_{cr}(t)$ and P_t=constant

$$P_T(t) = P_{cr}(t). \quad (6)$$

When $P_{cr}(t)$ varies as shown by curve 1 in figure 2: if $P_T(t)$ increases in time (curve 2) lifetime decreases as compared to the case when $P_T(t)$=constant ($t'_{i,1} < t_{i,1}$); if $P_T(t)$decreases in time according to curve 4, lifetime increases ($t''_{i,1} > t_{i,1}$).

For a certain evolution of load, $P_T(t)$, lifetime can be increased by decreasing deterioration, thus ensuring a curve that drops slowly over time (curve 3). In this case one gets: $t_{l,2} > t_{l,1}$; $t'_{l,2} > t'_{l,1}$ and $t_{l,2}" > t_{l,1}"$

Providing a slower deterioration rate over time and reducing the stress level (for example, by eliminating or reducing their duration) allows for an increased lifetime, as shown in figure 2.

If at some point t_l (fig. 3) the body deterioration (e.g. due to severe pneumonia) drastically reduces the body resistance expressed by the value of $P_{cr}(t)$, and the strong action of a virus momentarily increses the body stress expressed by the value of t $P_T(t)$, in such a way as to make possible the body death at $t'_i = t_0 + \Delta t$ when $P_T(t) = P_{cr}(t)$.

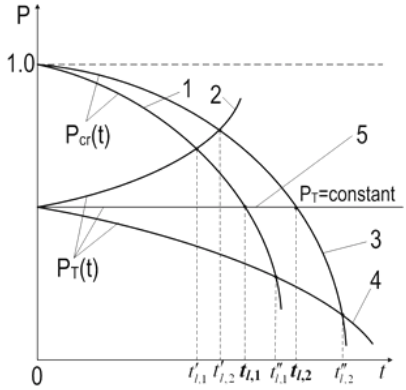

Fig. 2. Lifetime depending on the time variation of $P_{cr}(t)$ and $P_T(t)$

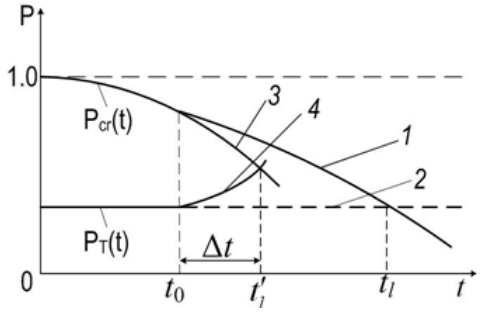

Fig. 3. Decrease of lifetime from t_l to t_l', caused by decrease of $P_{cr}(t)$ and increase of $P_T(t)$:1 –$P_{cr}(t)$ variation of body unaffected by illness; 2 – time variation of body stress expressed as $P_T(t)$, virus-free; 3 – $P_{cr}(t)$ variation after onset of disease; 4 – time variation of body stress after onset, for example, of strong virus action.

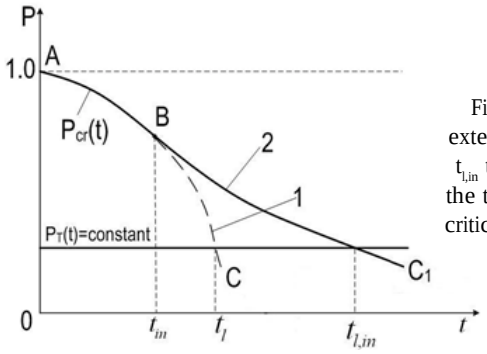

Fig. 4. Lifetime extension from t_l to $t_{l,in}$ to by reducing the time variation of critical participation, $P_{cr}(t)$

By changing the lifestyle, by avoiding negative stress generating influences, by resorting to age-appropriate treatments etc. one can reduce the temporal variation of deterioration dD(t) / dt, whose effect is that $P_{cr}(t)$ decreases more slowly over time, which is an expression of the body resistance to external actions (curve 3, instead of curve 1 in fig. 2).

The most profitable situation corresponds to the transition, at point B, from the convex curve ABC of $P_{cr}(t)$ to a concave curve BC_l(fig. 4). The transition is marked by the inflexion point B of curve $P_{cr}(t)$. The intersection of $P_T(t)$ with the convex curve 1 (ABC) yields lifetime t_l, while the intersection with curve 2, ABC_l(convex-concave), may yield lifetime $t_{l,in} >> t_l$. The shorter the time taken to cause the inflexion, t_{in}, the higher the difference ($t_{l,in} - t_l$).

The transition from curve BC to curve BC_l involves a decrease of the slope (derivative) of function $P_{cr}(t)$ with respect to time. Out of relation (5) one obtains

$$\frac{dP_{cr}(t)}{dt} = -\frac{dD(t)}{dt}.$$

A slower variation of $P_{cr}(t)$ is the result of a slower deterioration variation over time.

Neverthelss, lifetime extension may be also obtained by reducing the level of stress/stresses, which reflects the reduction in time of the total participation of external actions. In figure 5, *a* for the same $P_{cr}(t)$ - curve 2 – the transition to $t = t_l$, from the total participation of the specific energies corresponding to external loading, $P_T(t)$ = constant, at $P_T(t)$ that slowly diminishes in time (curve 3) has for an effect the lifetime increase from t_l at $t_{l,B} > t_l$. If at $t = t_l$ (fig. 5,b) one resorts to the reduction in time of participation $P_T(t)$ - curve 1 – and, additionally, the shape of the variation curve$P_{cr}(t)$ from curve 2 to curve 3, the lifetime rises to $t_{l,c} > t_{l,B} > t_l$.

For living organisms, for example: - the transition from $P_T(t)$=constant to curve 1 (fig. 5, *b*) means changing lifestyles, reducing stresses, no exposure to shocks etc.; -

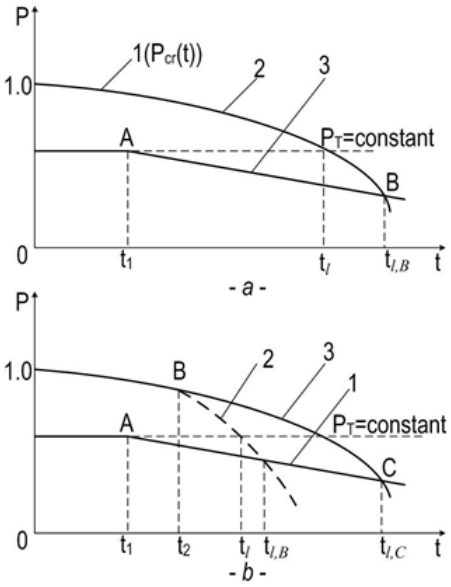

Fig. 5. Time until the damage / death of matter when: *a.* – at moment t_1 loading starts decreasing in time; *b.* – at moment t_1 the loading decreases in time, while at moment t_2 it decreases the time variation of critical participation

the transition from curve 2 to curve 3 of $P_{cr}(t)$ means increasing the body resistance (vitamin cure, repair, transplant etc.). In engineering structures, an increased or extended lifetime can be obtained by: - decreasing the values of operational parameters (pressure, temperature, rotation, etc.), which translates into lowering $P_T(t)$, curve 1; - increasing the resistance (curve 3) by appropriate and timely repair.

For the transition from the qualitative analysis to quantitative lifetime determination it is necessary to know the behaviour of matter undergoing the load and establish a method of cumulating the loads effects. In the case of a drug, for example, it will be necessary to pass from qualitative indication to the precise quantitative data of the influences and interactions with other drugs etc.

Deterioration and irreversibility

Material deterioration means reducing its capacity from a certain point of view [13; 14]. It has been found that with the passage of time, matter deteriorates. Material deterioration as an irreversible and continuous process is genetically inscribed in all the forms of material existence, is dependent on time and is defined as the ratio of the structural specific energy $\Delta E_s(t)$ irreverisibly lost and the initial structural specific energy, $E_s(0)$,

$$D(t) = \frac{\Delta E_s(t)}{E_s(0)}, \qquad (7)$$

where $\Delta E_s(t) = E_s(0) - E_s(t)$, where $E_s(t)$ is the structural specific energy at moment t. It follows that

$$D(t) = 1 - \frac{E_s(t)}{E_s(0)}. \qquad (8)$$

At the initial moment $E_s(t) \equiv E_s(0)$ so that $D(t) = 0$. When the structural energy is exhausted, $E_s(t) = 0$, the result is D(t) = 1. For the time variation of D(t) two extreme curves may be highlighted (fig. 6):

- deterioration increases rapidly in the first part of the lifetime, for example up to $t / t_l \approx 0.10$ (curve 1), then it increases very slowly until lifetime has run out at $t / t_l = 1$;
- deterioration increases slowy throughout most part of the lifetime, for example up to $t / t_l \approx 0.90$ (curve 2), then it increases rapidly until lifetime has run out (at $t / t_l = 1$).

In case D(t) varies linearly with duration (curve 3, fig. 5), the variation rate of deterioration dD(t) / dt is constant.

The dimensionless concept of *deterioration*, D(t), is similar to the concept of *degree of irreversibility* of some

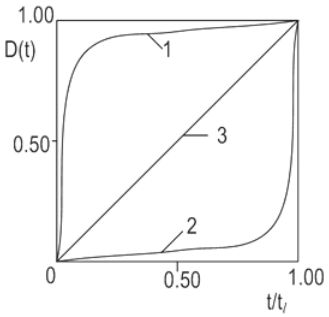

Fig. 6. Variation of matter deterioration depending on ratio of time t/t_l, where t_1 is the lifetime

process, a dimensionless concept introduced by the principle of irreversibility [12]

$$\xi_{ir}(t) = \frac{E_{ir}(t)}{E_t(0)}, \quad (9)$$

where $E_{ir}(t)$ is the amount of energy irreversibly converted into heat; $E_t(0)$ total amount of energy involved in the process at t = 0. As time passes, the amount of energy irreversibly converted also increases as well as the degree of irreversibility so that

$$\xi_{ir}(t) = \begin{cases} 0, \text{ at } t = 0; \\ 1, \text{ at } t = t_{ir}, \text{ when } E_{ir}(t) = E_t(0). \end{cases}$$

$\xi_{ir}(t)$ and D(t) are time dependent dimensionless variables (evince the exhaustion in time of the process they refer to) and increase form zero to 1.0 at best.

One has found that [12],

$$\eta_p(t) + \xi_{ir}(t) = 1. \quad (10)$$

where $\eta_p(t)$ is the process efficiency.

On the other side, form relation (5),

$$P_{cr}(t) + D(t) = 1. \quad (11)$$

From expressions (10) and (11), it follows that the process efficiency and the degree of irreversibility, as well as the critical participation of specific energy and the deterioration co-exist and are complementary.

In the processes taking place as a result of a constant amount of energy feeding the system both $\eta_p(t)$ and $P_{cr}(t)$ diminish over time as the degree of irreversibility and deterioration grow over time, respectively.

Cumulation of effects

The cumulation of effects, an important issue in the assessment of lifetime, lies in determining the total effect X caused by the action of several stresses Y_i. Such are for example an engineering structure under multiple loadings: forces, bending moments, torsional moment etc., or loadings that determine the manifestation of diseases: diabetes plus a cardiovascular disease or concurrent thyroid and heart disease etc. The question arises: how much is the overall effect and how is it calculated if the loadings Y_i are known?

The effects of such loadings can be mechanical, thermal, electrical, magnetic, chemical, biochemical, nuclear etc. With current methods one cannot determine, before the experiment, if the load is critical or not! The classical principle of the superposition of effects states that *the total effect is equal to the algebraic sum of their individual effects*

$$X = \sum_i X_i. \quad (12)$$

This relationship is valid only if:

-effects X_i are measured with the same units;

-dependence between load and effect is linear, i.e., according to the relationship

$$Y_i = B \cdot X_i, \ 1 \quad (13)$$

where B is a material constant. With a few exceptions, most of the behaviour laws used at present in various chapters of science are written as in relation (13).

In fact, in most cases the behaviour of the matter is non-linear. Dependence y(x), both for the living and the inanimate matter, can be expressed by the following general power law,

$$Y_i = C \cdot X_i^k, \quad (14)$$

where C and k are the matter constants in the respective case of load.

The use of relation (12) in the case of nonlinear behaviour has led to incorrect results. If two loads of the same nature and type, Y_1 and Y_2, act on a material body and produce main effects X_1 and X_2, then $Y_1 = C \cdot X_1^k$. and $Y_2 = C \cdot X_2^k$.

If the total load $Y = Y_1 + Y_2$ produces total effect X, one may write

$$Y = C\left(X_1^k + X_2^k\right)$$

and respectively

$$Y = C \cdot X^k.$$

By comparing the latter relations we get,

$$X = \left(X_1^k + X_2^k\right)^{\frac{1}{k}}, \quad (15)$$

which shows that in the case of non-linear behavior

$$X \neq X_1 + X_2$$

or, generally,

$$X \neq \sum_i X_i, \quad (16)$$

which means that the *total effect is different from the sum of partial effects.*

In the quantitative analysis of the cumulaiton of stress effects one should introduce the influence of the load variation rate ($\nu_Y = dY / dt$), because the effect depends on it. For example, water under load at relatively low speeds behaves like a fluid, while when subjected to high frequency oscillations, or hited by a high speed solid body it behaves like a solid. But the human body is composed mostly of water. It is obvious that the load rate influences the body behaviour.

Also, in cumulating the effects one must take into consideration the way loads are applied, whether simultaneously or successively, because the total effect is influenced by it. For example if y_1 is applied by shock and simultaneously, y_2 is applied statically, the total effect is different from the case when these stresses are applied successively. After applying y_1 by shock its action becomes static and its effect is that of a static load; in this case two static effects are superposed.

The loading of an engineering structure is quasiconstant over time, whereupon sometimes accidental loads are superposed or - in some cases – loads occur in succession (pressure, then temperature variation etc.). A similar problem is encountered when a patient is treated with different medications: for example, against blood pressure, diabetes, flu etc. How should one administer these drugs: simultaneously or sequentially? One might get an answer to these questions from the development of a quantitative theory that will make it possible to move from the current qualitative analysis of present day medical treatment, to refined treatments and medicine prescriptions based on precise quantitative correlations expressed by mathematical formulas.

Synergistic effects

Synergy means the simultaneous action, in the same sense, of several agents: biological, chemical, physical etc. Synergy is often taken to mean that the ultimate effect of several simultaneous actions is greater than the sum of their individual effects. As the opposite can also be true, i.e. the total effect may be less than the sum of individual effects, one defines as:

-*positive synergistic effect the case when the* total effect is higher than the sum of individuale effects $\left(X > \sum_i X_i\right)$;

-*negative synergistic effect the case when the* total effect is lower than the sum of individuale effects $\left(X < \sum_i X_i\right)$.

If the effect of two or more actions on a body is stronger / higher than the sum of individual effects (X_i)caused by each action, it means that the total effect X satisfies the condition,

$$X > \sum_i X_i \qquad (17)$$

where $X_i = f(Y_i)$. Such a relation defines positive synergy and it is obtained if the exponent from the law of behaviour (14) k < 1. For example, when $X_1 = 2$ and $X_2 = 9$ one obtains $\sum_i X_i = 11$. If matter behaves nonlinearly and features k = 0.5 then, according to relation (15), one gets $X = \left(2^{0.5} + 9^{0.5}\right)^{\frac{1}{0.5}} = 19.485$, which is higher than 11, that is $X > X_1 + X_2$.

One will not have the same thing if the exponent k > 1. For example, if k = 2 we get $X = \left(2^2 + 9^2\right)^{\frac{1}{2}} = 9.2195$, which is lower than the sum $X_1 + X_2 = 11$.

In conclusion, for:

k < 1 one obtains a *positive synergistic effect*; the total effect is higher than the sum of partial effects ($X > \Sigma X_i$);

K = 1, total effect is equal to the sum of partial effects ($X = \Sigma X_i$);

k > 1, one obtains a *negative synergistic effect* as the total effect is lower than the sum of partial effects ($X < \Sigma X_i$).

The synergistic effect is a result of the value of the exponent from the law of matter behaviour.

Several examples:

- sulfur dioxide alone has broad-spectrum biological effects on the human body, from simple irritation of the airways up to the challenge of chronic diseases (emphysema, asthma), until air concentrations of 1g/m^3 might cause death [15]. For sulfur dioxide, critical concentration in terms of the human body is $(c_{cr})_{SO2}$ = 1 g / (m^3 . air)[15]. At certain concentrations of sulfur dioxide in the air, plants evince biochemical and physiological effects ultimately leading to necrosis, reduction in growth, increased susceptibility to pathogens and finally a decrease in their the quantity and quality [15];

-in the superposition of sulfur dioxide effects with particulate matter in suspensions, such as ash dust, there is obtained a positive synergistic effect. The combination of these pollutants may determine increased mortality through cardiorespiratory disorders and impaired lung function;

- short-term exposure to nitrogen oxides (especially NO and NO2) leads to changes in the respiratory function (emphysema, increased susceptibility to bacteriological lung infections);

-when mixed with ozone as well as in the presence of particulate matter, nitrogen oxides have *positive synergistic effects* [15].

Extension of lifetime

Deterioration brings about a reduction in critical participation (5).

- In general, due to cracks, flaws etc. deterioration in *mechanical structures* increases over time from the initial value $D_i(\theta) \geq 0$ to a certain end value $D_e(t)$. If the structural deterioration is not monitored, the deterioration increases to a maximum value, $D_{e,max}(t) \leq 1$when the damage of the structure is produced (fig. 7).

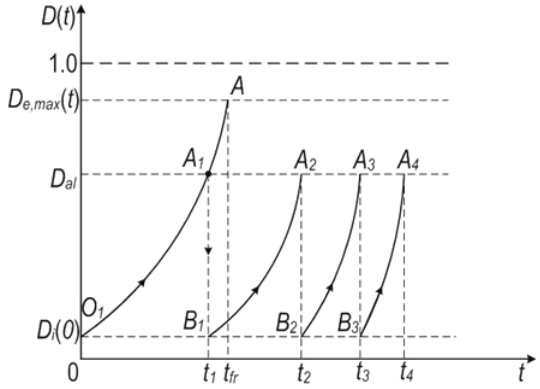

Fig. 7. Variation in time of mechanical structure deterioration: - initial O_1A - down to fracture (t_{fr}) and O_1A_1 until the maximum allowable deterioration, $D_{al} < 1$, at $t = t_1$; - after successive repairs, B_1A_2, B_2A_3 and B_3A_4

Life extension can be done by successive structural repairs. On reaching the maximum allowable deterioration D_{al} after operational life t_1, the structure is brought, by repairing, to the initial $D_1(0)$ state of deterioration. Once it has become operational again - in general – the allowable deterioration is attained after time interval $\Delta t_2 = t_2 - t_1$. The structure is repaired again at t_2, then it continues to work for a while $\Delta t_2 = t_3 - t_2$ until the deterioration rises and becomes D_{al}. Again it is repaired at moment t_3 and it lasts $\Delta t_3 = t_4 - t_3$ until a new repair is needed.

Experimentally it was found that the durations of life extension are increasingly smaller: $\Delta t_3 < \Delta t_2 < \Delta t_1 < t_1$.

At a certain moment it becomes no longer economical for the structure lifetime to be extended through repair, since deterioration grows relatively fast while the extension lifetime becomes too short.

The critical participation $P_{cr}(t)$increases due to repairs that minimize deterioration, at moments t_1; t_2; t_3 (fig. 8).

Through successive repairs, in the case presented in figures 7 and 8, the lifetime of a mechanical structure increased from t_{fr} to t_4.

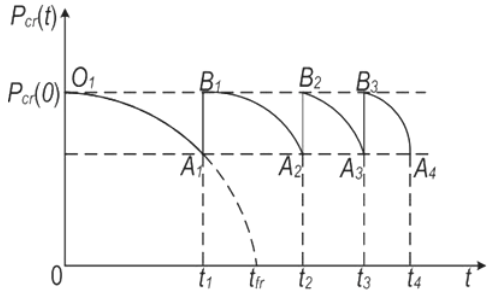

Fig. 8. Variation in time of critical participation: - initial O_1A_1; - after successive repairs (A_1B_1; A_2B_2; A_3B_3) of mechanical structure the critical participation becomes B_1A_2; B_2A_3; B_3A_4 ...

The repairs of engineering structures are made, for instance, by: welding pieces or cracked weld; replacing worn parts (bearings, filters, gaskets, gears, chains of distribution, brake pads, pipes etc. ...); replacing subassemblies with a clearly stated role (electric motors, pumps, injectors, compressors etc.).

The issue of lifetime extension in *living organisms* is the same. In general, however, after the *repair* there is no return to the initial state t_{c1}, for example, and consequently, the maximum critical participation that may be attained becomes increasingly smaller (fig. 9). Without treatment or *repairs* the living organism dies after interval $t_{v\,i}$.

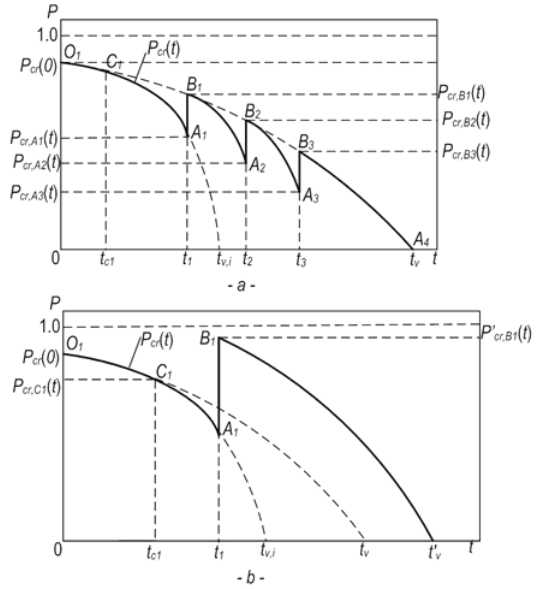

Fig. 9. Variation in time of critical participation in living organisms: *a.* with return – after treatments – to the initial curve $P_{cr}(t)$, prescribed to the body. Initial variation over time, O_1A_1 and after successive *repairs* or medical treatments (B_1A_2; B_2A_3; B_3A_4); *b.* in the case of *reviving* the body by increasing its resistance, that is by increasing $P_{cr}(t)$ by repairing in A_1,up to $P_{'cr,B1}(t) > P_{cr,\,C1}(t)$

If one applies appropriate successive treatments one can increase the critical participation (which stands for the body resistance to external *loadings*). For example, at t_1, $P_{cr}(t)$ is increased with A_1B_1, then it naturally drops on the curve B_1C_2. If one intervenes in, A_2 at moment t_2 and resets the body resistance, $P_{cr}(t)$ rises up to B_2 a.s.o. The lifetime resulting *repairs* and/or successive appropriate treatments may be extended up to $t_v > t_{v,i}$ (fig. 9, *a*).

If the body immunity or biological resistance decrease at point C_1 (fig. 9, *a*) due to illness, an accident, malfunction of one/several organs, then the critical participation is reduced, for example according to curve C_1A_1, and it reaches $P_{cr,A1}(t)$. If no steps are taken or the body restoration fails, $P_{cr}(t)$ then decreases continuously and when $t_{v,i}$ - it dies.

If in A_1, at moment t_1, through treatment, repair etc., the biological resistance of the body *is restored*, one returns along A_1B_1 to value $P_{cr,B1}(t_1)$ on curve $P_{cr}(t)$ characteristic of the respective body. In similar situations determined by the decrease of the body resistance as marked by points A_2 and A_3 on the characteristic curve $P_{cr}(t)$, there is come back to points B_2 and B_3, respectively, on the original curve $P_{cr}(t)$ by treatments and appropriate repairs. In this way, one can ensure, through successful r*epairs*, that the lifetime t_v prescribed in the genetic code is attained.

In the aftermath of an illness etc., $P_{cr}(t)$ diminishes in C_1 down to A_1 (fig. 9, *b*). If the body is *repaired*, so that (for example, through transplant etc.) path A_1B_1, is covered, the original curve is left behind $P_{cr}(t)$, one gets to $P'_{cr,B1}(t_1) > P_{cr,C1}(t)$ which means a lasting revival of the body. That is how one can enhance the lifetime, for example from t_v to t'_v (fig. 9, *b*).

One particular feature is that in the case of living organisms, as a result of natural deterioration, the critical participation decreases continuously by ageing etc. (fig. 9). One finds that after *repair* and/or treatment, the critical participation that can be attained is lower than the initial one ($P_{cr,B_1}(t) < P_{cr}(0)$; $P_{cr,B_2}(t) < P_{cr,B_1}(t)$ in the case of figure 9, *a*. It is sometimes possible — that critical participation might exceed the one at the moment when the accelerated deterioration of the body started (fig. 9, *b*). For example, $P_{cr,}\,_{B_1}(t_1) > P_{cr,}\,_{C_1}(t)$.

The *repair* of living organisms is possible by: heart transplant, liver transplant, kidney transplant, lung transplant, heart surgery, abdominal surgery, etc.

Treatments applied to living organisms have for their aim: resetting the concentration of oligoelements, vitamins etc.; countering the action of viruses, bacteria etc.; restoring the concentration of components (proteins, white blood cells, calcium, glucose, sodium, potassium, chloride, triglycerides, iron, cholesterol, magnesium etc.) within the normal range etc.

Making use of the concept of specific energy participation in correlation with the concept of deterioration (5), dimensionless variables dependent on behavior, introduced by the principle of critical energy [16; 17], allowed the qualitative analysis of the problem of extending life for both engineering structures and living organisms.

Conclusions

The qualitative analysis of lifetime issues in engineering structures and living organisms enabled the comprehension of the interactions that determine it and emphasized the possibility of its unitary approach as a matter of principle.

The concept of deterioration that underlies life analysis was correlated to process irreversibility. It was found that the deterioration and critical participation of specific energy coexist and complement each other. Based on these two concepts a new philosophy has been developed with respect to the calculation of lifetime applicable to living organisms and engineering structures.

The analysis showed that in the case of nonlinear behavior, the total effect is different from the total sum of individual effects. The nonlinear behavior is responsible for the synergistic effect. It is therefore necessary to develop a theory for cumulating nonlinear effects, common to both engineering structures and living organisms. For living organisms, this would allow to prescribe drugs and the development of precise treatment based on mathematical correlations.

The analysis of the lifetime issue has shown the analogy involving its use when dealing with both mechanical structures and living organisms. This was possible by resorting to the use of concepts introduced by the principle of critical energy

References

1. GILLANDERS A., Reflexology a Step-by-Step Guide, Gaia Books Limited, London, 1995.

2.CEPISCA, C., ANDREI H., BACANU M., Electromagnetical Pollution (in Roumanian language), Editura Electra, Bucure°ti, 2002.

3.FATEMI A., YANG L., Cumulative fatigue damage and life prediction theory; a survey of state of the art for homogeneous materials, Int. J. Fatigue, 20, 1998, p. 9-34.

4.KAMINSKI M., On probabilistic fatigue models for composite materials, Int. J. Fatigue, **24**, 2002, p. 477-495.
5.PALMGREN A., Die lebensdauer von Kugellagern, Zeitschr. VDI, **68**, 1924, p. 339-410.
6.MINER M. A., Cumulative damage in fatigue, J. Appl. Mech., **67**, 1945, p. A159-A164.
7.NINIC D., A stress based multiaxial high-cycle fatigue damage criterion, Int. J. Fatigue, **28**, 2006, p.103-113.
8. ROBINSON E.L., Effect of temperature variation on the creep strength of steels, Trans. ASME, **60**, 1938, p. 253-259.
9. JINESCU V.V., The Superposition of Effects Produced by Buckling, Creep and Fatigue, Int.J.Pres.Ves.&Piping, **53**, 1993, p. 377-391.
10. JINESCU V.V., The Principle of Critical Energy in the Field of Materials Fracture Mechanics. Int.J.Pres.Ves.&Piping, **53**, 1993, p. 39-45.
11.KACIANOV L.M., Time of the Rupture Process under Creep Conditons, Izv. Akad. Nauk SSSR, Otd. Techn. Nauk., 1968, p. 26-31.
12.JINESCU V.V., The Principles and Laws of Energonics, Politehnica Press, Bucharest, 2003.
13.JINESCU V.V., Some General Considerations Relating to the Deterioration of Matter, Int.J.Pres.Ves.& Piping, **62**, 1995, p. 31-37.
14.JINESCU V.V., Tratat de Termomecanicã, vol.1, Editura AGIR, Bucureºti, 2011.
15.IONEL IOANA, UNGUREANU C., Centrale termoelectrice, Editura Politehnica, Timiºoara, 2004.
16.JINESCU V.V., Energonica, Editura Semne, Bucuresti, 1997.
17.JINESCU V.V., Principiul energiei critice si aplicatiile sale, Editura Academiei, Bucuresti, 2005

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