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Review Article

A Review of Some Biomechanical Principles That Govern the Response Characteristics of Anatomic Materials

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Abstract Biomedical/biomechanical engineers testifying on behalf of their clients are often asked to render expert opinions about whether or not certain anatomical tissues/organs will fail when exposed to various types of biomechanical loading. Questions arise as to whether or not the loading poses a "risk factor" for potential failure of the material so-exposed; and whether or not the loading on the respective tissues/organs is "excessive, unsafe," and/or otherwise trending toward the ultimate "wear out" of the material, with significant pathological consequences. In order to express their opinions to a reasonable degree of scientific certainty, these expert witnesses must have a working knowledge of certain, very basic biomechanical response characteristics of biological materials. Toward that end, formulated in this paper is a paradigm for the evaluation of expert testimony. The model is based on five fundamental principles that govern such material responses, along with specific implications that can be deduced from them. These implications derive from a consideration of three attributes of living (mainly *soft*) biological tissues, that are conspicuously absent in all other materials subjected to the same or similar types of loading. The three attributes are: *viscoelastic* material properties, the ability of these organs/tissues to *adapt*, and their capacity to heal.

Keywords biomechanics; repetitive motion; soft tissue mechanics; forensics; litigation; work-related-musculoskeletal-disorders (WMSD's); wear-out; biomedical engineering

1 Introduction

Biomedical/biomechanical engineers testifying on behalf of their clients are often asked to render expert opinions about whether or not certain anatomical tissues/organs will fail when exposed to various types of biomechanical loading such as compression, tension, bending, shear, twisting, "prolonged repetitive motion," and vibration. (*Note:* quotation marks are used to emphasize that the terms so-identified are generally not operationally defined, much less objectively quantified [19]). Questions arise as to whether or not the loading poses a "risk factor" for potential damage to the material so-exposed; and whether or not the loading on the respective tissues/organs is "excessive, unsafe," and/or otherwise trending toward the ultimate "wear out" and "failure" of the material, with significant pathological consequences.

In order to express their opinions to a reasonable degree of scientific certainty, these expert witnesses must have a working knowledge of certain, very basic biomechanical response characteristics of biological materials. Toward that end, formulated in this paper is a paradigm for the evaluation of expert testimony. The model is based on five fundamental principles that govern such responses, along with specific implications that can be deduced from them. These implications derive from a consideration of the three attributes of *living* (mainly *soft*) biological tissues such as muscles, tendons, ligaments, spinal discs, and cartilage. These three attributes: *viscoelastic* material properties, the ability of these organs and tissues to *adapt*, and their capacity to *heal*, are conspicuously absent in all other materials subjected to the same or similar types of loading.

It is *not* the intent of this paper to offer a comprehensive discussion of the enormous scientific literature, huge body of knowledge, and current state-of-the-art as it relates to *each* of the respective biomechanical principles described. Indeed, the author is well-aware that the areas addressed by each principle represent vast, highly complex fields of knowledge and research. Some of these are the subject of much debate, a certain degree of ongoing controversy, and as-yet strange, unresolved mysteries. Rather, what is addressed are fundamental *aspects* of biological material behavior that must be considered in order to ensure the scientific viability of evidence presented in litigation that professes to attach biomechanical significance to the failure of such materials. Keeping that in mind, consider what follows to be *guidelines* that should help biomedical/biomechanical engineers to formulate meaningful scientific opinions related to their expert testimony.

2 The principle of physiologic adaptation

It is a well-established fact that prolonged mechanical loading of anatomical tissues/organs *within normal limits* (i.e., inside the operating range within which these are designed to function without consequence) stimulates the activation of physiologic adaptation mechanisms [2, 16, 24]. Adaptation mechanisms act to alter some or all of the following:

(i) the anatomical *structure* of the material (e.g., hypertrophy to reduce stress levels, reorientation of architectural configuration to optimize load distribution, and atrophy resulting from prolonged lack of use);
(ii) the *operating set points* toward which the tissue migrates (*note: operating set-points* are reference load values established on the basis of the metabolic requirements and strength capabilities of the tissue to support the load in question. Metabolic requirements include optimized, minimum energy considerations. Strength capabilities also include gender, age, genetics, tissue properties, and many other *confounding variables*, including smoking and congenital defects);
(iii) *control biochemical and mass transport parameters* that include, for example, key hormones, enzymes, and neurotransmitters, as well as cell membrane permeability characteristics;
(iv) tissue/organ *transfer functions* (*note:* transfer functions are material parameters that define its loading/response attributes, or input/output characteristics);
(v) *biochemical composition* (e.g., a redistribution of elastin and collagen fibers, and changes in the *types* of the latter);
(vi) tissue/organ *material properties*,

to list but a few. Such alterations allow the material to best accommodate the loading to which it is exposed, with a minimum of effort. Note, however, that the operative words above are *within normal limits*. Indeed, to a biomechanical engineer, the word "excessive" should be taken, quite formally, to mean *exposure of organs and tissues to loadings that fall beyond the upper limit of the operating range within which the material is designed to function without consequence* [19]. Thus, we are led to consider the following.

3 The principle of anatomic engineering design for specific biomechanical operating ranges

The "normal" (most common) operating range for anatomical tissues manifests as a continuum. It is bounded at one extreme by a lower limit, below which a *use it* or *lose it* principle is activated. At the other extreme there exists an upper limit that, among other things, depends on the time-history of the loading to which the material is subjected [23]. In the case of steady-state, continuous loading, the upper limit is called the *Ultimate Strength* of the material, beyond which it fails in one or more of the following *modes*: tension, compression, torsion, bending, twisting, and so on. In the case of time-dependent, unsteady, cyclic, repetitive loading, the upper limit is called the *Endurance Limit* (or *Endurance Strength*) of the material, below which it can be (hypothetically) loaded repetitively *forever* without failing due to "cumulative" effects. Above the endurance limit, the material eventually fails under *fatigue loading*.

In general, a material that can withstand high stresses *together with* considerable deformation is called a *ductile tough* material; anatomical examples include skeletal and cardiac muscles. Ductile tough materials are *tougher* (i.e., they require a greater amount of energy to get them to fail) than those that can resist high stresses, but have little or no capacity to deform significantly in the process. These, like teeth and bones, are generally *hard, brittle* materials.

Ductile tough materials are also tougher than the ones that have a high capacity for *deformation*, but can only withstand relatively low stresses without failing. Anatomical examples include some types of smooth muscle and ligaments, which are called *ductile soft*, or *plastic* materials. Of significance to the biomechanical engineer is the fact that *cyclic fatigue failure is typically characteristic of hard, brittle materials that have little ability to deform, or ductile-soft, plastic materials that have a low tolerance for stress* (see, e.g., [26, 27]). Moreover, in general, the endurance strength of a viscoelastic material is somewhat lower than its ultimate strength. The difference between the two is specific to the material, itself, depending, for example, on its degree of anisotropy, inhomogeneity, nonlinear behavior, and embedded imperfections. Also coming into play are thermodynamic variables such as temperature, pressure, and humidity.

Taking all of the above into consideration—for the purpose of analyzing and discussing the biomechanical behavior of most anatomical soft tissues/organs—their operating range, spanning the continuum of loading levels between the two extremes defined above, may be conveniently subdivided into three basic regions. The regions are named and identified with respect to specific loading "threshold" values. These will depend on whether or not organ system *operating set points*, or input/output *transfer functions* (load-to-response ratios), or both, will need to be changed or otherwise modified in order for the material to accommodate the loading to which it is exposed. At each loading threshold, or *break point*, adaptive mechanisms (*Principle 1*) can change one or both of these material response characteristics. Thus, in accordance with a reasonable paradigm proposed by Mueller and Maluf [10], we have the following.

Region 1 (defined by a decreased material stress tolerance). In this region, the biomechanical loadings (steady-state or cyclic) to which the tissue/organ is subjected are below a threshold value (break point) that signifies a lower limit to the loadings that one typically encounters while performing

"routine" activities of daily living. However, as low as that lower limit might be, it is still above the threshold loading below which a *use it* or *lose it* principle will be activated. That is to say, prolonged exposure of the material to this range of reduced, "sub-par" loading levels will cause it to *atrophy* (*adapt*) down to the minimum structural configuration, chemical composition, and metabolic milieu that is required to "barely" accommodate said loading—but the tissue will not totally disintegrate or suffer damage for lack of use. It is still being "used," so one will not "lose" it; but the material is minimally loaded, either in a steady state, or cyclicly.

As a result of prolonged tissue exposure to load levels in Region 1—from the *use-it-or-lose-it break point* up to the *lower-limit-of-routine-activities-of-daily-living break point*—there will ensue consequential tissue atrophy, neurotransmitter/hormonal/enzyme changes, altered cell membrane permeability, biochemical composition adjustments, and compromised tissue material properties. These will have resulted from an adaptation-induced lowering of the *absolute magnitude* of organ system operating set points. Recall that the latter are "reference," optimum stress-exposure values to which the tissues attempt to equilibrate as a function of a *minimum* energy principle that governs metabolic processes/physiologic function [1,12,16,17]. Thus, *threshold, critical load levels for tissue damage and subsequent adaptation are likewise lowered.* In other words, adaptive mechanisms in the *decreased stress tolerance region* of tissue loading cause not only operating set-points *within* the region to shift as necessary, but also they lead to a basic downward shift of *the entire set of break points that demarcate the lower and upper limits of the respective loading ranges involved*. Thus, the tolerance of the subject tissue to subsequent loading *across-the-board* as well as its ability to *adapt* to increased load levels are both compromised.

However, the downward shift of threshold break points is *uniform*; they all move by the same amount, so that threshold ranges (i.e., upper break point minus lower) remain the same. The result is that the adaptive mechanisms involved in this loading region uniformly change *only* the organ system's *operating set-points*, not its input/output transfer functions and load-to-response ratios. The latter remain unchanged, which means that the tissues/organ-systems involved here continue to *respond* the same way, independent of the actual biomechanical loading to which they are subjected—in *this* operating range of the tissue.

Region 2 (defined as requiring only "routine maintenance"). Continuing with the paradigm proposed by Mueller and Maluf [10], we have, in this so-called, "routine activities of daily living" region, biomechanical loading situations (steady-state or cyclic) of the material that are again above the *use-it-or-lose-it* threshold break-point. But now, the loading is also *above* the threshold level (lower break point) for typically "routine" activities of daily living, which is the lower operating limit for this region. The upper operating limit is the break-point beyond which still more-significant adaptive mechanisms would need to be activated in order for the material to tolerate the loading to which it is subjected, without experiencing significant adverse consequences. By "more-significant" we mean that, beyond the upper operating limit of this region, the material involved needs to have altered *both* its *operating set points and* its *input/output transfer functions* if it is to continue to accommodate said loading without failing.

In this second loading region, the above does not need to happen—adaptation requires only that the operating set points be changed—for example, the area over which the load is distributed—in order for the tissue/organ to operate within acceptable stress limits; its transfer functions can remain status quo. Thus, tissue operating set points are allowed to *float*—to respond to the loading *within* this region by moving up or down as necessary. However, unlike Region 1, under prolonged exposure to loading in Region 2, floating set-points derived from corresponding adaptive mechanisms do *not* cause the *threshold* break points to shift uniformly across-the-board. Rather, the absolute values of the *threshold* break-points remain constant under continuous loading, such that the corresponding "routine maintenance loading range" does not, itself, shift up or down as a function of the load, itself, as it did in Region 1. In loading Region 2, operating set points can change *within* the region, but its limits (break points) remain fixed—nothing changes; the material handles *routinely* the loading to which it has become "accustomed," without the need to change its input/output *transfer functions*; the tissue/organ always responds the same way to the same type of loading.

And although the material is in a constant state of *dynamic adaptation* (*floating set-points*) to fluctuating stress levels, a steady-state *homeostasis* (I prefer the term, *stationarity*; see [18]) occurs when tissue *degeneration* (as loading levels drop) is exactly equal to tissue *regeneration* (as loading levels rise). In the steady state, there is only "routine" daily tissue turnover with *no* net gain (i.e., *hypertrophy*) or loss (atrophy); there is simply "routine maintenance" to "routine loading" that results from "routine activities of daily living"—hence the name given to this loading region. Moreover, should there occur daily "microtraumas"—such as they are (*note:* the term is rather ill-defined; see, e.g., [4,6,19])—under such "routine" daily loading, these too are "routinely" fixed overnight. That is to say, thanks mainly to the efforts of *Human Growth Hormone*, the damaged tissues heal completely; the term "cumulative trauma" is rather meaningless for viscoelastic biological materials.

Region 3 (defined by an increased material stress tolerance). In this region, biomechanical loading (steady-state or

cyclic) is above the upper limit for "routine" activities of daily living, but still below the *Ultimate Strength* (in the case of steady-state loading) or *Endurance Limit* (in the case of cyclic loading) for the particular tissue/organ involved. That is to say, in this region, the material is still "okay," in the sense that it can still undergo a net, *adaptive hypertrophy* to accommodate an "overload," and the process can proceed up to a maximum structural configuration and/or chemical composition (or both) that is required to accommodate this overload. Moreover—except in cases of *acute* impulsive trauma or impact loading—the tissue/organ will not mechanically fail upon being subjected to this loading, either as a result of exceeding the upper limit of normal, or as a result of fatigue. Quite to the contrary, essentially, the *opposite* will take place as did in Region 1.

Whereas in Region 1 the absolute magnitude of tissue/organ-system operating set points was *reduced* in response to prolonged exposure to sub-maintenance load levels, in this Region 3, these are actually *raised!* Likewise, *threshold load levels (break points) for tissue damage and subsequent adaptation are also raised*. In other words, adaptive mechanisms in the *increased stress tolerance region* of material loading cause *the entire set of break points that demarcate the lower and upper limits of the respective loading ranges involved to be basically shifted upward*—a strain-hardening or *dilatant* effect [11,17, 21]. That being the case, what are enhanced are both (a) the tolerance of the subject tissue to subsequent loading *across-the-board*, and (b) its ability to *adapt* to increased load levels. The tissue effectively becomes stronger! It is more tolerant of subsequent biomechanical loading and experiences injury at significantly higher levels of such loading, mainly due to its capacity to adapt.

This increase in strength with increased loading is typical of composite, nonlinear, viscoelastic materials that exhibit *dilatant mechanical behavior* (*ibid.*), such as muscles [11,17] and other types of soft anatomical tissues [21,30]. Moreover, as was the case in Region 1, here, too, the upward shift of threshold break points is *uniform*, across the board, so that threshold *ranges*—for example, from the upper limit of "routine" activities of daily living to the lower limit of the next Region 4—again, remain the same. This means that the adaptive mechanisms involved, again, change *only* the tissue/organ system's *operating set-points*, not its input/output transfer functions and load-to-response ratios. The latter continue to remain unchanged; the material continues to respond the same way, independent of the biomechanical loading to which it is subjected in this operating range, *up to a critical point!*.

That critical point is the loading-level extreme for which the material will still be able to adequately *recover* between bouts of increased loading. For time-dependent loading, this, of course, depends on the amplitude and frequency of the repetitive cycles. Beyond that point, as necessary, *both* system operating set points *and* input/output transfer functions will undergo whatever *adaptive changes* are required—either in structural configuration, or biochemical composition, or both—to successfully accommodate the loading to which the tissue/organ systems are exposed [16]. Nevertheless, should adequate recovery *not* be possible, the tissue *might* experience some daily minor damage ("microtrauma?"), in which case, in this region, again, such damage due to "overloading" is also *routinely* fixed overnight, thanks mainly to the efforts of *Human Growth Hormone* [5,14]. Indeed, that explains why at least 50%, to as much as 80% (women higher than men) of one's daily supply of growth hormone is released into the blood stream during the first few hours of sleep [14]. The action of this hormone is to repair and heal damaged tissues/organs completely, overnight, ensuring that any potential impairment to normal function is momentarily temporary, not permanent.

4 The principle of nonlinear viscoelasticity

Within each of the above three loading regions, one can derive self-similar [20,22], power-law [21,22,28] stress-strain or stress-strain-rate constitutive relationships—considered within the framework of nonlinear, structured continuum mechanics—that effectively define the corresponding tissue's "dose-response" to being loaded within that respective region. That is to say, consider a simple power-law equation such as,

$$\tau = k[\epsilon']^n,$$

where τ represents the load (stress, stress-rate) to which the tissue is subjected, ϵ' represents its response (deformation, deformation-rate), and "n" and "k" represent constitutive parameters—material properties upon which its response depends, including thermodynamic variables, piezoelectric properties, self-similarity, perhaps the load, itself, and so on (*ibid.*) (note that piezoelectric—literally "pressure-electric"—effects have been produced in a number of soft, as well as hard, tissues [25]. They appear to be associated with the presence of oriented, polarized, fibrous proteins such as collagen. Piezoelectricity may be a universal property of living tissue, and, as an electrical stimulant, may play a significant part in activating several physiological phenomena, such as mechanical-stress-related "injury potentials" [13,16]).

In the equation above, "n" is a non-linearity index (sometimes called a non-Newtonian index) that takes on values greater than unity for dilatant materials (such as soft anatomical tissues), and values less than unity for pseudoplastic materials (such as blood). The parameter "k" is a consistency index (Young's modulus for linearly-elastic solids; fluid dynamic viscosity for Newtonian fluids) that

depends mostly on the biochemical composition of the tissue/organ involved. Thus, specific values of "n" and "k" provide a total picture of the material's response to loading, and the likelihood that it will fail under certain conditions [21].

Note, in particular, that from the point of view of failure mechanisms for nonlinear *viscoelastic* materials—which includes virtually all biological organs and tissues—the *amount* of loading is of far less significance than the following points: (a) *how the load is distributed throughout the material* (i.e., *stress*, rather than "force"), and (b) the *time-rate-of-change of the load* (i.e., *strain-rate*, rather than "deformation"). This consideration leads us to the following.

5 The principle of loading history/parameters

At least the following biomechanical variables are required to uniquely define the *loading history* and *loading parameters* to which any given tissue/organ is subjected:

(i) the *type* of loading to which the material is exposed. For example, is it being pulled (i.e., in tension), squeezed (i.e., in compression), twisted (i.e., in torsion), sheared (i.e., "shaved"), bent (i.e., "folded up"), wrinkled (i.e., "crumbled-up"), or what?

(ii) the *magnitude* (in the case of steady-state loading), or *amplitude* (in the case of cyclic loading), of the load;

(iii) the *direction* (vector resultant) of the loading;

(iv) its *symmetry* (or lack thereof);

(v) its *duration* (the time-of-exposure of the tissue to this loading: when did it start? when did it end? how long did it last?);

(vi) its *distribution*, which converts *force* into mechanical (as opposed to psychological) *stress*. What is most critical for most materials is less the actual *force* to which they are exposed, as is the *distribution* of that force (e.g., across a given area) within that material. Mechanical *stress* contributes more to material failure than does *force*, per se.

(vii) its *dynamics*, for example, is it of a continuous, steady-state nature or is it time-dependent? If the latter, does it "ramp" up (or down)? Is it repetitive, pulsatile, cyclic, periodic?

(viii) if periodic, what is the *frequency* of the loading and, even more basic than that, what, exactly, *are* the criteria for classifying any particular type of loading as being "repetitive?" Moreover, how is "repetitive" to be distinguished from terms such as, "frequent," or "often", as opposed to "once-in-a-while," or "occasionally?" And finally, at what frequency does one cross the line from "periodic" loading to "vibration-exposure?" The following are suggested operational definitions and criteria for the frequency of exposure of biological tissues/organs to periodic loading. They are derived from a number and variety of sources, such as government reports (e.g., [15, 29]) and Ergonomics text books (e.g., [7,8]):

(a) *occasionally* shall mean that the material is subjected to a specific, well-defined cyclic loading from "very little" (undefined) up to no more than one-third (cumulative, not continuous) of a typical period of wakefulness, some 16 hours total. In other words, "occasional" is taken to mean anything less than a cumulative total of no more than 1/3 of a typical period of wakefulness;

(b) *frequently* shall mean that this same exposure occupies more than one-third, but less than two-thirds (again, cumulative) of a routine period of wakefulness; and that, during this period, the loading might be repeated from 12 to as many as 60 times per hour (i.e., a frequency of one cycle each minute);

(c) *constantly* shall mean that the tissue/organ is subjected to the loading at issue in excess of two-thirds of a typical period of wakefulness with, again, the frequency of exposure being as stipulated above.

Keeping in mind these subjective definitions of *occasional, frequent*, and *constant* exposure, we can get somewhat more specific, and objective, as follows:

(a) tissue/organ biomechanical loading is considered to be *somewhat repetitive* if the exposure frequency is less than 12 times per hour (one cycle every five minutes);

(b) simply *repetitive* if exposure frequency is between 12 and 60 times per hour (one cycle every five minutes to one cycle per minute);

(c) *moderately repetitive* if exposure frequency is between 60 and 900 times per hour (one cycle per minute to 15 cycles per minute or 4 seconds per cycle);

(d) *highly repetitive* if exposure frequency is between 900 and 1800 times per hour (one-quarter to one-half cycle per second), but for no more than 15-to-25 minutes at a time (15-minute intervals of exposure are defined to be "short bursts"); and,

(e) *excessively repetitive* if exposure frequency rates exceed 0.5 cycles per second, or two seconds per cycle for periods exceeding 25 minutes at a time.

Beyond that, cycling rates of 15 or more per second take us into the realm of *vibration*, up to about 2 kilohertz (2000 cycles per second; see [3,16,17]). Finally, along with the above criteria for time-dependent loading, one can define the *continuous* (not cumulative) *exposure* of the material to repetitive loading cycles as being of

(a) *long duration* if the continuous exposure persists for 2–8 hours during a typical period of wakefulness;

(b) *medium duration* if such exposure persists for no more than 1–2 hours during this same period; and

(c) *short duration* if the tissue/organ is subjected during this period to continuous cyclic loading for less than one hour total.

In summary, we see that biomechanical loading can vary from *occasional* (acute), *steady-state* (zero-frequency, not at all time-dependent) at one extreme—through a range that includes various regions of *pulsatile, time-dependent exposures* that have associated with them widely-varying loading frequencies and exposure durations—to *constant* (chronic), *vibration* (of order 15 cps to 2 kilohertz) at the other extreme. (*Note:* at tissue resonance frequencies between 5 Hz and 10 Hz, one begins to experience "jitter;" at 20–30 Hz, it is described as "chatter;" at 60–90 Hz, the sensation is one of "tremor;" and beyond 2 kilohertz, the vibration frequency is too high to elicit any type of material response—the tissue/organ basically does not even "know" it is being loaded at that rate (*ibid.*).) Continuing, then, with our discussion of biomechanical loading parameters, they also include the following:

(i) the *rate* of loading (for viscoelastic materials);

(ii) the shape of the *loading curve*, especially for cyclic loading. For example, is it sinusoidal, square-wave, rectangular, triangular, ramp-like, "scissor-like," and so on?

(iii) the loading *history*—taking into consideration such phenomena as *hysteresis, cascading effects, adaptation mechanisms*, and *previous unresolved tissue damage*. The latter weakens the material's ability to tolerate subsequent loading by lowering its failure threshold and compromising its ability to adapt. Related to this is the:

(iv) *memory* of the tissue/organ to previous exposure. This may bring into play possible *cumulative effects* of repeated exposure—not necessarily repetitive, but "often enough" to exceed the material's need to rest, recover, and, as necessary, heal. Note that "cumulative" becomes meaningful *only* in the latter case, which is to say, when the material is subjected to loading that *exceeds* its ability to tolerate it without consequence, as defined herein. Given such attributes as nonlinear viscoelasticity, adaptation, and healing, there really is no such thing as "cumulative biomechanical injury" for such soft biological tissues as muscles, tendons, and ligaments. The term is ill-defined, not scientific, and inconsistent with the mechanical behavior of the tissue. Continuing, biomechanical parameters also include the following:

(v) any *transients* that might appear within overall loading patterns; and, last, but certainly not least,

(vi) the "$F\triangle t$" profile of the loading, where "F" represents the magnitude (if steady) or amplitude (if cyclic) of the load, and "$\triangle t$," the duration of tissue exposure to the load. In this respect, for a given F,

(a) if $\triangle t < 0.01$ s (less than 10 ms), the material is said to have experienced a *jerk*—such as being ejected at high speeds from a jet fighter aircraft. In the limit, for $\triangle t \to 0$, a discontinuous step-loading function called a *Dirac Loading* is approached.

(b) if $\triangle t$ lies between 10 and 100 ms, the tissue/organ is said to have been subjected to *impact loading*—such as is typical for most automobile collisions.

(c) if $\triangle t$ lies between 0.10 s and about one-third of a second, the loading is said to be *impulsive*—which is typical of the most common types of *short-term* activities of daily living, such as *sudden* movements or "impulsive reactions" to startling events, classified as *acute function loading*.

(d) if $\triangle t$ is in the 0.333–1.333 s range (technically called a *pulse*, as in, the time period of a normal heart beat, as opposed to an *impulse*) or more (up to about 5 s), we are essentially in Region 2 defined earlier for "routine" human movement in the performance of normal activities of daily living. Recall that in this Region, *routine tissue maintenance* prevails. Many physiologic functions, such as the previously-mentioned beating of the human heart (resting *pulse-rate* of 60–80 beats per minute), or the *respiration rate* (at rest, 16–18 breaths per minute), lie in this $\triangle t$ range [16].

Potential failure of the material, with subsequent *inflammation* can occur if it experiences [10]:

(i) *excessive* (above the upper limit of the engineering design of the material) *loading* for very *brief* periods of time—on the order of "jerks" or "impacts;" or

(ii) relatively *mild* loading (e.g., lower half of Region 2), but *chronic* (or *continuous*, $\triangle t \to \infty$), with no time to rest, relax, or heal; or,

(iii) somewhat *medium* loading (e.g., upper half of Region 2), but applied quite "often," as defined herein.

In the realm of vibration, one prime cause of tissue damage is *resonance phenomena*, wherein the material is "excited" or driven at its *natural frequency* [3]. Short of that, *discomfort* is more of an issue than is vibration-induced *disease*, per se. The above considerations lead us to the following.

6 The principle of tissue/organ failure

If anatomical tissues/organs are subjected to steady-state and/or cyclic biomechanical loading that falls *below* the lower limit of Region 1, or *above* the upper limit(s) of Region 3, then the material involved will risk *failing* by mechanisms associated with the corresponding loading type and history. *Note:* obviously, *acute, excessive, traumatic* loading for example, falling down a flight of stairs, getting thrown from a horse, being involved in a motor vehicle accident, and so on—can definitely lead to *immediate* tissue/organ failure if it is subjected to extreme *jerks, impacts, or impulses*. That being said, our main concern in this paper relates to the following.

(i) *Prolonged* loading *above* the upper limits of Region 3 (i.e., the *maximum loading threshold*) which defines what we shall *mean* by the term "excessive" biomechanical loading. Recall that these upper limits are either the *Ultimate Strength* of the material in the case of steady-state loading, or its *Endurance Limit* in the case of cyclic loading. *Excessive loading* so-defined can result in tissue/organ failure, with symptoms of *physiologic* (as opposed to *mechanical*) fatigue, pain, discomfort, and/or, impaired function of the damaged material. The operative words here are
   (a) *excessive: beyond* the ultimate strength or endurance limit of the material, in the sense of exceeding its ability to tolerate the subject loading without consequence; and
   (b) *prolonged*: in the sense of prevailing continuously at rates faster than the material can recover and/or heal.

Stated another way: if one stays inside the operating range within which the material is *designed* to function routinely without consequence, due to its *nonlinear*, *viscoelastic properties* (including damping and dilatancy), then the corresponding tissue/organ can *adapt* in response to the loading to which it is subjected. This it can do by activating stress-induced (piezoelectric?) compositional, functional, structural, and other mechanisms that can *adjust* the material's operating set points and, if necessary, input/output transfer functions. Inside the material's designed operating range, it can also *heal* successfully, as necessary, on a day-to-day basis, so it is "safe" as far as its health is concerned, and *not* "at risk" of failing under such loading.

Moreover, in the *absence* of such clinical symptoms as fatigue, pain, discomfort, and/or impaired function, the tissue/organ is *not* in a condition considered to be *clinically significant*. That is to say, even though routine bouts of "microtrauma" *might* be occurring on a daily basis, the material is not in a "danger zone" for permanent, "cumulative" damage. As a matter of fact, that is one of the reasons that the very *existence* of "microtrauma"—"accumulating" in a "straw-that-broke-the-camel's-back" sense to ultimate failure in soft biological materials—is debatable (see, e.g., [4,19]); the condition presents with no *clinically significant symptoms*, and it has never been clearly, *operationally* defined or, for that matter, diagnostically observed and reported, to wit the following.

(ii) The *idea* of "microtrauma" *accumulating* to cause ultimate tissue/organ failure is a purely conjectural hypothesis, based on no *hard* scientific evidence for *soft* biological tissues. The presumption is formulated as an analogy to *fatigue failure* of solid materials, such as metal clothes hangers and paper clips. That is to say, most of us are familiar with the simple experiment where one takes a thin metal rod or wire (like a clothes hanger or paper clip), bends it back and forth several times in the same place, only to have it break apart at the loading site after but a few bending cycles, with a concomitant generation of a significant amount of heat, called *strain energy*, at the corresponding site of failure (see, e.g., [9, 23]). The "bending back-and-forth several times" constitutes repeated, alternating (or cyclic) mechanical stressing of the material—technically called *fatigue loading*—and the eventual "breaking apart at the site of loading" is technically called a *fatigue failure* of the material (*ibid.*).

Fatigue failure in a solid material generally begins as a brittle crack at the site of loading. The crack then propagates through the material, causing it to fracture at load intensities below what the material could "normally" be able to tolerate without consequence under continuous, *static* loading. Indeed, the majority of engineering failures of *solid* materials are caused by such *mechanical* (as opposed to *physiologic*) fatigue, often derived from exposure to prolonged vibration-loading.

Because of one's general familiarity with fatigue loading as a common cause of failure in *solid* materials, it is tempting—especially in our nation's courts of law—to apply this same paradigm to the biomechanical failure of *nonlinear*, *viscoelastic biological* materials, such as cartilage, muscles, tendons, ligaments, and spinal discs. The analogy suggests that such materials—when subjected to "repetitive" (again, a qualitative term used rather loosely), "excessive" forces (also not generally *operationally* defined, *objectively* measured, or systematically *quantified*)—will suffer some sort of tiny injury, a *fatigue-induced, soft tissue microtrauma*. For whatever reasons—also alluded to in vague, ambiguous, qualitative terms derived mostly from anecdotal evidence—the microtrauma does not totally heal… either at all… or certainly not *properly* (i.e., to "original specifications"), *completely*, or *fast enough* to maintain the state of health of the tissue/organ involved. Thus, over a period of time (how long?… anybody's guess!), there is an "accumulation" of *microtrauma* that eventually results in total anatomical tissue failure, with resulting pathological consequences.

There are several significant problems that render this type of analogy totally useless in establishing cyclic failure mechanisms for soft biological materials, among them are the following:

(i) the *cumulative trauma*, *fatigue-failure* paradigm for soft anatomical tissues and organs, suffers from a naïve understanding of the structure, function, and biomechanical properties of *nonlinear*, *viscoelastic* materials. The behavior of such materials lies somewhere in-between that of pure *fluids* at one extreme—for which fatigue failure is a totally meaningless concept—and *rigid crystalline solids* at the other extreme—which materials have no relation to soft biological tissues;

(ii) metal clothes hangers and paper clips do not have *nonlinear*, *viscoelastic properties* that can easily tolerate cyclic loading without consequence; they are not *designed* to be subjected to periodic loading with alternating periods of load reversals;
(iii) metal clothes hangers and paper clips cannot *adapt* to changing load conditions, as can *living* biological materials;
(iv) neither can metal clothes hangers or paper clips *heal!*.

Going one step further, the "excessive" biomechanical realm above Region 3, together with the "rarefied" realm below Region 1, are sometimes referred to collectively as

Region 4 (defined by loading that is beyond normal limits). This is the loading region wherein one might justifiably expect significant failure of biological materials—either by *use-it-or-lose-it*, net *atrophy mechanisms* below the threshold for Region 1, or by tensile, compressive, torsional, and so on, *mechanical failure mechanisms* above the upper limit(s) for Region 3. In the end, should material damage due to *exiguous* loading at one extreme of Region 4, or *excessive* loading at the other extreme, become significant enough, there could follow a pathologic, degenerative death (*necrosis*) of the tissue/organ involved, leading to its eventual demise (known as an *infarct* when the demise is associated with compromised blood flow to the organ/tissue involved). The anatomical region of necrosis is sometimes referred to as

Region 5 (resulting in biomechanical loading that is fatal). Fatal biomechanical loading can be sudden and *abrupt*—as is the case for *acute* excessive loading—or, it may be progressive and *degenerative*—as is the case for *chronic* excessive loading. In this region of ultimate tissue demise:

(i) its adaptive capability has been exhausted;
(ii) it has experienced damage beyond its ability to heal;
(iii) it has ceased offering any significant resistance to being loaded and deformed. In other words,
(iv) no longer can the material's nonlinear viscoelastic properties tolerate loading without consequence;
(v) no longer can feedback/feedforward control mechanisms succeed in altering the material's operating set points and/or input/output transfer functions in order to allow it to adapt to the loading to which it is being subjected; and
(vi) regenerative metabolism fails to restore the tissue/organ back to health because its loading exposure falls beyond such capability

. . . the tissue is finally gone!

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