of later stages of the disease) come into view, the **higher** potencies are required.

Generally speaking, diseases characterized by diminished vital action require **lower** potencies, while diseases characterized by increased vital action respond better to **higher** potencies, but this again is modified by the temperament and constitution of the patient.

If the grade of the disease is low and the power of reaction is low, the remedy must be given **low.** Incurable or terminal conditions, when the patient does not respond to well selected remedies, nor to intercurrent reaction remedies, given in potentized form and small doses, require us to resort to the **crude medicine** and increase the quantity of the dose up to the point of reaction: when reasonably sure of the remedy, give the **mother tincture** or a **low trituration,** first in moderate, then in increasing doses until the dosage is found to which the patient will react - the 'maximum dose' may sometimes be the *minimum dose necessary to bring about reaction.* This does not in the least degree violate the principle of the minimum dose *in such cases:* the principle of similars as applied in the selection of *both remedy and dose* is eternally and universally true. It is as true in terminal conditions in chronic diseases marked by gross pathological lesions, as it is in any other kind of case....

Thus the whole matter of the dose resolves itself into a problem of individualization. Looking at this subject broadly and having the highest degree of success in view, it is seen that it is as necessary to individualize the dose as it is the remedy, and ***that the whole scale of potencies must be open to the prescriber!***

We must learn how to judge the degree of susceptibility if we would be successful homeopathic prescribers; and this applies not only to the normal susceptibility of the patient as evidenced by his constitution, temperament, etc. but to the varying degrees of his susceptibility as modified by the character and stages of his disease and by previous treatment. At one stage he may need a low potency, and at another stage, a high potency.

The man who confines himself to the use of a single potency, or two or three potencies, be they low or high, is not availing himself of all the measures of his art *and will frequently fail to cure.*

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Brain Aging

Editor:

Elie Mechnikov, like Pavlov, built a broad understanding of the nature of animals from a basic study of the process of nutrition. He believed that in the development of an individual, and in evolution, there is an early stage in which a division of labor occurs, with some cells being responsible for movement, and other cells (the amoeboid phagocytes) being responsible for ingesting and digesting food. The nutritional function of these latter cells was the basis for building and shaping tissues, and as the complexity of the organism increased, the phagocytes remained central in the preservation of the integrity of the organism.

Some examples will illustrate the ways in which phagocytic white blood cells preserve integrity. In a wound, white blood cells arrive quickly. Foreign matter, such as bacteria, is ingested and destroyed. If the object is too large to ingest, the cells adhere to it, and seal it off. Other white blood cells participate in healing, wound closing, and scar formation. Damaged or virus - infected cells of the organism, or cancer cells, or "old" red blood cells are destroyed and removed. Tissue remodeling - ranging from the tadpole's shrinking tail to the reshaping of bone by osteoclasts - is handled by this sort of cell. (That is, removal, replacement, and alteration of tissue are handled by this system.)

The energy available to the white cells, and the condition of the various tissue cells, govern the processes of phagocytosis, healing, and tissue remodeling. Thyroid hormone and body temperature are important factors governing the activity of white cells. The rate of movement of white cells through the tissue is strongly dependent upon temperature[1] (with the rate increasing with an increase in temperature), and they are generally tolerant of very high temperatures.[2] The conditions governing resorption of the tadpole's tail are probably relevant to how white blood cells function in mammals. Either cold or hypothyroidism tends to suppress or delay the phagocytic activity in the tadpole tail. With an adequate temperature and thyroid hormone, the cells selectively remove the oldest cells first, indicating that they recognize cellular age.

Although Mechnikov believed that the phagocytes were responsible for the atrophy of aging, he also believed that bacterial toxins from the intestine dominated the aging process.

Bacterial endotoxin inhibits mitochondrial respiration, and this respiration is needed for the intramitochondrial conversion of cholesterol into pregnenolone. With aging, pregnenolone and its derivatives, progesterone and DHEA, decline sharply. The brain, the organ with the highest concentration of those stabilizing substances, has many systems for adapting to their decreasing concentration, but the immune system is probably less able to compensate for those aging changes.

The combination of an extreme decrease in concentration of the protective steroids in the brain, and impaired function of the phagocytes, might account for some of the features of Alzheimer's disease. In this disease, microtubules accumulate within nerve cells and other nerve cells die, leaving tangles of their axons, including microtubules. These cells are not removed, as dead cells normally are. Subnormal temperature and hypothyroidism probably contribute to the inertia of the phagocytes. (In mammalian cells, microtubules dissolve at low temperatures, and are stable at higher

temperatures. Phagocytosis seems to depend on a functional microtubule system within the white cells.)

But the reason for the nerve cell's death, and for the tangles of filaments within living cells, seems to be a metabolic problem of the nerve cells themselves. A few years ago, there was talk of a similarity between the "amyloid" deposits, and the blood protein, pre-albumin. Since that protein transports vitamin A and thyroxin, I wondered about the possible effects of a vitamin A deficiency. Vitamin A regulates lysosomes, and so a deficiency might promote the accumulation of intracellular debris. It is an antioxidant, and so a deficiency might tend to induce the stress-hypoxia proteins, and it is used massively in the synthesis of steroids (for example, progesterone supplementation spares vitamin A). But possibly most important is the "de-differentiation" that occurs in many cells in a vitamin A deficiency. In the skin and mucous membranes, a vitamin A deficiency acts like an excess of estrogen, to promote the formation of keratin.

Twenty years ago, English researchers found virus-like shapes in the brains of old people, and suggested that senility might be caused by a virus. Later, one of them found that aged brain tissue was antigenically similar to scrapie-infected brain tissue from a young animal, and antigenically distinct from healthy young tissue.[3]

In size and over-all structure, keratin filaments are similar to the scrapie particles, and to the filaments that accumulate in Alzheimer's disease. I think of keratin as a protein made by a cell which has lost the energy to make more functional proteins. Normally, keratinized cells are formed by rapid cell division at a body surface, where little energy is available. In chronic vitamin A deficiency, the keratin-forming cells divide more rapdily than normal. Dandruff and the moist white equivalent of dandruff on mucous membranes, leucoplakia, are promoted by a deficiency of vitamin A, or an excess of estrogen. Radiation often causes a similar hyperkeratosis, probably by destroying vitamin A, and, like estrogen, by creating a respiratory effect. I suspect that the plaques in Alzheimer's disease are the brain's equivalent to the plaques of leucoplakia, or of dandruff, and that the overgrowth of the glial cells is the result of the same tendency of cells to divide rapidly, while the non-dividing neurons accumulate an excess of fibrous proteins that can't be degraded.

In degenerative diseases, the stress - and age - induced accumulation of iron and other mitochondria-toxic material (e.g., calcium, aluminum, and lipid peroxidation products including age-pigment) and the failure of detoxifying systems make therapy with ordinary nutritional supplements fairly ineffective. Direct supplementation of the various natural protective substances (or their analogues) in addition to the protective vitamins (especially E) and minerals (especially magnesium) is more appropriate.

The supplementation of prenenolone, etc., will allow dietary vitamin A to be spared for other purposes, including regulation of mitosis, differentation, and oxidation.

GABA-related metabolites, such as GHB, butyric acid, succinic acid, and the butyrobetaines, have multiple protective functions, including promotion of respiration and pregnenolone synthesis, regulating gene expression, and reducing damage from glucocorticoids.

DHEA seems to be involved in various regenerative processes, and a deficiency of it is prognostic of increased probability of death from various causes, including cancer, heart disease, and AIDS.

Progesterone has the special status of being an essential nerve growth factor, and generally blocks the catabolic actions of the glucocorticoids and estrogen, thereby protecting all tissues, from brain cells to white blood cells.

Thyroid hormone protects against stress and supports normal differentiation.

Short and medium-chain saturated fatty acids provide a safe source of energy, as well as having hormone-like and adaptogenic effects. The short-chain saturated fatty acids are important in regulating bowel flora. Mechnikov's idea of altering the flora with cultured milk was on the right track, but much more needs to be done on bacterial nutrition and toxin formation.

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More Letters

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