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# On Growth, Form, and Function—A Fantasia on the Design of a Mammal

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*The development of a system's biology, as a common construct for both physiologist and engineer, requires both a theory of structures (form) and a theory of dynamics (function). A dynamic organizing principle—"homeokinesis" for the living system was proposed earlier. Based on thermodynamic reasoning, homeokinesis attempts to capture the physical essence of homeostasis. Now, a primitive foundation is proposed from which a large family of design characteristics might emerge, by self-organization, in complex biological organisms. This foundation is directed at the emergence of major form parameters of the entire class of mammalia, from 3 gm adult shrews to 100,000 kg whales.*

## Introduction

In a prior study a dynamic foundation was proposed for regulating and control functions in the complex biological organism [1].[1] In this study, drawing inspiration from D'Arcy Thompson and Réne Thom, a dynamic functional basis is proposed for organismic form. It is clear, in both systems' start-up and maintenance (for example from materials' turnover studies), that form is maintained by dynamic processes rather than by material cast into passive mold.

But what are the physical processes that might govern form? Some primitive engineering aspects of mammalian design will be sketched (it was first outlined in [2]). The key principle will be an acoustical-hydrodynamic-mechanical forcing from the beating mammalian heart as a regulatory signal for growth, rather than a requirement for a detailed genetic blueprint. Engineers will recognize that an engineering theory of similitude is being cast. As a first attempt, it must perforce be crude. Most biologists will not only consider the paper highly speculative, but inverse in logic. Nevertheless the paper persists in exploring the consequences of the following: If a fetal heart is beating in a gelatinous embryonic media, can this provide an organizing signal for growth?

It is not the premise that organization emerges only from the one dynamic signal of a beating fetal heart, but given its "regulatory" self-organization competence, the "control" task of the genetic code is considerably eased. In particular, it is not known what codes a mammal; but given that a mammal is coded for, with a mammalian heart and a kidney in embryo, its possible causal dynamic consequences are explored.

## Some Known Experimental Results

Experimental correlations derived from mammals [1, 3] provide the following power-law relations:

$$l_a = \frac{V^{0.425}}{1.70} \qquad \Delta V = \frac{V}{900}$$

$$l_a = 21\, d_a \qquad f_0 = \frac{22.7}{V^{0.25}}$$

$$(Q_b)_0 = \frac{V^{0.85}}{109} \qquad A = 10\, V^{2/3}$$

These are major correlations between adult body volume $V$ and cardiovascular parameters—aorta length $l_a$, aorta entrance diameter $d_a$, resting blood flow $(Q_b)_0$, stroke volume $\Delta V$, resting heart rate $f_0$, and animal surface area $A$. Assuming that average body tissue has essentially the density of water, then body volume $V$ is proportional to body weight $W (V = W/\rho)$.

The correlations are derived from a wide range of animals and weights. It is fortunate that the data may be centered on a fairly standardized animal. The studies of Patel, et al. [4] have provided a standard aorta form for a 23 kg dog. The logarithmic midpoint of mammals is very near this weight, and measured performance data for these dogs conform quite well to the entire range and scatter for all mammals. Thus for this "midpoint": $d_a = 2$ cm, $l_a = 42$ cm, $v_0 = 15$ cm (mean blood velocity in aorta at rest), $V = 23{,}000$ gm, $f_0 = 1.84$ bps (= 110 bpm), $(Q_b)_0 = 47$ ml/sec (= 2.8 lpm), $\Delta V = 25.5$ ml.

## Added Vascular Details

Additional topological-geometric details of the vascular sys-

[1] Numbers in brackets designate References at end of paper.

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tem, experimentally derived, are found in [5]. In summary: There is

1 a few degrees decreasing angular taper throughout the arterial system in the direction of flow.

2 a significant branching within about 3 diameters at any arterial point.

3 establishment of "levels" from a branch point to an equal bifurcation.

4 an equal bifurcation within about 20 diameters at any level (e.g., 15–25).

5 about 6 branches per level (e.g., 5–8).

6 conservation of flow area at arterial levels above 1 mm diameter, namely $d_0^2 = \Sigma d_i^2 \approx N d_1^2$ ($d_0$ = an entrance diameter, $d_i$ = branched entrance diameters for the next level, $d_1$ the average entrance diameter of branches, $N$ = the average number of branches); increase of flow area for smaller tubes, namely $d_0^{2.7} = \Sigma d_i^{2.7} \approx N d_1^{2.7}$.

7 an extensional hoop stress propagated via the arterial wall with a velocity (Moens-Korteweg velocity) of about 500 cm/sec in large arteries.

8 essentially undamped waves found in the aorta.

9 a near constant mean resting blood velocity in the aorta entrance of 15 cm/sec.

10 a rubber-like Young's modulus E for tissue of about 50 psi (= 3.5 atm. = $3.5 \times 10^6$ dynes/cm$^2$); a water-like density of 1 gm/cm$^3$.

11 a peak sustainable elastic strain in elongation for tissue of the order of 30 percent. (This is the same value for the stretchiest elastomer known, latex rubber. It is assumed to be a property of highly reliable long chain spiral-like elastomeric configurations, molecules and the like.)

12 a mean arterial pressure of the order of 100 mm Hg (This, it is premised, is the consequences of a mammalian kidney, which is obviously coded for genetically and also appears in embryo. Other phyla may operate a kidney filtration system with 25 mm Hg, the hydrostatic pressure sustainable within the vasculature by the osmotic differential associated only with protein synthesis. The mammalian development has provided a higher pressure design.)

## A Casual Round of Engineering Estimation

**Arterial Hoop Stress ($\sigma_1$).**

$$2\sigma_1 s = p_1 d \qquad \sigma_1 = \frac{p_1}{2}\frac{d}{s}$$

$\sigma_1$ = wall stress $\quad$ $p_1$ = internal pressure (100 mm Hg)

$s$ = wall thickness $\quad$ $d$ = arterial diameter

**Hoop Strain ($\epsilon_1$).**

$$\epsilon_1 = \frac{1}{2}\frac{p_1}{E}\frac{d}{s} \quad \therefore \quad \frac{s}{d} = \frac{1}{2}\frac{p_1}{\epsilon_1 E} = 0.07$$

Thus the wall thickness to diameter ratio emerges out of the tolerable strain.

Some might consider our logic at this point circular. It is not. We ask the question: What if tissue were highly elastomeric, as extensible as any other material we know? We find that this assumption leads to a valid consequence. It does not prove our assumption, just that—by abduction—we are building toward a self consistent model.

**Moens-Korteweg Velocity (C).**

$$\therefore \quad C = \sqrt{\frac{E}{\rho}\frac{s}{d}} = 470 \text{ cm/sec.}$$

Thus the propagation velocity emerges from the tissue modulus to density ratio and the wall thickness to diameter ratio.

**Animal Weight (i.e., Volume).** Tuck the animal into a compact cube. Its length is then of the order of its aorta length $l_a$. Its lateral half-width or half-thickness is of the order of the second level "branches" from the aorta, $l_a/\sqrt{N}$. (Since the periphery must also be reached by blood vessels, and because flow area is conserved. From the latter, it follows that $d_a^2 = N(d_a^2/N)$. But $l$ and $d$ are proportional. Thus, if the first level is of length $l_a$, the second level is of length $l_a/\sqrt{N}$.) With $N$ of the order of 6, the width and thickness, $2l_a/\sqrt{N}$, is of the order of $l_a$ also. Thus

$$V = l_a^3 \quad \therefore \quad l_a = V^{1/3}$$

**Animal Area.** The compact area would be $6\,l_a^2$. Thus

$$A = 6l_a^2 = 6V^{2/3}$$

But because of compact shape, the actual unfolded area—arms, legs, head extended—can be "seen" to be about double. Thus

$$A = 12V^{2/3}$$

**Blood Flow.**

$$(Q_b)_0 = \frac{\pi}{4} d_a^2 v_0 = \frac{\pi}{4}\left(\frac{d_a}{l_a}\right)^2 l_a^2 v_0$$

$$l_a/d_a = 21 \quad \therefore \quad (Q_b)_0 = \frac{V^{2/3}}{37.6}$$

**Heart Beat Frequency.** If the arterial "terminus" is nearly end-stopped at highly resistive levels, it acts as a quarter wave length line

$$f_0 = \frac{C}{4L}$$

Since the undamped levels (> 1 mm) preserve area at bifurcations, their lengths will additively contribute to the quarter wave length (Rayleigh). They form a series—first length = $l_a$, second = $l_a/\sqrt{N}$, third = $l_a/(\sqrt{N})^2$, ..., last = $20 \times 0.1$ cm = 2 cm. (The diameter for undamped waves, as stated previously, is about 1 mm and its terminal length would be about 20 times greater. At this point it is only an assumption that the damping has changed its character in tubes below 1 mm. We have only indicated so far that there is a change in the character of the branching, specifically that mean velocity no longer is preserved, but begins to fall for tubes below 1 mm.) Summing by the geometric series rule of the first term minus the last term over one minus the ratio

$$L = \frac{l_a - 2}{1 - \frac{1}{\sqrt{N}}} = 1.7\,(l_a - 2) \quad \therefore \quad f_0 = \frac{74}{V^{1/3} - 2}$$

Note that the assumption of a change in character (e.g., damping) at about 1 mm arterial diameter suggests a limiting mammalian length of the order of 2 cm. More of this later.

In a crude fashion (but less than an order of magnitude error over eight decades) "armchair" estimates have been obtained, correlating cardiovascular parameters and mammalian weight out of a very simplistic mechanical-acoustic-hydrodynamic model. Thus if the beating mammalian heart and kidney are considered primary "causal" elements, then the mammalian bulk and its supportive perfusional streams of blood, nutrient and oxygen needed to support that bulk can emerge. But there seems to be significant discrepancies in the "geometric" similarity base. Some dynamic modifications are required.

## Preliminaries to a Second Modeling Round—Potential Foundations for Vascular Development

On hydrodynamic reasoning, the fetal heart presumably pro-

vides a pressure-flow pulse to develop a circulation of nutrient. To illustrate, a liquid squirt continually acting within a soggy sand or gel volume would develop a liquid filled circulating cavity and return. The assumption here is that the fluid must be extracted from the gel in the return in order to repump it. Else a much more extended system can be developed if the pumped stream were independently supplied. It is also assumed that there is a directed asymmetry in the squirt.

Somehow the flow resistance developed within the soggy bed will govern the amount of flow in transit and the extent of the quasi-cylindrical or pyramidal domed cavity that develops because of the deformable nature of the gel-like medium, governed at most by a surface tension-like elastic resilience. It is a $\rho v_0^2/2$ stress that is likely formative at the ramified cavity wall (or since the flow is impulsive and triangular [5], it is more like $\rho(6v_0)^2/2$ that presents the pulsing stress). It is plausible that this impulsive signal is responsible for the emerging cavity of length approximately three times its diameter, with a parallel counterflow return.

But any cavity that develops is still extendable as a continuing instability. It is premised that the cavity will branch by further budding. Such unstable branching will continue until a characteristic number of unequal branches bud, before finally an equal bifurcation takes place. (Genetic pressure will dispose the developmental axis to follow a characteristic organ pattern, but that is not the present concern.) An elegant point at which to note that budding competition in development is at the top of the aortic arch, as it differs among various phyla, species, and shows variations from individual to individual (e.g., see Grant's *An Atlas of Anatomy*). More on this point later.

The net effect of fluid pulsing is that propagative phenomena determine the extent of the animal to its further reaches; resistive (viscous) dissipation limits the transport processes out in any neighborhoods reached; and the deformational motional characteristics of the overall body determine the returning venous fluid as counter flow (namely the venous return is favored as cavities limited by venous collapse).

Why growth of the animal? The chemical character of homeostasis, namely essential regulation of chemical concentration, makes chemical flux transport proportional to $v_0$. Thus supply and demand are concordant. But cellular tissue must be at or near (e.g., 30 micrometers) the transport system. Thus cells grow up as the lining of structures along the transport path. In particular, cells first form an elastic lining for the vascular system. They bond electrically. The extent of their growth, is perhaps governed, as shown, by a level of supportable stresses.

But there is one developmental axis different from the other two. Two dimensions can be imagined horizontally. There is a third gravitationally determined axis in the gel. That axis cannot be determined solely by $\rho v_0^2/2$, it must also respond to $\rho gh$. Namely there is a characteristic dimension $h_0 = (6v_0)^2/2g$. Using $v_0 = 15$ cm/sec, $h_0 = 4$ cm. It is suggested that $h_0$ is the peak height of a single pulse of blood for all mammals at rest, per operating stroke. (Not the peak height that would be reached if the heart underwent a complete ejection, but the operative height per resting heart pulse.)

How is this dimension $h_0$ to be incorporated into design? One can imagine that if there was a characteristic horizontal dimension $l_a$ which is governed by whatever resistance limits it, in the gravitational direction the system could not show the dimensional $l_a$ but the harmonic mean height $(l_a h_0)^{1/2}$. Use will be made of this idea later on.

Finally, it should be noted that the self-organizing and maintaining design forces make it impossible that the size of the animal be designed for peak hydrodynamic performance but for average performance. That would suggest that the average performance of the animal is near its rest state rather than its peak state. Thus the critical parameter $v_0$ associated with rest is essentially a design parameter representative of mammalian operation rather than the peak average velocity (e.g., 7 lpm human cardiac output rather than peak levels of 20–25 lpm).

These ideas are used in a second design round.

## A Second Round of Design

**Limiting Mammalian Size (Small Limit).** It is only possible to develop the aorta cavity if the aorta is at no point overdamped. From this, an estimate will be made of the smallest size mammal (or actually the smallest sized aorta). Hydrodynamic studies (Iberall (1950), see [5]) show that a transmission line is overdamped when the damping coefficient $Z$ is less than or equal to 1 ($Z = \alpha^2$, $\alpha$ is Womersley's coefficient).

$$Z = 1 = \frac{r^2\omega}{\nu} = \frac{d_a^2}{4}\frac{2\pi f}{\nu}$$

$\nu$ = kinematic viscosity (= 0.035 poise for blood)
Substitute the "exact" experimental results

$$d_a^2 f = \frac{2}{\pi}\nu = \left(\frac{d_a}{l_a}\right)^2 l_a^2 f = \left(\frac{d_a}{l_a}\right)^2 \frac{V^{0.85}}{1.70^2}\frac{22.7}{V^{0.25}}$$

$$\therefore \quad V^{0.60} = 1.25, \qquad V = 1.5 \text{ ml}, \qquad W = 1.5 \text{ gm}.$$

This, compared with 3 gm shrews, seems to be a very respectable estimate. This and all smaller aortas would be overdamped, and there would be appreciable viscous loss in the aorta. Thus actual adult animals have to be at the next level, about 3 gm in weight.

Thus to have an animal disk-like in cross-section, whose axis is $l_a$ and whose lateral half-span is $l_a/\sqrt{N}$, it is only the second level, which extends to the periphery, that can be dissipative.

Summarizing, the aorta (and first branches) exist for underdamped propagation out to the far reaches of the animal; the heart exists to pump pulses against the viscous losses in channels developed in its gel surround, including one such dimension against gravity; the aorta cavity grows to a diameter at which the elastic cellular lining can tolerate the pulsing hoop stress.

**The Rest Velocity in the Aorta.** It will now be shown how $v_0 = 15$ cm/sec can be predicted. Examine the $Z = 1$ criterion for frequencies in the 10 to 1000 bpm range. It predicts diameters of the tenths of mm to a few mm at most. At these resistive sizes, Poiseuille flow can be assumed. Summing pressure drops across levels

$$\Delta p = 85 \text{ mm Hg} = \Sigma \frac{128}{\pi}\frac{q\mu l}{d^4}$$

$$= 32\mu\left(\frac{l}{d}\right)\Sigma\left[\frac{v_1}{d_1} + \frac{v_2}{d_2} + \ldots + \frac{v_n}{d_n}\right]$$

A drop of 85 mm Hg across the resistive levels was chosen instead of 100 in order to allow for the venous return drop. At these resistive levels, to compute the $v/d$ ratios

$$d_1^{2.7} = N d_2^{2.7} \qquad v_1 d_1^2 = N v_2 d_2^2$$

$$\therefore \frac{v_2/d_2}{v_1/d_1} = N^{\frac{1}{2.7} + \frac{2-2.7}{2.7}} = N^{1/3} = 1.21$$

$$\frac{85}{760} \times 10^6 = 32 \times 0.035 \times 21\left[\frac{v_0}{0.1} + 1.21\left(\frac{v_0}{0.1}\right) + \ldots + \frac{3.2}{35}\frac{v_0}{0.0012}\right]$$

The first term $v_0/0.1$ is the approximate start (1 mm tubes still have near $v_0$ velocity [5]). The ratio 1.21 was computed as above. The last term comes from the universal tree model in [5], namely the last arteriole terminal level, of about 12 mi-

crometers, has an area ratio of 35 to 3.2, where 3.2 is the aorta area. Then

$$4750 = v_0 \frac{76.3 - 10}{1.21 - 1} \quad \therefore \quad v_0 = 15 \text{ cm/sec}$$

a satisfying estimate.

Actually a value for $v_0$ can be estimated level by level from Iberall's empirically derived table [5] as if each level were an aorta (namely for tubes near or above 1 mm).

$$(\Delta p)_{\text{per level}} = 32 \times 0.035 \times 21 \left[ \frac{v_0\sqrt{f}}{\sqrt{0.0223}} \right]$$

since $fd^2 = 0.0223$.

| $d_{av.}$-cm | $\Delta p$-mm Hg | f-bps | $\Delta p/\sqrt{f}$ | $v_0$-cm/sec |
|---|---|---|---|---|
| 0.47[1] | 0.6[1] | 0.101[2] | 1.89 | 15.8[3] |
| 0.13 | 2.3 | 1.33 | 2.00 | 16.7 |
| 0.08 | 3.1 | 3.50 | 1.66 | 13.9 |
| 0.031 | 6.4 | 23.2 | 1.33 | 11.1/0.65 = 17.2[4] |
| | | | | 15 cm/sec.av. |

[1]From table in [5];

[2]From $fd^2 = 0.0223$;

[3]$v_0 = \dfrac{\sqrt{0.0223}}{32 \times 0.035 \times 21} \dfrac{10^6}{760} \dfrac{\Delta p \text{ mm Hg}}{\sqrt{f}} = 8.35 \dfrac{\Delta p \text{ mm Hg}}{\sqrt{f}}$;

[4]the correction 0.65 from $v_2/v_1$ for tubes below 1 mm.

Thus $v_0$ is the velocity at the entrance diameter $d_a$ that is competent to overcome the viscous losses in the dissipative levels. (This is the horizontal design; $g$ has not been involved.)

**A Limiting Size for Mammals (Large Limit).** As a reminder that gravity is a limitation in mammals, a crude model estimate will be provided for a large size limit. No preparation has been made to distinguish the priorities in development of skeleton versus a high pressure kidney versus a thermoregulation mechanism in mammals. Thus the estimte is highly provisional.

The mammal does not unfold as a cube. It follows the aorta axis. Thus it is called upon to "rest" often on an area of $l_a^2/2N$ (i.e., a "sitting" position) rather than $l_a^2\sqrt{N}$. Whether aquatic or not, an exposure which may streamline the shape, this contact area remains a significant cross-section for stress. Thus one can imagine how the peak growth stress is involved in bearing gravitational load. Greater surface stress would "pinch" or ulcerate tissue if maintained.

$$\frac{l_a^2}{2N} \sigma_{\max} = \text{surface force} = \text{Wg}$$

$$\frac{(W/\rho)^{0.85}}{2 \times 1.70^2 N} \frac{p_1}{2} \frac{d}{s} = \text{Wg}$$

$$W^{0.15} = 29.8 \quad \therefore \quad W = 4{,}000{,}000 \text{ kg}$$

Using what will be the subsequent theoretical estimate for $l_a$ versus $W$.

$$l_a^2 = \frac{(W/\rho)^{0.8}}{1.32^2}$$

$$W^{0.2} = 49.3 \quad \therefore \quad W = 290{,}000 \text{ kg.}$$

These estimates may be compared with 120,000 kg observed. (Whales may reach the 90–100 ft length). But the detailed computations are suspect. All that can be said is that stress dependence on $g$ produces large limiting sizes of respectable magnitudes.

**Animal Size.** If $L$ is regarded as a propagational length developed horizontally against diffusional forces in mammals (i.e., against viscosity), then its vertical diffusional-propagational length would be $[(6v_0)^2L/2g]^{1/2}$. Thus

$$V = (l_a)(l_a) \sqrt{\frac{(6v_0)^2}{2g}} l_a = 2.0\, l_a^{5/2} \quad \therefore \quad l_a = \frac{V^{0.4}}{1.32}$$

**Blood Flow.**

$$(Q_b)_0 = \frac{\pi}{4} d_a^2 v_0 = \frac{\pi}{4} \left( \frac{d_a}{l_a} \right)^2 v_0 \frac{V^{0.8}}{1.32^2} \quad \therefore \quad (Q_b)_0 = \frac{V^{0.8}}{65.5}$$

**Stroke Volume.** It is assumed that the first cavity (i.e., up through the aortic arch) has a "height" of the order of $(3 + 1)d_a$ before budding and developing the rest of the arterial system.

$$\Delta V = (d_a)(d_a) \sqrt{\frac{(6v_0)^2}{2g}} (4d_a) = 4d_a^{5/2} \quad \therefore \quad \Delta V = \frac{V}{1000}$$

**Heart Frequency.**

$$f_0 = \frac{(Q_b)_0}{\Delta V} = \frac{15.4}{V^{0.2}}$$

Numerically these results are hardly different from the experimental, and may in fact be better than the "experimental." (For example, with regard to blood flow, the result splits the difference between Klieber's $W^{0.75}$ dependence and Iberall's [3] $W^{0.85}$ dependence. Also $f_0 \alpha V^{-0.2}$ may be better than $V^{-0.25}$.)

**Topological Insight Into Development.** Why there is a curved aorta cannot be fully justified, but some interesting comments can be made. Imagine a bilaterally symmetric channel emerging from the heart pulse, as the aorta does in frog. A curled up fetal form in mammals may result in an asymmetric winner-take-all in the descending aorta. But more to the point, the central cavity extends to the top of the arch. At that point there is branching competition to develop what will be the descending aorta and the right and left common carotids and subclavians. Why the descending aorta wins is not clear at present. But in this modified second round view, the "first" arterial level is this "central" cavity, this first branch. The heart's stroke volume thus grows to fill this cavity. The transmission characteristics develop from this central cavity.

This changes my topological view of the mammals and adds one new level. It also abolishes the design problem of emergence of the developmental axes for head, hands, and feet. In two dimensions the mammal emerges disk-like in which the heart and ascending aorta form the central cavity, with the various sectors leading main branches off the central cavity. The main arterial branches ramify so as to preserve flow area as they extend out to the periphery. Now the geometric deformations of this topology can take place. The disk can be invaginated so as to provide a fetal appearance. Individual sectors can be split off now to provide the isolated snake-like appearance of arms, legs, or head. Any of these may be as long as an aorta, because now they don't differ that much from an aorta. One can now reenter Iberall's generalized arterial tree [5] and add another level, namely the "20–30" branches of the aorta are now more comprehensible. The aorta more nearly makes up two levels, so that an average of 5.6 branches per level becomes more comprehensible throughout.

| Level | No. of branches [5] | New estimate |
|---|---|---|
| 1 | 1 | 1 |
| 2(new) | | 5.6 |
| 3 | 20 | 31 |
| 4 | 260 | 175 |
| 5 | 800 | 980 |
| 6 | 7,000 | 5,500 |
| 7 | 30,000 | 31,000 |
| 8 | 200,000 | 171,000 |
| 9 | 2,000,000 | 960,000 |
| 10 | 8,000,000 | 5,400,000 |
| 11 | 30,000,000 | 30,000,000 |
| 12 | 200,000,000 | 168,000,000 |

## Summary and Discussion

Space will not permit a fuller third round of discussion and justification of these results. Thus they can only be summarized briefly. Fluid pulsing asymmetrically (in space) from a fetal heart, with its pacemaker cells, develops fluid filled channels in a soggy gel. The continued pulsing causes the channels to widen, elongate and bud, whereby both the channels and the budding channels grow. These channels become lined with cells because nutrient is being delivered to this liquid-gel interface. The cells bind elastically by their intercellular "cementing" (Loewenstein). But they are limited in their ability to resist stress. So they grow. By diffusion through their membranous linings, they permit other cells to grow nearby (at distances not appreciably greater than 30 micrometers). Thus the body of connected cells grows out. The response is to the pulsing heart, whose rate governs the size of the arterial system, the size of the animal, and its own growing size and stroke. The scale of stress (i.e., internal pressure) is set by the ingenious design of the mammalian kidney, whose filter-discharge characteristics determine the operating pressure level.

But gravity enters into the picture. As a final theme, we will portray it again. Imagine an animal extended to dimension $l_a$. Its vascular system branches so as to form a disk of approximate area $l_a^2$. But at least a third dimension must discover gravity $g$. This may be viewed as a similitude argument. Let

$$V = f(l_a, g, v_0)$$

Why $v_0$? An animal grows according to a chemical dictum. It has a carrier, blood, distributing "homeostatically" regulated nutrients in some range of characteristic velocities. This is required hydrodynamically. It cannot grow to support its peak average velocity, e.g., 50 cm/sec. Thus it is limited in growth more nearly to its rest velocity, e.g., 15 cm/sec. At this point the decision has not been made as to what limits the velocity $v_0$ to a characteristic value, although it turns out to be design to overcome viscosity. But given a characteristic velocity, not necessarily constant for all mammals until demonstrated, then the suffused volume may be expected to grow to equilibrium in proportion to that $v_0$. It is also not known what the structural dependence on $l_a$ is going to be, $l_a$ being the scale of what can be reached by transport. Thus

$$V = v_0 f(l_a, g) \propto v_0 l_a^a g^b$$

Dimensionally

$$\text{cm} \quad 3 = 1 + a + b \qquad \text{sec} \quad 0 = 1 + 2b$$

$$\therefore \quad b = -1/2, \qquad a = 5/2$$

Thus

$$V = \frac{A l_a^{5/2}}{g^{1/2}} \, v_0$$

This is a gross—macroscopic—design of the animal. It doesn't account for the microscopic processes, e.g., chemical delivery or frictional losses.

Of course if the large scale design were not gravity dependent, the design would have to be formulated differently. And this issue raises very basic questions about developmental changes under low gravity that space will not permit discussing here.

But now it turns out that $v_0$ is an absolute constant for all mammalian sizes because the transport requirements at the microscopic level and the viscous losses are both proportional to velocity. In effect, locally the "designer" no longer knows what mammal he is in at the local tissue level. All animals are a "shrub of shrews." Somewhere above that level, not too far removed in space, there is an essentially universal constant velocity that will supply the local tissue. Thus

$$V = A l_a^{5/2} \sqrt{\frac{v_0^2}{2g}}$$

and the only issue is scale. The choice is made of 6 $v_0$ as the peak velocity during ejection [5].

$$V = l_a^{5/2} \sqrt{\frac{(6v_0)^2}{2g}}$$

Its carving competence, surprisingly—perhaps coincidentally at this point—is remarkably good. The rest of the design tends to follow from a similar view.

It is believed that this scheme proposed lends clues to the principles of self organization in living systems.

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