

Theoretical prediction of a time-at-pressure curve for avoiding pressure sores

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Abstract—A theoretical development of the pressure versus time curve for the onset of pressure sores is presented which is based upon the use of dimensional analysis. Comparison with available experimental data from humans and from dogs and swine indicate that this approach agrees well with experiment and also shows some promise for predicting the effects of skin elastic modulus and skin blood flow on the allowable pressures which can be safely tolerated by specific individuals.

Key words: *dimensional analysis, pressure sores, skin blood flow, skin elastic modulus.*

BACKGROUND

The risk of developing pressure sores arises whenever tissues are exposed to excessive or prolonged mechanical loading. Although healthy, able-bodied individuals are always forewarned of excessive pressures by feelings of local discomfort, and can take the necessary corrective action, this is not the case for spinal cord injured (SCI) patients or for anesthetized patients in surgery. For this reason, both of these groups are at higher risk for pressure sores, as are diabetics and the elderly. For SCI patients, pressure sores represent a major problem

in their rehabilitation, particularly because of the high cost and long time required for healing such sores.

Criteria for determining safe levels of pressures and times of exposure, without risk of developing sores, have been established for animals in which pressure sores were artificially produced (3,6) and for seated humans who were examined for signs of “impending” pressure sores (11). In these and in other investigations (4,5), an inverse relationship was observed between pressure level and time of exposure, but the pathogenesis of pressure sores remains obscure. It is known that the development of pressure sores involves many possible contributing factors, and several hypotheses have been presented (7,8,10).

To this date, the only attempt at a theoretical analysis was made by Reddy, Cochran, and Krouskop (9), who used a cylindrical tissue model combined with Darcy’s law for flow through a porous medium. Their mathematical analysis showed that the time to reduce interstitial fluid volume by 50 percent, at a given applied pressure, corresponds closely to the allowable or safe pressures measured by Reswick and Rodgers (11). It should be noted that the same hyperbolic relationship ($\text{pressure} \times \text{time} = \text{constant}$) can also be obtained by simply assuming that flow rate is proportional to applied pressure, and replacing flow rate by the ratio of decreased volume to time.

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ANALYSIS

The present paper is concerned with a more general analysis of the pressure necessary to produce a pressure sore as a function of the time of exposure. This theoretical development is based on the application of dimensional analysis (2). The problem of pressure sore development represents a classic case of a complex physical problem which lends itself to such a generalized type of analysis. That is, the problem itself is clearly too complex to even contemplate writing a mathematical equation expressing the precise dependence on each of the many variables. But, by simply listing and characterizing the suspected primary variables, we can construct a functional mathematical relationship based on the fundamental observation that any equation relating physical quantities makes no sense unless the dimensions on both sides of the equation have the same units of measure. For example, a time cannot be equal to a distance, nor can a mass be equal to a volume.

In order to apply this technique to the problem of pressure sore development, we shall first assume that there is a definable pressure that will initiate a pressure sore, and that it will depend primarily upon the physical properties of the tissue in question and the blood flow through it, as well as the time of exposure. This statement can be made mathematically in the following form:

$$p_s = f(\rho, Q, E, t) \quad [1]$$

where p_s is the allowable pressure, with the units of force per unit area; $f()$ represents an unknown function of the variables in parentheses; ρ is tissue density, with the units of mass per unit volume; Q is local blood flow, with the units of volume per unit time; E is tissue elastic modulus, with the units of force per unit area; and t is time, with the units of time.

Note that Q refers to the local blood flow *before* the loading is applied. In other words, it represents the amount of blood flow to be obliterated by the application of pressure p_s .

The model is a simple one, with no geometry involved (such as location or shape of underlying bone). The tissue is assumed to be homogeneous, so that its properties can be defined by a single value of E and a single value of ρ . Such a measure of E might be readily obtained by measuring skin dis-

placements under loading applied with a rigid cylindrical indenter, and using the equations of Timoshenko and Goodier (13) for homogeneous materials under such loading.

At this point, in accordance with dimensional analysis, we assume that Equation [1] can be expressed as a product of unknown powers of each of the variables, and we proceed to replace each of the variables by its units in terms of mass, length, and time (noting that force = mass \times acceleration). In so doing, we generate the following relationship:

$$(M/LT^2)^a = k(M/L^3)^b(L^3/T)^c(M/LT^2)^d T^e \quad [2]$$

where M, L, T represent mass, length, and time, respectively, and a, b, c, d, e represent unknown powers or exponents for each of the variables in Equation [1]. Then, by setting up equations for the unknown powers such that the exponent of each of the dimensional variables M, L, T is the same on both sides of Equation [2], that is,

$$\begin{aligned} \text{For } M: & a = b + d \\ \text{For } L: & -a = -3b + 3c - d \\ \text{For } T: & -2a = -c - 2d + e \end{aligned}$$

we can solve these equations simultaneously to find that

$$d = a - b; \quad c = 2b/3; \quad e = -4b/3$$

When we substitute these values into Equation [2], simplify, and translate into the form of Equation [1], we find that the resulting functional relationship can be expressed in the form:

$$p_s/E = f[\rho Q^{2/3}/Et^{4/3}] \quad [3]$$

In other words, the dimensionless pressure p_s/E is a function of only one dimensionless quantity, namely $\rho Q^{2/3}/Et^{4/3}$. Further, since the equation involves only dimensionless quantities, this result is very general and does *not* depend upon such dimensional variables as the size or weight of the subject. This means that if we were to plot the results of a *single* set of experiments in this form, it should apply to *all* subjects. It is customary in engineering practice to determine the nature of the general function f from such a single set of experiments.

In the case of pressure sore experiments, we can use the experimental results of Kosiak (6) for dogs, and those of Reswick and Rodgers (11) for human subjects, as seen in **Figure 1**. However, only contact pressure and time were measured in both of these

AVAILABLE EXPERIMENTAL PRESSURE VERSUS TIME CURVES FOR ANIMALS AND HUMANS.

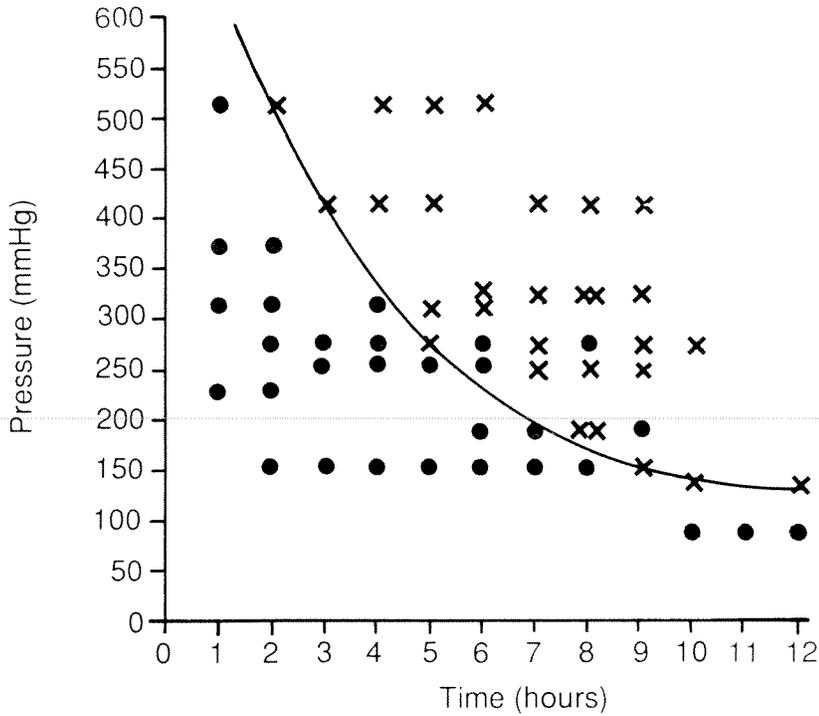


Figure 1a. Pressure versus time for actual skin ulceration in dogs. From Kosiak (6). (x = ulceration; • = no ulceration)

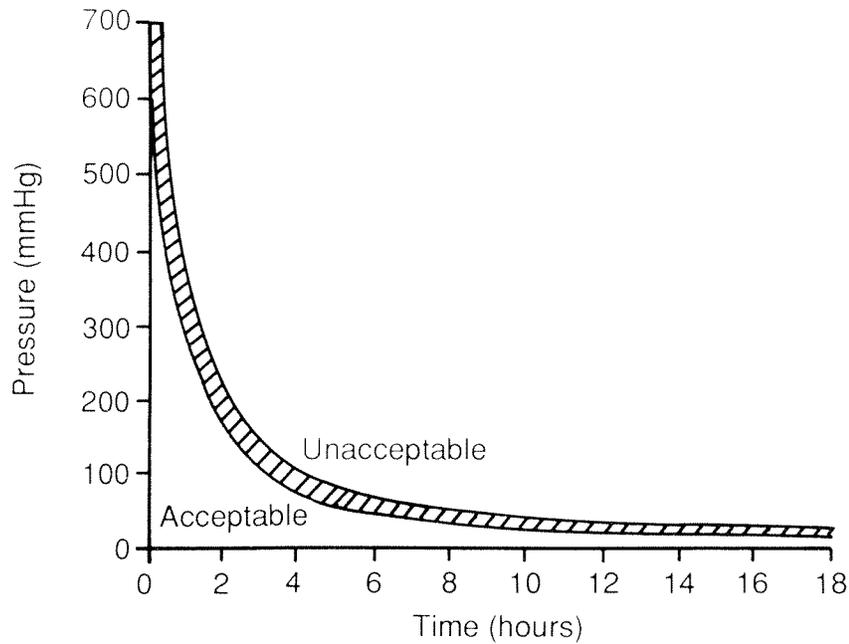


Figure 1b. Pressure for evidence of “impending” pressure sores in seated humans. The two curves are for different humidity conditions at the interface. From Reswick and Rodgers (11).

EXPERIMENTAL DATA REPLOTTED IN THE FORM OF EQUATION [4].

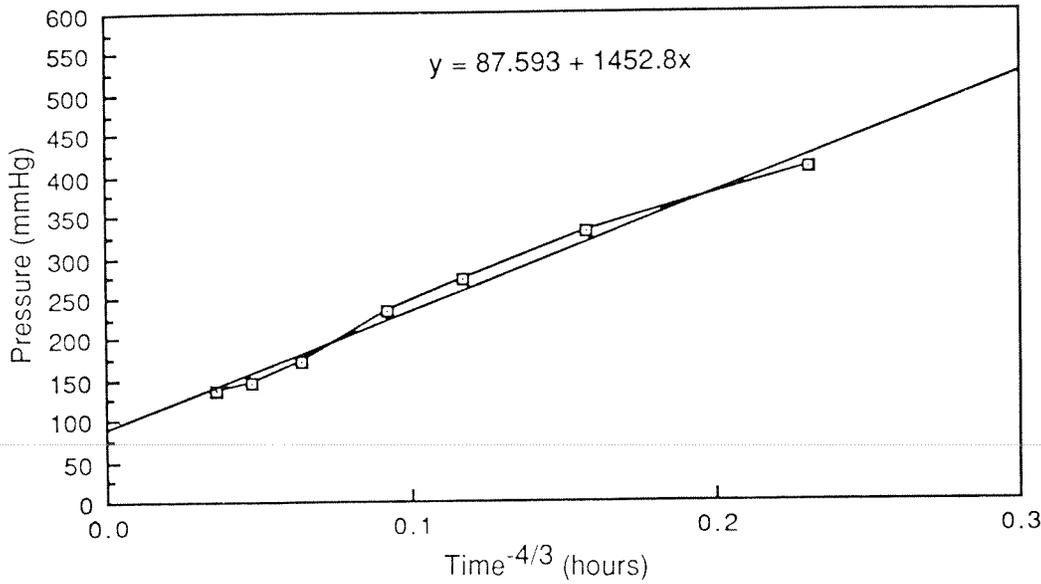


Figure 2a.
Animal data from Kosiak (6).

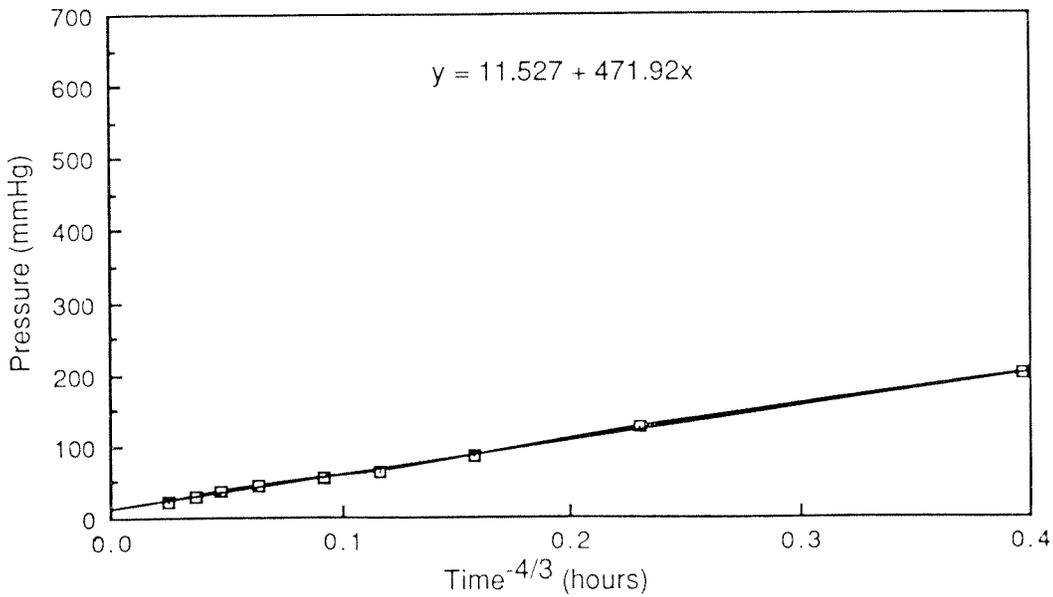


Figure 2b.
Human data from Reswick and Rodgers (11).

and in other experimental studies. Therefore, we shall assume the simplest possible form of the function in Equation [3], namely a straight line relationship, and see how the data compare with the predicted equation. With this assumption, and the

further assumption that E and $\rho Q^{2/3}$ were the same for all subjects tested in each set of experiments, we can write Equation [3] in the form:

$$p_s = A + Bt^{-4/3} \quad [4]$$

If we now replot the data of **Figure 1** in this form (i.e., p_s versus $t^{-4/3}$), the results are shown in **Figure 2** and the agreement is seen to be excellent for both sets of experiments, provided that we select A and B to match each set of experiments. That is, in both cases, the experiments show a linear relationship between p_s and $t^{-4/3}$. This is also the case for the more recent data of Daniel and Wheatley (3) for normal and paraplegic swine (see **Results**).

This finding indicates that the general functional relationship of Equation [3] can be stated more specifically as:

$$p_s/E = K_1 + K_2\rho Q^{2/3}/Et^{4/3} \quad [5]$$

where K_1 and K_2 are “universal” or absolute dimensionless constants. That is, they are the same for *all* experiments. Thus, a general equation for the pressure p_s can be written in the form:

$$p_s = EK_1 + K_2\rho Q^{2/3}t^{-4/3} \quad [6]$$

The quantity E in the above equations represents a “lumped” elastic modulus for the tissue under load and can be determined from the skin displacements produced by loadings applied with a cylindrical indenter, namely (13):

$$E = P(1 - \nu^2)/2rw \quad [7]$$

in which ν represents Poisson’s ratio (approximately 0.5), r is the radius of the indenter, and w is the displacement caused by the load P . Note that this equation holds only for small displacements, and the determination should *not* be made over a bony prominence, since the underlying bone would reduce displacements, thereby adding an apparent “stiffness” to the measurement of E .

RESULTS

It will be noted in **Figure 2** that the experimental data of both Kosiak (6) and Reswick and Rodgers (11), when fitted to the theoretical predictions of Equation [4], indicate a residual value of pressure p_s at $t = \textit{infinity}$ (i.e., at $t^{-4/3} = 0$). Thus, in view of the definitions of the measured pressure in each set of experiments, the intercepts of the two straight lines of **Figure 2** indicate: a) that a pressure of 100 mm Hg will never produce actual ulceration of the skin in dogs; and, b) that 10 mm Hg will not produce even the warning signs of impending ulcer-

ation in humans, regardless of the time of exposure. However, it is important to note the difference in the load distributions for the two sets of experiments, as well as the species difference. Specifically, the animals of Kosiak’s experiments were subjected to concentrated pressure loads transmitted by the head of a syringe plunger, whereas the humans of Reswick and Rodgers’ experiments were subjected to the distributed pressures of normal sitting. These differences are important, partly because of the “hammock effect” or membrane tension developed in the epidermis, and partly because of the significant shear or “rim forces” produced at the edges of the indenter, as pointed out by Bennett and Lee (1).

The data of Kosiak (6) and of Reswick and Rodgers (11) are again shown in their original form in **Figure 3**, where they are compared with the theoretical calculations of Reddy *et al.* (9), and with those of the present analysis. The present analysis evidently gives better agreement with experiment, particularly in the animal experiments of Kosiak (see **Discussion**), and further predicts a finite pressure which can be sustained for indefinite times without giving rise to pressure sores.

The present theory has also been compared with the data of Daniel and Wheatley (3) for normal and paraplegic swine, with similar results. That is, the linear relationship between p and $t^{-4/3}$ was confirmed with correlation coefficients of 0.996, 0.996, and 0.999 for the three conditions studied by those investigators; i.e., for “no tissue damage,” for “muscle damage,” and for “muscle and skin damage.”

DISCUSSION

Note that the present theoretical formulation says nothing about local geometry or distribution of loading. That is, we have not included the effects of contact area, bone depth, pressure distribution, or contact surface conditions such as moisture. The rationale for this apparent omission lies in the assumption that the formation of pressure sores is dependent primarily on tissue properties, irrespective of the type of loading. This is the same rationale that has led to determination of material properties of metals in engineering by testing standard shapes of specimens under measured uniaxial applied loadings to calculate such properties as Young’s modulus

COMPARISON OF EXPERIMENTAL DATA WITH PRESENT ANALYSIS AND WITH ANALYSIS OF REDDY, *ET AL.* (9).

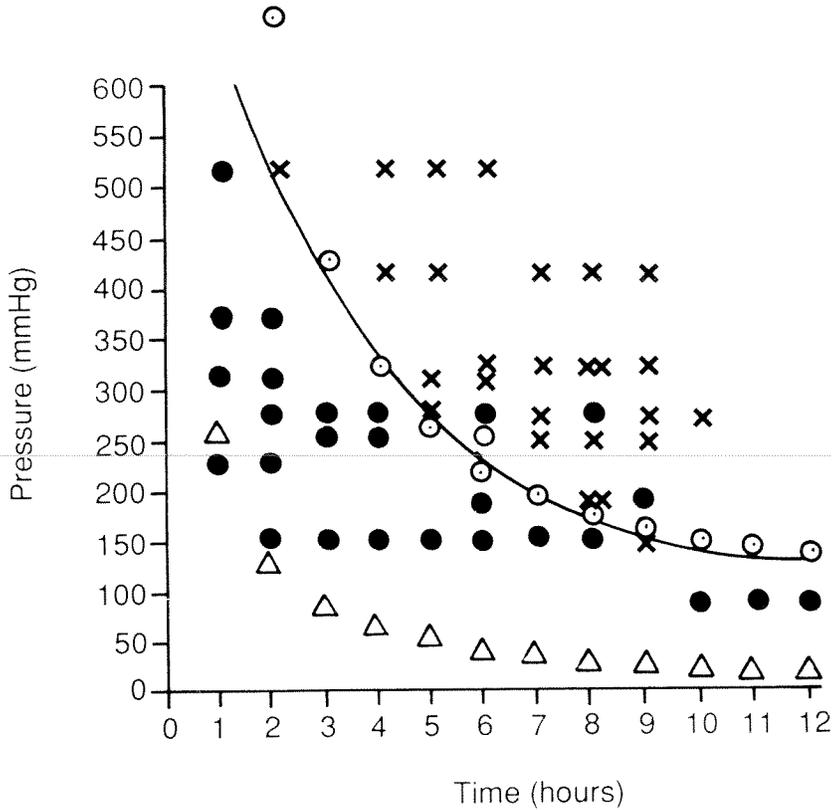


Figure 3a. Data of Kosiak (6) for onset of skin ulceration in dogs. (x = ulceration; • = no ulceration; ⊙ = present theory; Δ = theory of Reddy, *et al.*)

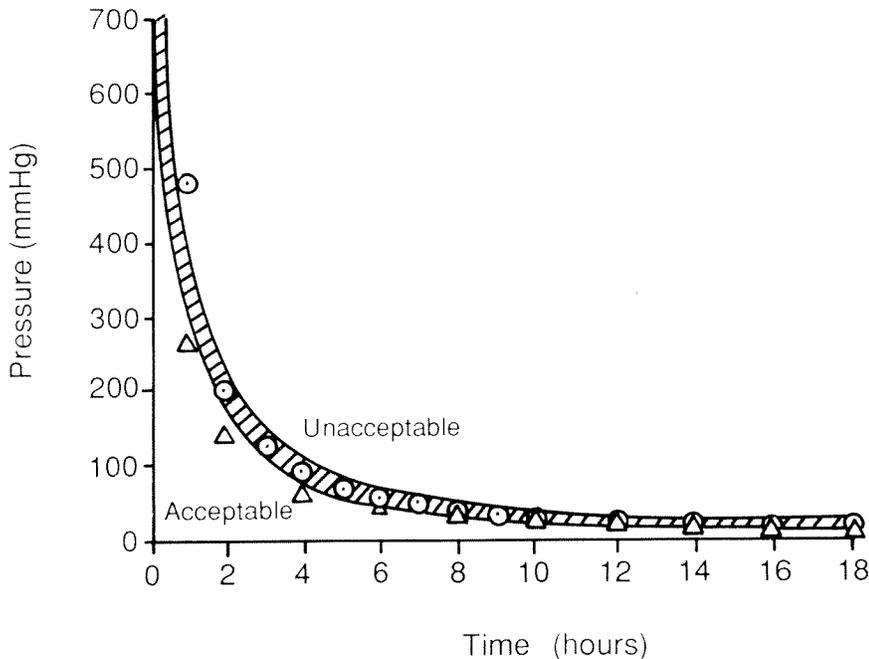


Figure 3b. Data of Reswick and Rodgers (11) for impending pressure sores in seated humans. (⊙ = present theory; Δ = theory of Reddy, *et al.*)

of elasticity and Poisson's ratio of lateral displacement. These properties are then considered to be characteristic of the material itself, irrespective of the loading. Determination of the displacements associated with a particular loading is another matter.

In the physiologic case of pressure sores, it must be realized that material properties of human tissue, including blood flow, will be influenced by many factors, such as nutritional and emotional state, blood pressure, infection, disease conditions, age, and sex. Furthermore, the actual contact pressure encountered in sitting will depend upon bone depth and other anatomical factors as well. But, judging from the comparison of the theory with available experiments, the experiments were evidently influenced primarily by differences in loading distribution and the definition of the measured pressure p_s , which seem to have altered only the constants in Equation [4].

One advantage of the theoretical expression of Equation [6] over the existing experimental data and the theory of Reddy *et al.* (9) is that it gives a method for predicting the effects of such parameters as tissue density, blood flow, and elastic modulus on the acceptable range of pressures that can be tolerated without developing pressure sores. Specifically, we see from Equation [6] that an increase in tissue elastic modulus, blood flow, or tissue density would be expected to increase the seating pressures that can be tolerated. Although tissue density can be expected to remain relatively constant, we might expect decreased elastic modulus and skin blood flow (and hence allowable pressure) with aging, decreased skin blood flow in diabetes, and decreased elastic modulus in spinal cord injury, the latter because of decreased collagen levels (12). But, judging from the quality of the curve fits with the experimental data shown, either these factors were relatively unimportant in each set of data, or else they did not vary within the populations tested. Differences between the values of skin blood flow and elastic modulus for the animals and the humans could be significant, but we cannot tell from the measurements that were made.

It should be pointed out that both the present analysis and that of Reddy *et al.* (9) involve arbitrary constants to establish a "theoretical" curve of pressure versus time. The single constant appearing in the theory of Reddy *et al.* (9) was

calculated based on a 50 percent reduction in interstitial fluid volume, although there appears to be no basis upon which to select that percentage of volume as corresponding to either impending or actual pressure sores. Thus, it is clear from **Figure 3** that the assumption of 50 percent volume reduction does agree with the experiments of Reswick and Rodgers (11) for impending sores in humans, but using the physical constants quoted by Reddy *et al.* (9) would, in fact, require more than a 200 percent reduction in interstitial fluid (clearly impossible) to produce such agreement for actual sores in the animal experiments of Kosiak (6) at 150 mm Hg.

To make the best use of the present analysis, it would seem that a repeat of the experiments of Reswick and Rodgers (11), which include measurements of initial blood flow and elastic modulus, would be most useful in that it would give specific values for the universal constants K_1 and K_2 of Equation [6]. This would then furnish the information needed to generate the general dimensionless Equation [5] to be plotted in the form of p_s/E versus $\rho Q^{2/3}/Et^{4/3}$ (a straight line) which could be applied to specific cases where E and Q are known in order to determine allowable pressures for avoiding pressure sores. Prediction of allowable pressures for a specific individual by using such a plot would then require the measurement of local blood flow (with no loading) and elastic modulus for that individual. A "lumped" elastic modulus for this purpose can be obtained by measuring small skin displacements under loads applied with a cylindrical indenter and using Equation [7] to calculate E . However, since collagen loss is more likely to cause elastic changes at higher deformations (when collagen fibers are extended), it may be more practical to use some alternate (albeit less rigorous) definition of the lumped parameter E , such as the pressure required using a spherical indenter to produce a specified larger indentation, expressed as percent of indenter diameter.

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