Application of the Systolic Stiffness Concept to Assess Myocardial Function in Developing Hypertension

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SUMMARY

The concept of end-systolic myocardial stiffness permits the quantification of preload effects on fiber shortening and changes in the slope (max $E_{sv}$) of the end-systolic stress-strain relation, which, if linear, reflect changes in the inotropic state. As an application of this new concept, the end-systolic stress-strain and shortening-afterload relations were evaluated on the basis of data from dogs studied during development of perinephritic hypertension. End-systolic stress-strain relations were linear before and 2 weeks after the induction of hypertension and the end-systolic pressure-diameter relations were not always linear. The shortening-afterload relations obtained directly from raw data points displayed enhanced contractility in the hypertensive state under beta-adrenergic receptor blockade. However, the preload-corrected shortening-afterload relations demonstrated that contractility was unchanged in hypertension. Hypertensive hearts operated at higher preload than normotensive hearts at any afterload levels. This discrepancy between the conventional method without preload-correction and the preload-corrected analysis indicates the importance of preload-correction on shortening-afterload relations in hypertension.

Additional Indexing Words:

Systolic myocardial stiffness concept  End-systolic stress-strain relations  Fiber shortening-afterload relations in canine heart  Preload and afterload corrections  Perinephritic hypertension  Ventricular function
OVER the past two decades, many indices of myocardial contractility have been developed on the basis of the force-velocity concept and have been discussed in great detail.1)-6) Generally, the isovolumic indices display a preload dependence at low preloads,5),7) while the ejection phase indices exhibit both a preload and an afterload dependence.2) In particular, fiber shortening and the velocity of fiber shortening, widely used in the assessment of myocardial contractility, have a marked dependence on preload.2),5) For the past 15 years, the end-systolic pressure-volume relationship has also been applied to assess ventricular contractility.8)-10) On the basis of the Suga-Sagawa ventricular elastance concept,8) a new concept of systolic myocardial stiffness has been developed.11) This new concept enables one to obtain the entire ejection fraction-afterload relationship or shortening-afterload relationship for a given preload, thus providing a more sensitive method for comparing myocardial contractile states between ventricles. This analysis requires the simultaneous measurement of LV pressures and dimensions. However, this analysis has not yet been applied to the development of a disease state involving myocardial hypertrophy. Accordingly, the main goal was to utilize the new concept of systolic myocardial stiffness in assessing myocardial function during the initial development of hypertension, a state characterized by mild hypertrophy and enhanced sympathetic tone with normal systolic function under beta-adrenergic receptor blockade.3)

Based on LV pressure and ultrasonic crystal short-axis measurements, the purposes of the present study were (1) to validate the hypothesis that the end-systolic stress-strain relation is linear and that the slope (max Eav) of this relationship may be employed as an index for assessing changes in the inotropic state in the early development of perinephritic hypertension, (2) to develop the entire shortening-afterload relationship for any given preload and to compare it with conventional shortening afterload relationships that are not preload corrected and (3) to examine the nonlinearity of the end-systolic pressure diameter relation.

**TERMINOLOGY**

Since there is little consensus on the definitions of many terms employed in cardiac mechanics, a summary of terms is included here.

**Stress difference (σ, g/cm²):**

Total stress difference $\sigma_v = \sigma_{rc} - \sigma_{re}$ is the difference of the average circumferential ($\sigma_{rc}$) and radial ($\sigma_{re}$) stress components,12) assuming that the LV may be represented by a cylindrical annulus at the site where the short-axis
measurements are made.

Strain difference ($\varepsilon_e$, dimensionless):
Associated strain difference, $\varepsilon_e = e_{eC} - e_{re}$, is the difference of the endocardial circumferential ($e_{eC}$) and radial ($e_{re} = -e_{eC}$) strain components in the cylindrical annulus.\textsuperscript{12)

Circumferential endocardial natural strain ($e_{eC}$, dimensionless):
$e_{eC} = \log(D/D_0)$, where $D$ is the instantaneous endocardial short-axis diameter, $D_0$ is the endocardial short-axis diameter at zero systolic stress, and the natural logarithm is employed.

Average systolic myocardial stiffness ($E_{av}$, g/cm²):
$E_{av} = \sigma_e/e_c = \sigma_e/(Ke_{eC})$ where $K = 4/3$ for a cylindrical model.\textsuperscript{12)

End-systole:
Time at which systolic myocardial stiffness attains its maximum value ($E_{av}$)$_{es}$, where the subscript “es” denotes the end-systolic state.

Preload, (($\sigma_{eC}$)$_{ed}$, g/cm²):
Preload is defined as the average circumferential stress in the cylindrical annulus at end-diastole; end-diastole was measured immediately prior to ventricular contraction.

Afterload, (($\sigma_{aft}$), g/cm²):
The average circumferential stress at end-systole ($\sigma_{aft} = (\sigma_{eC})_{es}$).

Endocardial shortening, ($S$, %):
$S(\%) = 100 \times (D_{ed} - D_{es})/D_{ed}$ where $D_{ed}$ and $D_{es}$ are respectively the endocardial short-axis diameters at end-diastole and end-systole.

METHODS

Experimental considerations
The data from 5 mongrel dogs were collected in a previous study on the effects of perinephritic hypertension.\textsuperscript{3) The instrumentation, model of perinephritic hypertension and protocols have been previously described in detail.\textsuperscript{3) Briefly, under anesthesia with sodium pentobarbital, the dogs were instrumented with ultrasonic crystals to measure LV short-axis diameter and wall thickness, a solid state miniature LV pressure transducer (P22 Konigs-
berg Instruments, Pasadena, CA) and an aortic catheter for calibration of the miniature pressure transducer. Perinephritic hypertension was induced by wrapping one kidney and performing a contralateral nephrectomy after the completion of baseline studies. All dogs were studied 2 weeks after induction of perinephritic hypertension. Postmortem data were compared with 6 sham-operated (unilateral nephrectomy) dogs. The alteration of loading conditions was performed by a graded infusion of phenylephrine (1, 2 and 5 µg/kg/min). Beta-adrenergic receptor blockade (2 mg/kg propranolol) was used to eliminate the sympathetic enhancement of contractility in the hypertensive state\(^3\) and the direct inotropic effect of phenylephrine.\(^{13}\)

Data analysis

The data were recorded on a multichannel tape recorder (Honeywell 101, Denver, CO) and played back on a direct-writing oscillograph (Gould-Brush Mark 200, Cleveland, OH). Left ventricular end-diastolic dimensions were measured immediately prior to the onset of ventricular contraction and the LV end-systolic dimensions were measured at the time of maximum systolic myocardial stiffness. The analog signals of LV pressure, short axis and wall thickness were then digitized using an IBM PC/AT computer. The sampling interval on the IBM PC/AT was 2.5 ms. This computer was used to calculate the instantaneous LV stresses, systolic myocardial stiffness, and the end-systolic stress-strain relations and shortening-afterload relations which are described in the next section in more detail.

Statistical analysis was performed using SPSS\(^{14}\) on the IBM PC/AT computer. The data are reported as mean±standard deviation. A paired t-test was used to determine whether there were differences between the values before and after the induction of perinephritic hypertension. An unpaired t-test was used to test for differences in postmortem data between hypertensive animals and sham-operated animals. Differences were considered significant at p<0.05.\(^{15}\)

Theoretical considerations

Expressions for average stress difference, preload, and afterload:

The average stress difference \(a_c\) is given by (Appendix 1):

\[
a_c = \sigma_{sc} - \sigma_{se} = 1.36 \times [PD/2h + PD/2(D+h)]
= \sigma_{sc}(1 + h/(D+h))
\]  

(1)

where \(\sigma_{sc}=1.36(PD/2h)\) is the average circumferential stress, \(P\) is the LV pressure and \(D, h\) are respectively the short-axis diameter and wall thickness. Note that the units of stress are g/cm\(^2\) and \(P\) is in mmHg.
Preload is defined as follows;

\[(\sigma_{pe})_{ed} = 1.36 \times P_{es}(D/2h)_{ed}\]  \(2\)

where the subscript "ed" denotes end-diastole. In this study, afterload \((\sigma_{alt})\) is given by

\[\sigma_{alt} = (\sigma_{re})_{es} = 1.36 \times P_{es}(D/2h)_{es}\]  \(3\)

where the subscript "es" denotes end-systole.

**End-systolic stress-strain relation:**
A modification of a method developed by Sunagawa et al\(^{16}\) was used to calculate the maximum systolic stiffness \((E_{av})_{es}\);

\[(E_{av})_{es} = \max \left[\sigma_{es}/(Kc_{es})\right] = \max \left[\sigma_{es}/((4/3)\log(D/D_0))\right],\]  \(4\)

was evaluated iteratively in the following manner:

1) A value for \(D_0\) was first assumed. For each intervention, \(E_{av}\) was evaluated from the onset of systole until it attained a maximum. By definition, this time is end-systole.

2) The stress \(\sigma_{ces}\) vs \(\log D_{es}\) points at these maxima were then plotted, and a new value for \(D_0\) was obtained by appropriate extrapolation to zero systolic stress. Specifically, both linear and second degree polynomial regressions were performed, and tests of significance of departure from linearity\(^{15}\) were conducted by F test.

3) This iterative procedure was continued until \(D_0\) converged to a constant value. If, in addition, the maximum systolic myocardial stiffness \((E_{av})_{es}\) is independent of the end-systolic stress \(\sigma_{ces}\), this implies linearity of the end-systolic stress-strain relation \((\sigma_{ces} vs \varepsilon_{es})\), which is represented in the form

\[\sigma_{ces} = \max E_{av} \times \varepsilon_{es}\]  \(5\)

where \(\varepsilon_{es} = 4/3 \log (D_{es}/D_0)\). Thus, \(E_{av}\) is the slope of the linear stress-strain relation (equation 5) and is analogous to \(E_{es}\) (the slope of the end-systolic pressure-volume relation) based on the Suga-Sagawa maximum ventricular elastance concept.\(^{10}\) Two to three beats were analyzed for each afterload level obtained by graded infusion of phenylephrine (1, 2 and 5 \(\mu\)g/kg/min).

**Shortening-afterload relation:**
The shortening-afterload relation \((S vs \sigma_{alt})\) (derived in Appendix 2) is given by:

\[S = 1 - (D_0/D_{es}) \times \exp \left[ (A + B\sigma_{str})/(4/3 \max E_{av}) \right]\]  \(6\)
where $S$ = endocardial shortening and all other quantities have been previously defined.

*End-systolic pressure-diameter relation:*

The end-systolic pressure-diameter relation ($P_{es} - D_{es}$) is plotted using raw data. The linearity of the relation is evaluated by F tests between linear regression and second degree polynomial regression. The end-systolic pressure-diameter relation is also derived theoretically from the end-systolic stress-strain relation (Appendix 3) and is given by

$$P_{es} = [(4/3) \max E_{av}/(\alpha + \beta D_{es})] \times \log (D_{es}/D_o) \quad (7)$$

**RESULTS**

*Effects of hypertension on cardiac chamber weight (Table I):*

In this study, mild LV hypertrophy was observed. The LV weight/body weight ratio increased ($p<0.05$) by 22% in the dogs with hypertension when compared with the group of sham-operated controls. There were no significant differences in body weight or right ventricular weight/body weight ratio.

*Effects of hypertension on LV dimensions (Table II):*

The left ventricular end-diastolic endocardial short axis ($D_{ed}$) increased with hypertension ($p<0.05$). However, the LV end-systolic endocardial short axis ($D_{es}$) was unaltered. The left ventricular endocardial short axis ($D_o$) at zero systolic stress and the LV short axis ($D_{ed}(60)$) at the common diastolic stress of 60 g/cm² were unaltered after induction of hypertension.

*Linearity of the end-systolic stress-strain relations (Fig. 1):*

Figure 1 displays the end-systolic stress $\sigma_{ces}$ vs log $D_{es}$ relations for one of the experiments (dog #1) in the normotensive (A) and hypertensive (B)
Table II. LV Dimensions Before and 2 Weeks After Induction of Hypertension

<table>
<thead>
<tr>
<th></th>
<th>Normotensive</th>
<th>Hypertensive</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV end-diastolic endocardial short axis, D_{ed} (mm)</td>
<td>38.9 ± 2.1</td>
<td>41.2 ± 2.5*</td>
</tr>
<tr>
<td>LV end-systolic endocardial short axis, D_{es} (mm)</td>
<td>29.8 ± 2.0</td>
<td>30.3 ± 1.3</td>
</tr>
<tr>
<td>LV endocardial short axis at zero systolic stress, D_o (mm)</td>
<td>26.0 ± 2.3</td>
<td>26.8 ± 2.3</td>
</tr>
<tr>
<td>LV endocardial short axis at diastolic stress of 60g/cm², D_{es}(60) (mm)</td>
<td>40.9 ± 1.7</td>
<td>41.8 ± 2.4</td>
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* Different from normotensive, p<0.05.

Fig. 1. End-systolic stress difference vs log D_{es} and associated stress-strain relations in the normotensive and hypertensive states for dog #1. Afterload was altered from baseline by phenylephrine (PE) doses of 1, 2 and 5 µg/kg/min. The end-systolic stress difference vs log D_{es} relations in normotensive (A) and hypertensive (B) states are linear. Extrapolation yielded the endocardial diameter D_o at zero systolic stress. The end-systolic stress-strain relations (C) in both states were obtained from the relations in panels A and B with D_o as the reference length. The linearity of these relations implies that the slope max E_{av} is independent of end-systolic stress (σ_{es}) and, hence, afterload (σ_{aft}). There was no significant difference between the slopes in the normotensive and hypertensive states. The end-systolic strain is defined by \( \varepsilon_{es} = (4/3) \log (D_{es}/D_o) \). See text for details.

states. F-tests\(^{15} \) indicated that these relationships did not depart from linearity. Extrapolation of these relationships yielded the endocardial
Table III. Max E$_{av}$, Preload and Afterload Data Based on a Cylindrical Model

<table>
<thead>
<tr>
<th></th>
<th>Normotensive</th>
<th>Hypertensive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slope of the end-systolic $\sigma_{es}$-$\varepsilon_{es}$ relation (max E$_{av}$), g/cm$^2$</td>
<td>1781±469</td>
<td>2118±251</td>
</tr>
<tr>
<td>End-diastolic stress ($\sigma_{ed(60)}$), g/cm$^2$</td>
<td>17.6±7.6</td>
<td>39.5±11.0**</td>
</tr>
<tr>
<td>End-systolic stress ($\sigma_{es(1)}$), g/cm$^2$</td>
<td>162±17</td>
<td>187±16*</td>
</tr>
</tbody>
</table>

Different from normotensive, * p<0.05, ** p<0.01.

diameter D$_{0}$ at zero systolic stress and hence the associated end-systolic stress-strain relations (Fig. 1C).

The slope (max E$_{av}$) of the end-systolic stress-strain relation. An index for assessing changes in myocardial contractility (Table III):

The slope (max E$_{av}$) of the end-systolic stress-strain relation ($\sigma_{es}$ vs $\varepsilon_{es}$) may be viewed as the myocardial counterpart to E$_{es}$ (slope of the end-systolic pressure-volume relation), which has been employed to assess changes in ventricular contractility.$^{5,8-10}$ In particular, if there is no significant difference in D$_{0}$ following certain interventions, max E$_{av}$ may be employed as an index for assessing changes in the myocardial contractile state. In this study, max E$_{av}$ was unaltered in hypertensives compared with normotensives (Table III).

Endocardial shortening-afterload relationships (Figs. 2 and 3):

Employing equation 6, preload-corrected shortening-afterload relationships (S vs $\sigma_{ad}$) were evaluated for all dogs in the normotensive and hypertensive states at an end-diastolic diameter (D$_{ed(60)}$) corresponding to a common end-diastolic stress $\sigma_{ed}=60$ g/cm$^2$. This value of $\sigma_{ed}$ was obtained either directly from the raw data or from curve-fits of $\sigma_{ed}$ vs D$_{ed}$. Figure 2 displays the endocardial shortening-afterload relationships for one animal (dog #1) in the normotensive and hypertensive states. The upper panel (A) is a conventional direct plot of raw data points without preload-correction. The lower panel shows the preload-corrected shortening-afterload relationships at a common preload of 60 g/cm$^2$. This figure indicates that the animal had greater shortening in a hypertensive state at any given afterload, but that preload-correction of shortening reduced this difference.

Since endocardial shortening also depends on afterload, comparisons
Fig. 2. Endocardial shortening vs afterload relationships in the normotensive and hypertensive states for dog #1. The upper panel (A) is a conventional direct plot of raw data points without the correction for preload. The lower panel (B) shows the preload-corrected shortening-afterload relationships at a common preload of 60 g/cm². Note that preload correction reduced the difference in shortening between the normotensive and hypertensive states at common afterloads.

of the contractile states between ventricles must be made at common levels of afterload. The degrees of shortening at given afterload levels were obtained for each animal by linear regression of the raw data without preload correction, and by equation 6 employing a preload-corrected analysis. At afterload levels of 200, 250, 300 g/cm², there were significant differences in shortening (S) between the normotensive and hypertensive groups before preload correction (Fig. 3A). However, no significant differences in S were observed between these 2 groups when corrections were made for preload (Fig. 3B).

End-systolic pressure-diameter relation:
Comparisons between linear regression and second degree polynomial regression of pressure-diameter relations showed significant curvilinearity in 2 dogs in the normotensive state and 2 different dogs in the hypertensive state. Figure 4 displays the end-systolic pressure-diameter relations in the
Fig. 3. Comparisons of contractile states between normotensive and hypertensive states at common levels of afterload of 200, 250, 300 g/cm². Shortening in each dog at each afterload level was obtained by linear regression of raw data points (conventional method (A)) and by equation 6 using preload-corrected analysis (B). Mean±SD, * p<0.05, ** p<0.01 vs normotensive.

Fig. 4. End-systolic pressure-diameter relations in dog #1 in the normotensive (A) and hypertensive (B) state. Solid and open squares correspond to raw pressure-diameter data points. Solid curves on the figure are mathematically derived from linear stress-strain relationships employing equation 7 in text.
same dog as in Fig. 1. An F test on the pressure-diameter data points of this dog showed significant curvilinearity in the hypertensive state. The solid curves in Fig. 4 are the calculated relationships based on equation 7.

**DISCUSSION**

Shortening-afterload relationships have been widely used in both experimental and clinical settings for assessing the myocardial contractile state, with only mild success due to the preload dependence of shortening. The present study not only provides a method for preload-correction of shortening, but enables one to describe the entire shortening-afterload relationship. These relationships have the advantage in that myocardial contractility can be quantified and comparisons of contractile states between ventricles can be made over wide ranges of afterload. Such comparisons cannot be conducted with current methods, since the shortening-afterload relations are generally evaluated on the basis of single points obtained from a number of animals or patients, and furthermore, are uncorrected for preload.

The linearity of the end-systolic stress-strain relations has been demonstrated in normotensive animals and has now been demonstrated in hypertensive animals. This linearity is an important and practical result since it implies that the slope (max Eav) is load independent and therefore may be useful for assessing changes in the inotropic state following certain drug interventions. Since systolic myocardial stiffness depends on the diameter at zero systolic stress (i.e. Do), this index can only be used to assess changes in the inotropic state if changes in Do are statistically insignificant following such interventions. It is primarily for this reason that max Eav cannot be employed for comparisons of myocardial contractility between different ventricles, giving added importance to the shortening-afterload relationship.

The conventional method without preload-correction yielded the result that contractility was enhanced in the hypertensive state, even in the presence of beta-adrenergic receptor blockade (Fig. 3A). However, when preload correction was made, contractility was shown to be unaltered in the hypertensive state (Fig. 3B). Figure 5 may explain one of the reasons why this particular study showed a discrepancy with the results of the conventional method. This figure displays the afterload-preload relations of pooled data points for all dogs in both normotensive and hypertensive states. In the normotensive state, afterload had a highly linear correlation with preload even among different ventricles. In situations where afterload is linearly dependent on preload, such as in the normotensive state in this study, the conventional method of shortening-afterload analysis should
yield a result qualitatively similar to the preload-corrected analysis. In the hypertensive state, although afterload is also linearly dependent on preload, the left ventricles operated at a relatively higher preload than in the normotensive state. This particular loading condition may explain the observation of enhanced contractility in hypertensives by conventional analysis.²)

In most previous studies, the end-systolic pressure-volume (ESPVR) and pressure-diameter (ESPDR) relations have been assumed to be linear. More recently, nonlinearity of the ESPVR has been demonstrated.⁴,¹¹,¹⁷) The present study confirms nonlinearity of the ESPDR, a result also obtained recently by Takeda et al.¹⁸) Thus, caution must be exercised when employing slopes of such relationships to assess changes in the inotropic state.

There are some limitations to the present study, which include 1) estimations of \( D_0 \) by extrapolation and 2) the assumption of cylindrical geometry for the LV at the site where the measurements are made. Future studies should therefore include measurements at very low afterload, and in addition, long-axis measurements should be made to confirm the results based on the cylindrical model.

In summary, there are three primary findings in the present investigation. First, the end-systolic stress-strain relationships are linear in mild hypertrophy. Second, the shortening-afterload relationships corrected for preload, indicate that myocardial contractility is unchanged in the early development of hypertension under the blockade of beta-adrenergic activity, in contrast to the result of enhanced contractility as predicted by conven-
tional methods currently employed. Finally, end-systolic pressure-diameter relations are not always linear, thus limiting the use of slopes of such relations for assessing changes in ventricular contractility.

Appendix 1

Expressions for average stress difference, preload and afterload:
We assume that the LV can be represented by a cylindrical annulus at the site where the short-axis measurements are made. Thus at any given radius r, the circumferential ($\sigma_C$) and radial ($\sigma_r$) stress components in this annulus are given by:

$$\sigma_C = 1.36 \times \frac{P \times a^2(1+b^2/r^2)/((b^2-a^2)}{b^2-a^2}$$

$$\sigma_r = 1.36 \times \frac{P \times a^2(1-b^2/r^2)/((b^2-a^2)}{b^2-a^2}$$  \hspace{1cm} (1.1)

where $P$ is the LV cavity pressure, and $a$ and $b$ are respectively the inner and outer radii. Note that $a=D/2$; $b=D/2+h$ where $D=LV$ short-axis diameter and $h=LV$ wall thickness.

The integrated mean circumferential and radial stresses are therefore expressed as:

$$\sigma_{C} = \int_{a}^{b} \sigma_C \, dr/(b-a) = 1.36 \times (PD/2h)$$

$$\sigma_{rC} = \int_{a}^{b} \sigma_r \, dr/(b-a) = -1.36 \times PD/2(D+h)$$  \hspace{1cm} (1.2)

hence the stress difference $\sigma_S$ is:

$$\sigma_S = \sigma_{sc}-\sigma_{rc} = 1.36 \times [(PD/2h)+PD/2(D+h)]$$

$$\sigma_S = 1.36 \times (PD/2h)[1+h/(D+h)] = \sigma_{rc}(1+h/(D+h))$$  \hspace{1cm} (1.3)

Preload and afterload are defined respectively by:

$$(\sigma_{sc})_{ed} = 1.36 \times (PD/2h)_{ed}; \quad (\sigma_{sc})_{es} = 1.36 \times (PD/2h)_{es}$$  \hspace{1cm} (1.4)

where ed and es refer to end-diastole and end-systole, respectively.

Appendix 2

Development of the shortening-afterload relationship:
The equations used to determine the shortening-afterload relation ($S$-$\sigma_{ aft}$) are:

$$\sigma_{css} = (4/3) \times \max \log (D_{es}/D_{o})$$  \hspace{1cm} (2.1)

$$S = (D_{ed}-D_{es})/D_{ed}$$  \hspace{1cm} (2.2)

$$\sigma_{css} = A + B\sigma_{css} = A + B\sigma_{aft}$$  \hspace{1cm} (2.3)

where $A$ and $B$ are regression constants obtained by linear regression.

Elimination of $D_{es}$ and $\sigma_{css}$ from the above equations yields the complex relationship between $S$ and afterload $\sigma_{aft}=(\sigma_{ec})_{es}$ namely,
Appendix 3

Derivation of the end-systolic pressure-diameter relation:

From the end-systolic stress-strain relation (equation 5 in the text) we have:

\[ \sigma_{es} = \max E_{av} \times \varepsilon_{es} = \left(\frac{4}{3}\right) \max E_{av} \times \log \left(\frac{D_{es}}{D_0}\right) \]  

(3.1)

Curve-fitting the ratio \( \sigma_{es}/P_{es} \) in the form \( \sigma_{es}/P_{es} = \alpha + \beta D_{es} \) we obtain

\[ P_{es} = \sigma_{es}/(\alpha + \beta D_{es}) \]

\[ = \left[ \left(\frac{4}{3}\right) \max E_{av}/(\alpha + \beta D_{es}) \right] \times \log \left(\frac{D_{es}}{D_0}\right) \]  

(3.2)

REFERENCES