SEMI-QUANTITATIVE ESTIMATION OF THE MEAN SYSTEMIC PRESSURE IN HUMANS, A PRELIMINARY REPORT

Senri Hirakawa*, Akira Sakai**, and Toshio Yaginuma***

The mean systemic pressure was estimated indirectly in humans from cardiac index and right atrial pressure under a set of obviously over-simplifying assumptions that (1) "normal" venous return curve for mean systemic pressure (Pms) of 7 mmHg gives, by definition, a venous return of 3.8 lit.min⁻¹M⁻² at zero right atrial pressure and (2) different human subjects have venous return curves with downslopes parallel to this "normal" venous return curve. Mean systemic pressure estimated in this manner was termed the graphically estimated mean systemic pressure (Pms.g). Pms.g. was examined for its numerical relationship with the cubital venous pressure (VP). In 38 pairs of comparison which was possible in 36 patients with or without heart diseases and congestive heart failure, Pms.g. tended to rise with VP. Moreover, VP tended to approach Pms.g. on the average and slightly, when the cubital venous pressure rose above the upper limit of the "normal" range, and also when the cardiac index was low, independently of the venous pressure.

If the obviously over-simplifying assumptions which formed the basis of this approach are adequate, these facts suggest that the chronic congestive right heart failure, with elevated cubital venous pressure, is associated with elevated mean systemic pressure. Possible significance of the apposition between VP and Pms.g. observed for higher VP and lower CI (cardiac index) was discussed.

The function of the venous system has been a subject of intensive studies (Landis et al. 1946, 1950; Burch 1954; Burch et al. 1956; Alexander 1954; 1956, 1963; Haddy et al. 1954; Wood et al. 1956, 1958; Wood 1965; Gauer et al. 1962; Wallis et al. 1963; Braunwald et al. 1963; Folkow et al. 1964; Bevegard et al. 1965; Shepherd 1966; Ito 1966; Webb-Peploe et al. 1968; Coffman et al. 1969). Recently, a new method was introduced to measure the total systemic vascular compliance in animals (Shoukas et al. 1971). A series of papers (Kondo 1970; Imai 1971) supported and substantiated the observation by Hayase that the plasma osmolality of the systemic venous blood tends to become higher than that of the systemic arterial blood in the patients with right heart failure (Hayase et al. 1970).

Our quantitative understanding of the role of the capacitance vessels in circulatory control has been much advanced by studies on the mean vascular (filling) pressure (Gurvich 1955, 1959, 1963a, 1963b). The mean systemic pressure represents the extent of the filling of the systemic circulatory system with blood, and it represents the mean driving pressure returning blood to the

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right heart (Guyton 1955). In other words, the mean systemic pressure is the "Vis a tergo" for venous return to the right heart.

It will then serve a useful clinical purpose, if the mean systemic pressure can be estimated, even grossly, in human subjects. Mean systemic pressure was estimated to be about 7 mmHg in normal human subjects as a round-figure (Guyton 1959, 1963a, 1963b), but there have been few attempts to estimate this pressure in individual living humans. An interesting attempt, presented by Hori (1970a, 1970b) utilizes a small shift of blood volume caused by a bout (about 10 seconds) of artificial tachycardia, with resultant fall in arterial pressure and rise in right atrial pressure; mean systemic pressure was obtained graphically as the pressure in a point of the systemic circulation where the pressure is not altered by this procedure.

This paper represents an effort to estimate the mean systemic pressure in humans indirectly and roughly, on the basis of a set of obviously over-simplifying assumptions. Since the validity of these assumptions cannot not be demonstrated at this moment, the present approach is only a preliminary one. The basic assumptions and procedures to estimate the mean systemic pressure will be described, and, as a partial defense of this approach, the graphically estimated mean systemic pressure (Pms.g.) will be compared with the cubital venous pressure, and the result will be discussed in its possible relationship to a previous study in animals (Hirakawa 1969) concerning the numerical relationship between the mean circulatory pressure (MCP) and peripheral venous pressure during the acute systemic "venous congestion".

This is a systematic presentation of what has been described briefly and fragmentally (Hirakawa 1968a; Hirakawa et al. 1968b).

THEORETICALS

The systemic venous return curves which relate the venous return (ordinate) to the right atrial pressure (abscissa), in dogs, remain on a plateau at all right atrial pressures more negative than -2 mmHg to -4 mmHg, but they have downslopes, venous return decreasing almost, but not completely, linearly with rising right atrial pressure until the curves intersect with the abscissa (Guyton et al. 1957). The downslopes of venous return curves are thus approximated very well by straight lines. Such a straight-line approximation has been used in constructing a venous return curve from the measurements of cardiac output, right atrial pressure and mean circulatory pressure (Smith et al. 1967). One of the present authors also constructed venous return curves in dogs with the straightline approximation (Hirakawa 1969). The downslope of systemic venous return curves in humans was also visualized as nearly straight by Guyton (1963b).

Moreover, Guyton visualized the slope of the venous return curves in the patients with congestive heart failure as something not appreciably different from the slope of "normal" venous return curves (Guyton 1963b).

There is no experimental evidence supporting the constant-slope-assumption, an assumption that a constant slope applies to the straightly falling phase of every venous return curve throughout all human subjects, with or without heart diseases; to the contrary, arguments can be raised easily in opposition to such a sweeping assumption. But as one of the 2 obviously oversimplifying assumptions in this study, a constant and unique value was assumed to apply to the downslope of all venous return curves.

The two simplifying assumptions made in this study were as follows: (1) "normal" venous return curve for a mean systemic pressure (Pms) of 7 mmHg gives, by definition, a venous return of 3.8 l/min·1·1·M² at zero right atrial pressure, and (2) different human subjects have venous return curves with downslopes parallel to this "normal" venous return curve.

Fig. 1 illustrates the graphic method used to obtain the estimated value of Pms. The approxi-
mate value of Pms obtainable in this manner is termed Pms.g. (graphically estimated mean systemic pressure).

Parenthetically, Pms.g. can be also obtained algebraically as $P_{ms.g} = \left(3.8/7\right) C_{I} + RAP$ where CI means cardiac index, with the omission of the dimension of $lOU/min \cdot m^{-2}$, and RAP means right atrial pressure in mmHg. Derivation of this relationship is simple. Since the slope of the straightly falling phase of the venous return curve is, by definition, $-(3.8/7) lOU/min \cdot m^{-2}/mmHg$, any venous return curve can be described in the form $Y = -(3.8/7) X + b$.

For a given cardiac index $Y = CI$ and right atrial pressure $X = RAP$, the venous return to be expected for zero right atrial pressure can be written as $B$, thus $b = B$. Then $CI = -(3.8/7) RAP + B$. Since $B/Pms.g = 3.8/7$, by substitution we obtain $Pms.g = \left(3.8/7\right) CI + RAP$.

METHODS

Protocols of a total of 36 patients with or without heart diseases admitted to the 3rd Department of Medicine, Kyoto University Hospital were reviewed for the right heart catheterization data and for the cubital venous pressure (VP\textsubscript{40}) data. The right heart catheterization and cubital venous pressure measurements were not performed simultaneously but separately at near-by points of time. The term VP\textsubscript{40} will be used in this paper to denote the cubital venous pressure referred to a zero-pressure reference point which was set 40 mm behind a point on the anterior surface of the chest at the level of the 4th intercostal space along the parasternal line.

The 36 patients consisted roughly of the following 4 groups: (1) non-cardiac diseases 6 (No.1–6), (2) aortic valvular diseases 7 (No. 7–13), (3) mitral valvular diseases with or without aortic valvar involvement 20 (No. 16–35), and (4) other diseases, including hypertension 2 (No. 14, 15) and cor pulmonale 1 (No. 36).

Right heart catheterization

Cardiac catheterization was carried out in the usual manner (Luisada 1958) as described elsewhere by one of the authors (Sakai 1966). To recapitulate the main points, pressure recordings were made with a saline-filled cardiac catheter and a Statham P24D transducer and a cathod-ray photographic recording system. The mean pressures were obtained by electronic integration. Cardiac output was calculated by Fick's principle with oxygen contents of blood being determined by the method of Van Slyke and Neil; the expired air was collected in Douglas bags and analysed by the modified Haldane apparatus. In cardiac catheterization studies, the zero-pressure reference level was set at the mid-thoracic point.

Peripheral venous pressure measurements

The cubital venous pressure was measured with a saline manometer, i.e., from the height of a column of 3.8% citric acid which has descended and reached an equilibrium with the pressure inside the antecubital vein of a half-supinated and half-abductted upper arm, with the patient in supine position. Pressure readings were made after repeated determinations at regular intervals for several minutes assured the stability of the venous pressure. Venous pressures were measured at bed-side and the zero-pressure reference point was set 40 mm behind (towards the spine from) the anterior surface of the chest, hence the symbol VP\textsubscript{40}. Since the anteroposterior diameter of the chest was not found in the protocols of many cases, VP\textsubscript{40} was converted, in the first approximation, into VP, readings of the venous pressure as referred to the mid-thoracic point, in mm Hg, by adding 55 mm H\textsubscript{2}O and dividing the sum by 13.6. Thus $VP = (VP_{40} + 55)/13.6$. The correction factor, 55 mm, was a round-figure for the distance between a point 40 mm behind the anterior surface of the chest and the mid-thoracic point.

The use of a round-figure in obtaining the venous pressure readings as referred to the mid-thoracic point (VP) formed a source of error in this study. Validity of the round-figure, 55 mm, was confirmed by a later study (Hirakawa et al., manuscript in preparation) where the relationship between VP\textsubscript{40} and venous pressure referred to the mid-thoracic point was studied in 196 simultaneous measurements. The anteroposterior diameter of the chest was considerably different from person to person, but the average relation was that of $VP_{ml} = 0.94VP_{40} + 59$ where $VP_{ml}$ was the venous pressure referred to the mid-thoracic point, in mm H\textsubscript{2}O. The round-figure for the correction term will be, from this result, 53 mm for $VP_{40} = 100$ mm H\textsubscript{2}O and 56 mm for $VP_{40} = 50$ mm H\textsubscript{2}O.

Graphic estimation of the mean systemic pressure

Graphically estimated mean systemic pressure (Pms.g.) was obtained in a manner described in "Theoretical", i.e., as shown in Fig. 1.

RESULTS

Table 1 lists the pertinent data for individual
<table>
<thead>
<tr>
<th>No.</th>
<th>Name</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Physiologic data</th>
<th>Calculated data</th>
</tr>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Index</td>
<td>Pms.g. mmHg</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>l/min (\text{m}^2)</td>
<td>Pms.g.—VP mmHg</td>
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<td>AS</td>
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<td>m</td>
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<td>9.7</td>
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<td>8</td>
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<td>19</td>
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<td>MS, AI</td>
<td>2.6</td>
<td>13</td>
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<td>MS</td>
<td>2.7</td>
<td>12</td>
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<td>49</td>
<td>f</td>
<td>MS, AI</td>
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<td>13</td>
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<td>f</td>
<td>MS</td>
<td>2.1</td>
<td>7</td>
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<td>f</td>
<td>MS</td>
<td>2.9</td>
<td>9.4</td>
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<td>HK</td>
<td>19</td>
<td>f</td>
<td>MS</td>
<td>3.9</td>
<td>10.3</td>
</tr>
<tr>
<td>24</td>
<td>KT</td>
<td>33</td>
<td>f</td>
<td>MS</td>
<td>2.5</td>
<td>7.5</td>
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<tr>
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<td>23</td>
<td>f</td>
<td>MS</td>
<td>3.0</td>
<td>9.5</td>
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<tr>
<td>26</td>
<td>NS</td>
<td>46</td>
<td>m</td>
<td>MS</td>
<td>3.7</td>
<td>9.7</td>
</tr>
</tbody>
</table>

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| 27 | NT | 18 | m | MS | 2.5 | 10 | 88 | 10.5 | 14.7 | 4.2 |
| 28 | YM | 23 | f | MI | 2.0 | 11 | 130 | 13.5 | 14.8 | 1.3 |
| 29 | IF | 19 | f | MIS | 5.3 | 5 | 118 | 12.7 | 14.8 | 2.1 |
| 30 | NK | 25 | f | MSI | 2.7 | 4 | 67 | 8.9 | 9 | 0.1 |
| 31 | NK | 24 | f | MI | 2.2 | 13 | 137 | 14.1 | 16.8 | 2.7 |
| 32 | NK | 39 | f | MS, ASI | 2.8 | 3 | 80 | 9.9 | 8.1 | -1.8 |
| 33 | KE | 49 | f | MIS | 1.8 | 8 | 65 | 8.8 | 11.3 | 2.5 |
| 34 | YN | 24 | f | MI (S) | 1.7 | 11 | 136 | 14.0 | 14.1 | 0.1 |
| 35 | WY | 44 | m | MS, AI, TI | 2.2 | 11 | 126 | 13.2 | 15 | 1.8 |
| 36-1 | UR | 62 | m | Cor pulmonale | 2.8 | 5 | 59 | 8.4 | 10.2 | 1.8 |
| 36-2 | UR | Same as above | 2.9 | 9 | 108 | 11.9 | 14.3 | 2.4 |
| 36-3 | UR | Same as above | 2.9 | 4 | 50 | 7.7 | 9.2 | 1.5 |

RAP: Right atrial pressure, Pms.g.: Mean systemic pressure estimated from cardiac index and right atrial pressure graphically. VP40: Cubital venous pressure, in mmHg, with zero-pressure reference point 40 mm behind the anterior chest wall at the level of 4th intercostal space. VP: Cubital venous pressure, in mmHg, with zero-pressure reference point at the mid-thoracic level. NCA: Neurocirculatory asthenia. AS: Aortic stenosis. AI: Aortic insufficiency with stenosis. MS: Mitral stenosis. PH: Pulmonary hypertension. MIS: Mitral insufficiency with stenosis. TI: Tricuspid insufficiency. Group B) a result such as shown in Table II was obtained. Thus the pressure-difference, Pms.g. - VP, did not differ significantly according as the venous pressure was normal (Group A) or elevated (Group B); cardiac index was quite comparable between the 2 groups.

In Fig. 3, the abscissa gives cardiac index in lit. min⁻¹ M⁻² and ordinate gives the pressure-difference, Pms.g. - VP in mm Hg. Although there is a considerable scatter of data, the average relation could be fitted by a regression line of Y = -0.8 + 0.72 X. This may be taken to mean that the cubital venous pressure (VP) is closer, on the average, to Pms.g. when the cardiac index is low than when it is normal.

When the cardiac index data were extracted from Table I and divided into two groups, i.e., normal (CI ≤ 2.5 lit. min⁻¹ M⁻², Group A') and low (CI < 2.5 lit. min⁻¹ M⁻², Group B'), it was found that the pressure-difference, Pms.g. - VP, again did not differ significantly according as the cardiac index was normal (Group A') or low (Group B'); cubital venous pressure was of comparable magnitude (Table III).

Putting these results (Fig. 2, 3 and Table II,
TABLE II  PRESSURE-DIFFERENCE, Pms.g. – VP, IN THE PRESENCE OF NORMAL VENOUS PRESSURE (VP < 11.4 mmHg, GROUP A) AND ELEVATED VENOUS PRESSURE (VP ≥ 11.4 mmHg, GROUP B)

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of measurements</th>
<th>Cardiac Index L/min/m²</th>
<th>VP mmHg</th>
<th>Pms.g. – VP mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>28</td>
<td>3.0 ± 0.8</td>
<td>8.2 ± 2.0</td>
<td>1.6 ± 2.4**</td>
</tr>
<tr>
<td>B</td>
<td>10</td>
<td>3.1 ± 1.3</td>
<td>13.3 ± 1.0</td>
<td>0.7 ± 1.6**</td>
</tr>
</tbody>
</table>

** 0.3 > p > 0.2 (mean ± SD)

Fig. 2. Graphically estimated mean systemic pressure (Pms.g.) plotted against the measured values of cubital venous pressure (VP). The data can be fitted by a regression line of Pms.g. = 4.7 + 0.65 VP.
- ● Non-cardiac diseases
  - □ Aortic valvular diseases
  - ○ Mitral valvular diseases, with or without aortic valve involvement
  - △ Other diseases

III) together, it can be stated that (1) VP rises with Pms.g. and that (2) although there is an average tendency for VP to approach Pms.g. when VP is above the upper limit of the "normal" and also when cardiac index is low, apart from the venous pressure, this trend of mutual opposition is not of such a distinct quality as making the pressure difference, Pms.g. – VP, significantly

smaller for higher VP, or for lower CI, than when these parameters were normal.

DISCUSSION

The result of theoretic studies is assumption-limited, and that of experimental studies is method-limited. Besides the two basic assumptions which formed the basis of this approach and which were described in "Theoretical", the present authors also assumed (1) validity of converting VP₄₀ to VP (venous pressure readings as referred to the mid-thoracic point), using a roud-

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figure as the correction term, and (2) validity of combining hemodynamic data and venous pressure readings obtained not simultaneously. These form the potential sources of errors.

Fig. 4 gives the pressure-difference, \( P_{\text{ms.g}} - VP \), as a function of \( VP \). The regression line, \( P_{\text{ms.g}} = 4.7 - 0.35VP \) is just what is expected from the earlier regression line, \( P_{\text{ms.g}} = 4.7 + 0.65 \text{ VP} \), of Fig. 2.

One of the consequences of this study was the fact that \( P_{\text{ms.g}} \) rose with \( VP \), as shown in Fig. 2. While this fact suggests an important conclusion that the chronic congestive right heart failure, with elevated peripheral venous pressure, is associated with elevated mean systemic pressure, it must be appreciated that the result could not be otherwise, so long as the two basic assumptions were used to estimate the mean systemic pressure.

Another consequence of the present approach was the suggestion that possibly cubital venous pressure becomes numerically close to the mean systemic pressure, when the cubital venous pressure rises above the upper limit of the "normal" range and, apart from this, when cardiac index is low. In view of the above-mentioned sources of errors in graphically estimating mean systemic pressure, the quantitative significance of regression lines, of Fig. 2 to 4, may be questionable.

A "normal" cardiac index of 3.8 l/min/m² was selected in this study because this was the mean value of cardiac indices of normal subjects determined in this laboratory (See Inagaki 1966). The assumption of zero right atrial pressure as the standard state may be considered unacceptable. Mean value of the cycle-averaged-mean right atrial pressure was reported to be normally 3.8 mm Hg (Luisada and Liu 1958) or 3.4 mm Hg (Hayase et al. 1969), to cite a few references. If the right atrial pressure of 3mmHg, not zero mm Hg, is adopted as the "normal" value, and combined with cardiac index of 3.8 l/min/m² and mean systemic pressure of 7mmHg, the graphically estimated mean systemic pressure would be lower than above-calculated by 2 to 3 mm Hg, depending on cardiac index. A simple calculation shows that the difference should be 0.77 x CI. mmHg. lit⁻¹ min. M².

A family of venous return curves appear to have almost parallel downslopes for mean systemic pressures above 7 mm Hg (Guyton 1963b, Fig. 90, 93, 96). Moreover, parallel venous return curves were assumed to exist in chronic congestive right heart failure for mean systemic pressures as high as 10 to 20 mm Hg (Guyton 1963, Fig. 188, 189). However, if one should assume, instead of a family of parallel venous return curves, a family of venous return curves in which downslopes become less and less steep with rising mean systemic pressure, this would produce an effect of rotating the regression line of Fig. 2 upwards to the left; then \( P_{\text{ms.g}} \) will rise VP with an angle of more nearly 45 degrees.

### TABLE III

**PRESSURE-DIFFERENCE, \( P_{\text{ms.g}} \) - \( VP \), IN THE PRESENCE OF NORMAL CARDIAC INDEX (CI ≥ 2.5 l/min/m², GROUP A) AND LOW CARDIAC INDEX (CI < 2.5 l/min/m², GROUP B)

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of measurements</th>
<th>Cardiac Index ( l/min/m² )</th>
<th>( VP ) mmHg</th>
<th>( P_{\text{ms.g}} - VP ) mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>( A' )</td>
<td>28</td>
<td>3.4 ± 0.8</td>
<td>9.1 ± 2.6</td>
<td>1.8 ± 2.2*</td>
</tr>
<tr>
<td>( B' )</td>
<td>10</td>
<td>2.0 ± 0.3</td>
<td>10.9 ± 3.4</td>
<td>0.4 ± 2.0*</td>
</tr>
</tbody>
</table>

* 0.1 > \( p \) > 0.05 (mean ± SD)

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Although the regression lines such as shown in Fig. 2 to 4 thus have dubious quantitative significance, it is true, as a matter of fact, that the numerical relationship between the graphically estimated mean systemic pressure (Pms.g.) and cubital venous pressure (VP), like Fig. 2, bears an at least superficial resemblance to the experimentally verified numerical relationship between the mean circulatory pressure (MCP) and forgeleg venous pressure (VPf) in anesthetized dogs in which systemic venous pressure was elevated to various extents by inflating a balloon in the right atrium (Hirakawa 1969).

In the animal experiment, interesting enough, the tendency for MCP and VPf to converge at high VPf was not a striking one just as it was the case in this study (Table II); thus the pressure gradient, mean circulatory pressure (MCP) minus central venous pressure (CVP), termed the “gradient for venous return (GVR)” according to Guyton (1955), did not diminish significantly even when the “venous congestion” was marked enough to cause the foreleg venous pressure to rise, from control values of 1.4±0.4 mm Hg (mean±S.E.), to 4.4±0.4 mmHg or even to 10.1±1.0 mmHg. Thus “there was roughly parallel increase in MCP and peripheral venous pressure during the “venous congestion” of mild degree, but the MCP and peripheral venous pressure tended to more or less converge during severe “venous congestion”. The over-all relation is expressed by a regression line, MCP = 5 + 0.8 VP.” (Hirakawa 1969).

Since the gradient for venous return (GVR) is a product of the venous return and resistance to venous return (RVR), GVR = VR x RVR (Guyton 1955), and since the venous return is equal to cardiac output in steady-state conditions, numerically GVR = CO x RVR. When one measures the venous pressure, (VP) in a vein located just distal to the right atrium, the pressure difference, Pms – VP, must be close to GVR, thus Pms – VP = CO x RVR. Therefore there are two factors capable of making VP numerically close to Pms; these are a decreased cardiac output and a low RVR. Results of an animal experiment supported this conclusion (Hirakawa 1969, Table III).

If the resistance to venous return (RVR) is held constant in practice or in theory, a low cardiac output should be associated with diminished pressure difference, Pms – VP, and this might be the explanation to be given to the regression line of $Y = -0.8 + 0.72X$, of Fig. 3, relating Pms.g. – VP (Y) to cardiac index (X).

Granted that the regression line of Fig. 2, Pms.g. – VP = 4.7 + 0.65 VP has a quantitative meaning, why, then, should VP become numerically close to Pms.g. at high VP’s in spite of the fact that RVR has been held constant in theory (see “Theoreticals”) and that cardiac index did not diminish with increasing VP, a fact presented in Table II?

To answer this question, it is no longer appropriate to regard the cubital venous pressure as something a little higher than the right atrial pressure. One must examine the nature of the circulatory state in which the blood pressure existing in the cubital vein becomes numerically close to the mean systemic pressure without decreased cardiac output or decreased resistance to venous return (RVR).

Since the mean systemic pressure is, according to Guyton (1963b, p. 261), equal to the total extra volume (EVs) of blood in the systemic circulation above that required to fill the system with no pressure divided by the total capacitance of the systemic circulation (Cs)

$$Pms = EVs/Cs = \sum EVi/\sum Ci = \sum CiP\i/\sum Ci$$

where EVi, Ci and Pi is the extra volume, capacitance ($\Delta V/\Delta P$) and blood pressure (P), of the i-th segment of the systemic vascular trees, the blood pressure existing in a segment is represented to the mean systemic pressure with the greatest weight when that segment has the largest capacitance of all the segments. Therefore, if the venous segment represented by the cubital vein has a disproportionately large capacitance ($\Delta V/\Delta P$) over capacitances of other segments of the systemic vascular trees, mean systemic pressure becomes numerically close to the cubital venous pressure.

However, the relative increase of the capacitance of the venous circuit was shown to diminish the resistance to venous return (Guyton 1963b, Fig. 104). Therefore, one must make another assumption that the arterial or venous resistance to blood flow has been increased to an extent just enough to restore the resistance to venous return back to “normal”.

In short, then, one can explain why the cubital venous pressure becomes numerically close to the mean systemic pressure in the absence of decreased cardiac output or decreased resistance to venous return, if one assumes (1) a re-distribution of relative capacitances within the systemic vascular trees in such a manner that the

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capacitance is relatively increased in the venous segment represented by the cubital vein, while it is decreased relatively in smaller veins distant from the right atrium, simultaneously with (2) an increase in the arterial or venous resistances to blood flow to an extent just enough to restore the resistance to venous return back to "normal".

These assumptions are not entirely unlikely to be materialized in the patients with chronic congestive right heart failure, because, for one thing, the capacitance (ΔV/ΔP) of the venous beds distal to (smaller than) the cubital vein was shown to be decreased in the patients with congestive heart failure (Wood 1965); fitting a tangent to his pressure-volume curves for forearm veins, it can be calculated that the capacitance (ΔV/ΔP) was about 0.16 ml/100 ml tissue/mmHg in normal subjects but it was as low as 0.10 ml/100 ml tissue/mmHg in the patients with congestive heart failure, for effective venous pressure of 7 mmHg.

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