Continuous Recording of Direct Arterial Pressure in Unrestricted Patients
Its Role in the Diagnosis and Management of High Blood Pressure


SUMMARY
We have compared casual indirect measurements of arterial pressure obtained 1) by the general practitioner (GP) and 2) in the outpatient clinic (OPC) with 24 hour continuous recordings of direct arterial pressure in two selected groups of unrestricted patients.

1) Eight asymptomatic, untreated patients with suspected hypertension.
2) Eight asymptomatic, treated patients whose indirect pressure readings seemed inappropriately high when considered against a general absence of target organ damage.

Both groups showed that usually there was good agreement between arterial pressure recorded indirectly by GP and OPC while continuous recordings showed wide variability of systolic and diastolic pressures over 24 hours and a significant fall during sleep.

The first group with suspected hypertension showed that the indirect measurements were not significantly different from the 24 hour direct recording. The second group of patients on treatment for hypertension showed a discrepancy, the direct readings being significantly lower than the indirect. This difference (approximately 30 mm Hg mean arterial pressure) would explain the lack of target organ damage and may have been due to the effect of exercise augmenting the hypotensive action of drugs or due to a well developed defense reflex which biased the indirect readings.

Additional Indexing Words:
Defense reflex    Sleep    Hypotensive drugs

THERE IS GOOD STATISTICAL correlation between casual blood pressure and life expectancy. However, the difficulty in obtaining a true reading of casual blood pressure is evident from the variety of methods devised. Armitage and Rose (1966) and Armitage et al. (1966) stressed that even under the most stringent conditions, the errors of measurement are great, and if a single reading is taken, about one third of subjects designated as having raised blood pressure (arbitrarily designated as over 160/90 in their study) proved to be normal on repeated measurement; the diastolic pressure fell by a mean of 12 mm Hg.

It is possible that an even better prognostic estimate could be obtained from the more accurate and more prolonged measurements now possible using portable

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nitrogen) of severe hypertension in spite of their abnormal blood pressure readings. They continued to follow their drug therapy (table 2).

All 16 patients freely consented to this study after it had been explained to them. The methods used for measuring direct arterial pressure and the electrocardiogram in unrestricted patients have been fully described elsewhere.\(^6\)\(^7\) The patients were studied over a 24 hour period from 9 a.m. to 9 a.m. During this time they attended the laboratory only once for 15 minutes after a 12 hour period in order to apply calibration to the tape and to service the perfusion chamber. All went about their daily routine at both work and home, and all patients slept at home.

The 24 hour recordings were replayed at 25 times the recording speed (less than 60 min). Automatic analysis of the tape recording derives systolic, diastolic, pulse, and mean pressures and pulse interval either individually or averaged over 10, 100, or 1000 beats.\(^8\)

A digital readout from this analyzer is, as yet, unavailable but a visual display aids analysis of the 24 hour records and presents the whole record in only two feet of paper (fig. 3). In this study arterial pressure was averaged over each 100 beats. Each record was divided into 12 equal parts and the average values in each part were determined visually. They are shown, together with the range, standard deviation, and standard error, in table 3.

The general practitioner and outpatient clinic physician used a mercury sphygmomanometer and stethoscope to determine indirect arterial pressure. Diastolic pressure was taken as the point of cuff muffle (fourth Korotkoff sound). The results listed in tables 1 and 2 represent the average of at least three readings of blood pressure taken on consecutive visits. For analysis of the results the mean blood pressure was calculated from these readings as the diastolic pressure plus one-third of the pulse amplitude.

**Statistical Analysis**

The average systolic, diastolic, and mean arterial pressure determined by the indirect and direct methods in each patient was obtained and the data tested by an analysis of variance. The error mean square was calculated from the total sums of squares after subtracting “between methods” sums of squares (differences between the indirect and direct values), “between patients” sums of squares (differences in the values between the patients). The significance of the differences in the mean values between the various methods of measuring pressure was tested by the variance ratio for the data as a whole and, using the Student’s t-test, the significance of differences between the individual values derived by each method was determined.

The standard error of the mean value of pressure measured by a single method was calculated as

\[
\text{error mean square} = \frac{\text{error mean square}}{N}
\]

where N is the number of patients.

**Results**

The quantitative analysis for the whole 24 hours is summarized in table 3. Only one patient (case 7) did not show a decrease in systolic and diastolic pressure during sleep although her mean pressure fell slightly (6 mm Hg). Comments in her diary indicated that her
Table 2

Clinical Characteristics and Blood Pressure Readings of Asymptomatic Treated Subjects (Group 2)

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>Duration hypertension (mo)</th>
<th>Affect</th>
<th>Fundi</th>
<th>ECG S1R5* (mm)</th>
<th>Chest Xray CT ratio</th>
<th>BUN (mg %)</th>
<th>Treatment</th>
<th>BP-GP</th>
<th>BP-OPC</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>53</td>
<td>F</td>
<td>72</td>
<td>Placid</td>
<td>AV Nip†</td>
<td>37</td>
<td>60</td>
<td>36</td>
<td>Bethanidine 100 mg, Methyldopa 750 mg, Aldactide</td>
<td>228/117</td>
<td>228/118</td>
</tr>
<tr>
<td>10</td>
<td>59</td>
<td>M</td>
<td>24</td>
<td>Nervous</td>
<td>Arterial narrowing</td>
<td>38</td>
<td>Normal</td>
<td>43</td>
<td>Aldactide 80 mg, Propranolol 50 mg</td>
<td>152/110</td>
<td>207/127</td>
</tr>
<tr>
<td>11</td>
<td>64</td>
<td>F</td>
<td>72</td>
<td>Placid</td>
<td>AV Nip</td>
<td>28</td>
<td>Normal</td>
<td>38</td>
<td>Propranolol 240 mg, Hydralazine 100 mg, Bethanidine 150 mg</td>
<td>178/110</td>
<td>178/110</td>
</tr>
<tr>
<td>12</td>
<td>49</td>
<td>F</td>
<td>156</td>
<td>Cheerful</td>
<td>AV Nip</td>
<td>28</td>
<td>55</td>
<td>33</td>
<td>Propranolol 240 mg, Propranolol 240 mg, Aldactide</td>
<td>188/130</td>
<td>218/130</td>
</tr>
<tr>
<td>13</td>
<td>21</td>
<td>F</td>
<td>6</td>
<td>Nervous</td>
<td>Normal</td>
<td>20</td>
<td>Normal</td>
<td>24</td>
<td>Propranolol 240 mg, Aldactide</td>
<td>164/117</td>
<td>160/118</td>
</tr>
<tr>
<td>14</td>
<td>64</td>
<td>F</td>
<td>48</td>
<td>Nervous</td>
<td>Normal</td>
<td>24</td>
<td>Normal</td>
<td>61</td>
<td>Propranolol 240 mg, Aldactide</td>
<td>203/102</td>
<td>208/103</td>
</tr>
<tr>
<td>15</td>
<td>47</td>
<td>F</td>
<td>180</td>
<td>Placid</td>
<td>AV Nip</td>
<td>27</td>
<td>Normal</td>
<td>18</td>
<td>Propranolol 100 mg, Methyldopa 750 mg, Aldactide</td>
<td>185/115</td>
<td>168/112</td>
</tr>
<tr>
<td>16</td>
<td>28</td>
<td>M</td>
<td>24</td>
<td>Cheerful</td>
<td>Normal</td>
<td>40</td>
<td>55</td>
<td>32</td>
<td>Propranolol 160 mg, Navidex K.</td>
<td>168/83</td>
<td>158/96</td>
</tr>
</tbody>
</table>

*S1R5 represents the sum of the amplitude of the S wave in lead V1 and the R wave in lead V6 of the 12-lead ECG.
†Arteriovenous nipping.

Abbreviations: BP = blood pressure; BUN = blood urea nitrogen; GP = general practitioner; OPC = outpatient clinic.
sleep was intermittent during the night of her study.

For the group as a whole, the arterial pressure fell by an average of 27/19 mm Hg during sleep. This was a significant fall when compared with waking hours ($P < 0.01$, systolic; $P < 0.05$, diastolic).

Over the entire 24 hours six of the eight patients had a mean pressure in excess of 110 mm Hg, and pressures of all eight occupied a wide range of values.

Figure 1 compares the average of all the general practitioners' readings of arterial pressure and the average of the outpatient clinic readings with the average over the whole 24 hours. The results from all eight patients have been grouped together. The 24 hour levels are lower than either of the indirect readings since they include the lower nocturnal readings. Despite this fact there was no statistically significant difference among the three sets of readings.

Figure 2 is a similar plot to figure 1, for mean arterial pressure but also separates out the direct mean pressure during waking hours. There was no significant difference among the four groups of readings.

Figure 3 is the complete record from case 3, a man whose readings over 24 hours were lower than either of his indirect readings. The interesting feature is the considerable rise in arterial pressure which occurs before he attends the laboratory in the evening (arrow). This level then slowly falls again.

**Group 2**

The quantitative analysis is summarized in table 3. All eight patients showed a fall in arterial pressure during sleep, the average fall being 40/16 mm Hg ($P < 0.001$, systolic; $P < 0.05$, diastolic).

Over the whole 24 hours five patients had a mean arterial pressure greater than 110 mm Hg, but only

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**Table 3**

**Summary of Data Derived from Continuous Arterial Pressure Recordings**

<table>
<thead>
<tr>
<th></th>
<th>Systolic</th>
<th></th>
<th>Diastolic</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>sd</td>
<td>SEM</td>
<td>Range</td>
</tr>
<tr>
<td><strong>Group 1</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Awake</td>
<td>174.5</td>
<td>20.8</td>
<td>7.6</td>
<td>100-231</td>
</tr>
<tr>
<td>Asleep</td>
<td>148.6</td>
<td>14.9</td>
<td>6.2</td>
<td>90-230</td>
</tr>
<tr>
<td>24 hrs</td>
<td>164.7</td>
<td>29.1</td>
<td>8.0</td>
<td>90-231</td>
</tr>
</tbody>
</table>

**Group 2**

<table>
<thead>
<tr>
<th></th>
<th>Systolic</th>
<th></th>
<th>Diastolic</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>sd</td>
<td>SEM</td>
<td>Range</td>
</tr>
<tr>
<td>Awake</td>
<td>176.1</td>
<td>34.9</td>
<td>11.2</td>
<td>100-340</td>
</tr>
<tr>
<td>Asleep</td>
<td>135.7</td>
<td>10.3</td>
<td>4.9</td>
<td>88-226</td>
</tr>
<tr>
<td>24 hrs</td>
<td>171.9</td>
<td>39.1</td>
<td>16.5</td>
<td>88-226</td>
</tr>
</tbody>
</table>

---

**Figure 1**

Summary of the grouped data from both treated and untreated patients giving the average systolic and diastolic pressures measured by the general practitioner (GP), outpatient clinic physician (OPC) and the continuous recording (24 hr). The symbols within the bars represent ± 1 se of mean for the reading measured by any method. Black dot indicates statistically significant at probability level of 5% or less.

**Figure 2**

Summary of the data for mean arterial pressure (MAP) in both groups of patients. The "awake" period during the 24 hours is also plotted. Symbols ± 1 se refer to the mean reading measured by each method; black dot indicates statistically significant difference at probability level of 1% or less.
one had a mean pressure greater than 120 mm Hg. There was wide variation in the group especially in the levels of systolic pressure (fig. 4). Figures 1 and 2 compare the indirect and direct readings for the group as a whole in the same manner as described for group 1. The direct readings were significantly lower than either of indirect measurements \( P < 0.01 \), systolic; \( P < 0.001 \), diastolic; \( P < 0.001 \), mean. Over the entire 24 hours the average mean arterial pressure for grouped data was some 25–30 mm Hg lower than indicated by the indirect method. There was no obvious relationship between the ingestion of antihypertensive drugs and changes in blood pressure.

**Discussion**

In two selected groups of subjects, this study has shown that there is usually good agreement between the arterial pressure recorded indirectly by the general practitioner, and that found by the outpatient clinic physician, contradicting the assumption that a hospital environment is more likely to arouse an "alerting" or "defense" response and lead to higher pressures than those in the doctor’s surgery.

The first group of subjects (1–8) with suspected hypertension showed that these indirect measurements correlated well with the 24 hour direct recording, although there was some intrasubject variation. 2/8 showing lower readings by the 24-hour method (cases 2, 3). However, both these men showed elevations of pressure that were transient and apparently environmentally determined, particularly by the prospect of attending the hospital within a few hours. Figure 3 shows quite clearly how the arterial pressure in case 3 rises in anticipation of attending the laboratory in the evening and falls gradually afterward. These patients may be close to borderline hypertension, but they may, on the other hand, be revealing what several authors have described as "an undue defence reflex" (for review, see Pickering²). In this context, Stewart⁴ has reported his findings in a group of 12 young men whose initially high blood pressure fell slowly over a period of five to eight years without treatment. There is a clear indication that patients such as these merit repeated observation, and that a single casual reading, or 24-hour recording, provides insufficient criteria for diagnosis and treatment.

It is perhaps surprising that in group 1 there was such good agreement of casual with 24-hour blood pressures since the more prolonged recordings would include exercise which would be expected to raise arterial pressure, particularly if it included so called "static" exercise such as carrying.¹⁰ Indirect blood pressure measurement by sphygmomanometry is fraught with potential error because of faulty instrumentations, clumsy technique, and the problems of cuff and sound.¹¹ Systolic pressures may be underestimated and diastolic pressures overestimated by an average of 10 mm Hg². This fact may account for the lower diastolic pressure obtained by the direct method in the figure. Previous studies of the physician’s influence on blood pressure have suggested that both his presence or a hospital setting increase blood pressure and that frequent,
semiautomatic blood pressure recordings taken by the ambulatory patient away from the medical environment usually are considerably lower than the office casual blood pressure. It could be argued that our patients in group 1 have a large defense reflex and react just as adversely to portable machinery as they do to the physical presence of doctors, though on balance we think this unlikely.

The second group of patients on treatment for hypertension were selected because their outpatient readings seemed inappropriately high when considered against the lack of evidence of circulatory damage. In this group all eight patients had substantially lower pressures measured over 24 hours. This discrepancy was most marked with diastolic pressure, which was within the acceptable limits for patients under treatment. Errors inherent in the indirect technique for measuring arterial pressure have already been discussed and may have played some part in this discrepancy, but it is very unlikely to be the whole reason. The difference may have been due to the well-known effect of exercise in augmenting the hypotensive action of drugs, although the intraarterial pressure was often low even when the subject was sitting quietly. We did not find any obvious time relationship between the ingestion of drugs and the level of arterial pressure. Another possibility, particularly in view of their benign course, is that their clinic readings were biased by an unusually sensitive defense reflex.

The recordings in both groups confirmed the wide variability of arterial pressure, particularly in relation to environmental events, demonstrated earlier in our laboratory. They have reaffirmed the significant fall in arterial pressure which occurs during sleep irrespective of treatment. The falls in pressure in patients with high arterial pressures were greater than those recorded by Bevan et al. (9/2 mm Hg benign phase, 20/9 mm Hg malignant phase), but one must remember that these patients slept in the hospital whereas the present group all slept at home, and therefore, more likely reflect the true fall.

Our results show how easy it could be to overtreat certain patients on the basis of casual readings but nevertheless such readings remain at present the mainstay of clinical assessment.

Acknowledgment
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