Dynamic Reactions Produced by Deflating a Blood Pressure Cuff

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SUMMARY

Intra-arterial pressure was recorded at various points in the compressed segment of brachial artery during deflation of a standard blood pressure cuff in human subjects with arms of normal girth. Cuff pressure and the Korotkoff sounds also were recorded simultaneously. The data were analyzed in terms of the various dynamic reactions produced by cuff deflation, which may influence the auscultatory indications of systolic and diastolic blood pressure. Cuff pressure was incompletely transmitted to the compressed arterial segment. As a result, muffling, the auscultatory indication of diastolic pressure, occurred at a cuff pressure higher than the directly recorded intra-arterial diastolic blood pressure. The first Korotkoff sound, on the other hand, provided a close approximation of intra-arterial systolic pressure. This may be due to a delay in penetration of the diminutive pulse waves into the distal part of the compressed arterial segment at systolic levels of cuff pressure. This effect appears to compensate for other influences tending to raise the indirect reading of systolic blood pressure. The extent of the delay in penetration should be dependent on the length of the collapsed segment, which in turn is a function of cuff width.

Additional Indexing Words:
Intra-arterial pressure
Muffling
Korotkoff sounds
Blood pressure measurement

The auscultatory technique for estimating blood pressure is one of the most important and frequently used clinical measurements. Yet, the physical basis of the method is poorly understood. The origin of the Korotkoff sounds still remains an unsettled question. In addition, there are several other aspects of the technique that have not been clarified. For example, it is not known whether the intra-arterial pressure in the occluded segment is at the same or a different level than the cuff pressure. Neither has it been determined in man whether significant augmentation of systolic pressure caused by summation of reflected waves occurs proximal to the occluded segment. Although it has been postulated that the compression is greater in the center than at the ends the length of the occluded segment remains unknown. Finally, the pressure gradient through the compressed segment during the various stages of cuff deflation has not been determined in man.

The arterial pressure probably cannot be measured with great precision by the indirect method. Nevertheless, clarification of the hemodynamic changes induced by the compressing cuff would provide a sound physical basis for establishing the criteria to be used in estimating systolic and diastolic blood pressure by the auscultatory method. In the present report some of the dynamic reactions produced by the compressing cuff are elucidated and their implications with regard to the auscultatory criteria for estimating blood pressure are discussed.

Methods

Complete and technically satisfactory experiments were obtained in six male patients between the ages of 26 and 38 years. Patients
with obesity or cardiovascular abnormalities were excluded. The purpose and nature of the procedure were explained to each patient prior to obtaining his consent.

After anesthetizing the skin and subcutaneous tissue with lidocaine a 16-gauge thin-walled hypodermic needle was inserted into the brachial artery in the region of the antecubital fossa. A 30-cm length of vinyl tubing (0.028 inch ID) was inserted through the needle and threaded up the brachial artery approximately 20 cm. The needle was then removed and compression applied immediately over the puncture site.

The blood pressure cuff was the standard wrap-around type held in place by a "Velcro" backing. The inflatable bladder measured 12.5 by 23 cm and the nylon cloth covering, 14 by 58 cm. It was applied to the upper arm in the usual manner. To obtain fluoroscopic visualization of the location of the catheter tip thin lead strips were applied along the width of the cuff with adhesive tape spaced at 2-cm intervals from the proximal to the distal edge. The catheter was filled with 50% diatrizoate solution (Hypaque), and under direct observation, using an image intensifying fluoroscope, the tip of the catheter was positioned with reference to the lead strips. The diatrizoate solution was then withdrawn and the catheter filled with heparin-saline.

The proximal end of the catheter was connected to a P23db Statham strain-gauge pressure transducer fixed at heart level. The catheter-transducer system was thoroughly and repeatedly flushed with heparin-saline solution to exclude air bubbles and prevent clotting. Intra-arterial pressure was recorded continuously before and during inflation and deflation with the catheter tip placed just above the proximal edge of the cuff. The catheter tip was then withdrawn to the lead marker 2 cm distal to the proximal edge and the pressure recordings were repeated during cuff inflation and deflation. In this way a sequence of eight recordings of intra-arterial pressure were taken during cuff inflation and deflation spaced at intervals of 2 cm from just above the proximal edge to just below the distal edge of the blood pressure cuff.

The Korotkoff sounds were recorded simultaneously with the pressure recordings by means of a Sanborn dynamic microphone applied lightly to the arm with a rubber strap. The microphone was positioned over the brachial artery in the antecubital fossa. It was connected to a Sanborn amplifier with a high-pass filter of 12 db per octave and a nominal frequency cutoff of 100 cycles/second. The cuff was inflated rapidly by a hand bulb. An adjustable bleeder valve was used to control deflation, which occurred at a rate of approximately 3 mm Hg per second.

The cuff was connected via a T tube to both a mercury manometer and a P23db strain-gauge pressure transducer.

Simultaneous tracings of intra-arterial pressure, cuff pressure, and Korotkoff sounds were recorded on magnetic tape at a speed of 7½ inches/second.* This permitted playback on a Sanborn multichannel recorder at various paper speeds between 5 and 50 mm/second as desired. In one experiment the intra-arterial pressure in the opposite arm was recorded simultaneously through a 20-gauge hypodermic needle connected directly to a P23db Statham transducer.

Calibration of pressure transducer was performed at the end of each experiment by applying static pressures from 0 to 200 mm Hg. No evidence of linearity or hysteresis was found. The frequency response of the catheter-transducer system was tested for us by Dr. Peter Luchsinger, using a mechanical pulse-wave generator. When carefully filled to exclude air bubbles the pressure recording system was flat and free from phase shift to 30 cps.

The blood pressure gradient through the compressed segment was reconstructed from the sequence of recordings as follows: first, the series of intra-arterial pressure recordings taken at 2-cm intervals from just above to just below the cuff was inspected to determine whether the blood pressure remained stable during the sequence. This was done by measuring the average intra-arterial systolic and diastolic blood pressure, which was recorded during the 1-minute interval preceding cuff inflation at each of the eight catheter positions in the sequence of recordings. If the average systolic or diastolic blood pressure during these pre-inflation control periods showed deviations in any of the eight recordings greater than 5 mm Hg as compared to the other control readings the results for that sequence were discarded. In six of the subjects tested the sequence of control pre-inflation blood pressures showed deviations within the acceptable range, and these were used in plotting the blood pressure gradients.

Each recording contained three channels of information, the Korotkoff sounds, the intra-arterial blood pressure taken at a given catheter-tip position, and the cuff pressure. The location of the first Korotkoff sound (K1) was noted on the record, and the cuff pressure was measured at this point. Intra-arterial systolic and diastolic blood pressure also was measured. Next, intra-arterial blood pressure was measured at the point at which cuff pressure was 50 mm Hg above the

*Model PR-3300, Consolidated Electrodynamics Corp. Pasadena, California.
level of the cuff pressure present at the time of K1 as well as at various cuff pressures below this level. Then muffling was identified on the record as the point at which the initial high-frequency component of the Korotkoff sounds suddenly diminished in amplitude. Cuff pressure as well as systolic and diastolic intra-arterial blood pressure was measured at this point. Intra-arterial blood pressure also was measured at cuff pressures 30, 20, and 10 mm Hg above muffling.

The above-described measurements were made on each of the recordings representing the eight catheter-tip positions spaced 2 cm apart in the sequence of recordings taken through the compressed arterial segment. These measurements were then used to plot the spatial intra-arterial pressure gradients shown in figures 3 and 4.

Results

Penetration of the Pressure Pulse into the Compressed Arterial Segment

When the cuff pressure was 50 mm Hg above systolic blood pressure, pulsatile pressure fluctuations were not present in the compressed arterial segment. As cuff pressure was lowered, pulsatile pressures penetrated distally under the cuff. The initial pulsations were very small but grew progressively in amplitude as cuff pressure was lowered toward the systolic level.

Figure 1 shows Korotkoff sounds and intra-arterial blood pressure recordings from two of the eight catheter positions as recorded in one of the subjects. The simultaneously recorded cuff pressures have been omitted. The upper record was taken 8 cm distal to the proximal edge of the 14-cm wide cuff (1 cm distal to the midpoint of the compressed segment), whereas the lower was taken 2 cm further distally at a point 10 cm from the proximal edge of the cuff.
In each record the Korotkoff sounds are shown above the intra-arterial pressure recordings. The two tracings represent the area of transition in the pressure recordings between the proximal and distal regions of the compressed segment.

In the upper recording taken at the 8-cm position small pressure fluctuations penetrated through to this level prior to the onset of the first Korotkoff sound. In more proximal locations of the catheter these pressure fluctuations appeared progressively earlier in the deflation cycle. Note that the pulse pressure increased gradually to the point of muffling (indicated by M in the sound tracing). The increase of pulse pressure was due to a fall in the diastolic level as the cuff pressure was gradually reduced, thus permitting a greater pulse amplitude to penetrate into the compressed segment.

The recording taken 2 cm distal to this point (lower half of figure 1) shows marked changes in the intra-arterial pressure tracing. No pressure fluctuations occurred until the onset of the first Korotkoff sound, indicating complete arterial occlusion at or just proximal to this point prior to K1. The intra-arterial pressure preceding K1 was much lower than at the 8-cm location, being somewhat less than 40 mm Hg as compared to approximately 100 mm Hg at the point 2 cm proximally (upper half of figure 1). Thus, just prior to K1 a pressure gradient in excess of 60 mm Hg was present over this 2-cm length of compressed arterial segment. The initial pressure pulse that penetrated to the 10-cm level occurred coincident with K1 and was greatly altered in contour as compared to the pressure pulses recorded proximal to this level. The wave front was exceedingly steep, ending in a spike of very short duration. The diastolic pressure rose during the first eight pulse cycles following K1, which was opposite to its behavior in the segment 2 cm proximal. Although the absolute blood pressure was lower than in the proximal segment at K1, the pulse pressure was greater.

The rate of penetration of the arterial pulse into the proximal region of the compressed segment at suprasystolic cuff pressures varied somewhat in the different subjects, the two extreme examples being shown in figure 2. In all subjects penetration was negligible at a cuff pressure 50 mm Hg above the level of K1. At 20 mm Hg pulsations had progressed into the proximal third of the compressed segment in three subjects and to the mid-segment region in the three others. At a cuff pressure 5 mm Hg above level of K1 the presence of pulsations still was limited to the proximal half of the compressed segment in the former subjects but had moved a few centimeters beyond the midpoint in the latter. The initial pulsation appearing in the distal 2 cm of the compressed segment and beyond was simultaneous with the appearance of the first Korotkoff sound in all subjects.

**Pressure Gradients at Suprasystolic Cuff Pressures**

The pressure gradients in the compressed arterial segment at the level of K1 as well as

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**Figure 2**

Graph showing relationship between the supra-systolic level of cuff pressure shown on the ordinate and the farthest penetration of the pressure pulse into the compressed arterial segment shown on the abscissa. Two examples are shown. See text for further details.
Graph showing systolic intra-arterial pressure on the ordinate, and position with relation to the cuff from which the pressure recordings were taken on the abscissa, in a 31-year-old normal man. Intra-arterial systolic pressure gradients are shown at cuff pressures of 50, 30, and 0 mm Hg, respectively, above the level of the onset of the first Korotkoff sound. Intra-arterial pressures at 50 and 30 mm Hg above K1 were nonpulsatile except near the proximal edge of the cuff.

At 50 and 30 mm Hg cuff pressure above this level are shown in figure 3. The abscissa indicates the location under the cuff, and the ordinate the level of intra-arterial systolic pressure. The gradients were reconstructed from pressure recordings taken at eight successive catheter locations spanning the region from the proximal to the distal edges of the cuff as described in the methods section.

At suprasystolic cuff pressures the pressure gradient increased through the proximal half of the compressed arterial segment; the greater the cuff pressure, the greater was the slope of the pressure gradient (fig. 3). Distal to the midsegment intra-arterial pressure fell at first gradually and then steeply. The region of steepest fall varied somewhat in the different subjects but usually was within the boundaries encompassed by the segment 8 to 12 cm distal to the proximal edge of the 14 cm cuff. The pressure drop occurring in this distal segment varied between 80 and 90 mm Hg in the different subjects.

Thus, at suprasystolic cuff pressures the compression was not uniform, but rather the transmitted pressure was greatest in the middle of the compressed arterial segment and least near the ends where the pressures tended to equilibrate with the intra-arterial pressures existing beyond the respective edges of the cuff.

At the time of K1 there was a positive systolic pressure gradient through the proximal 8 cm of the compressed segment (fig. 3), which averaged 6.7, SD 2.5 mm Hg in the different subjects (table 1). At 10 cm distal to the proximal edge the intra-arterial systolic pressure fell moderately, averaging 10.6,

**Table 1**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Proximal to cuff</th>
<th>At mid cuff</th>
<th>Diff.</th>
<th>Distal to cuff</th>
<th>Diff.*</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.W.</td>
<td>99</td>
<td>108</td>
<td>-9</td>
<td>40</td>
<td>-68</td>
</tr>
<tr>
<td>J.P.</td>
<td>122</td>
<td>130</td>
<td>-8</td>
<td>51</td>
<td>-79</td>
</tr>
<tr>
<td>T.J.</td>
<td>89</td>
<td>97</td>
<td>-8</td>
<td>15</td>
<td>-82</td>
</tr>
<tr>
<td>L.C.</td>
<td>120</td>
<td>126</td>
<td>-6</td>
<td>55</td>
<td>-71</td>
</tr>
<tr>
<td>W.W.</td>
<td>112</td>
<td>114</td>
<td>-2</td>
<td>41</td>
<td>-73</td>
</tr>
<tr>
<td>C.B.</td>
<td>123</td>
<td>130</td>
<td>-7</td>
<td>53</td>
<td>-77</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td></td>
<td>+6.7</td>
<td>-74.3</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td></td>
<td>2.5</td>
<td>5.3</td>
<td>2.4</td>
</tr>
</tbody>
</table>

*Difference between measurement and midsegment systolic pressure.
Pressure Gradients from K1 to Muffling

As cuff pressure was lowered below the level at K1 the systolic pressure gradients gradually diminished (fig. 4). In most subjects systolic pressure gradients disappeared at cuff pressures approximately 10 mm Hg above muffling, although in two a small negative gradient in the distal segment persisted to the level of muffling, when it also disappeared.

The diastolic pressure gradient rose considerably through the proximal half of the compressed segment at the time of K1 (fig. 4). This was due to the fact that cuff pressure closed the midsegment region when the intra-arterial pressure fell below the transmitted cuff pressure. Thus, diastolic pressure in the midsegment at the time of K1 was at the level of effective transmitted cuff pressure. Between 8 and 12 cm distal to the proximal edge of the cuff (in most subjects in the shorter distance of 10 and 12 cm) diastolic pressure fell steeply, averaging 57.2, ± 9.3 mm Hg, in the different subjects. Although diminishing gradually, a steep negative-pressure gradient continued in this segment throughout the period of loud sounds. At a cuff pressure 20 mm Hg above muffling the diastolic pressure gradient through the proximal half of the cuff was positive, averaging 17.0, ± 4.7 mm Hg, and was negative through the distal half, averaging 31.2, ± 7.2 mm Hg (fig. 4 and table 2).

The onset of muffling was accompanied by a distinct change in the pressure gradient, characterized by the disappearance of both the positive gradient in the proximal segment and the negative gradient in the distal.
Diastolic pressure henceforth remained essentially unchanged through the compressed segment (table 2 and fig. 4).

**Effective Pressure Transmitted from the Cuff to the Underlying Artery**

As noted above, the pressure exerted by the cuff on the compressed arterial segment is greatest in the middle of the segment and least at the ends. The difference between intra-arterial pressure in the maximally compressed midsegment and the cuff pressure should provide a measure of the transmitted pressure loss. However, since the occluded segment functions as a closed end-reflecting point, the systolic pressure recorded just proximal to this region may be raised above the level of transmitted pressure because of the conversion of kinetic energy of flow to pressure energy. Nevertheless, the transmitted pressure loss can be estimated during complete occlusion, when pulsatile pressures are not present in the midsegment. At lower cuff pressures it may be estimated from the difference between cuff pressure and midsegment diastolic pressure, since the diastolic level is not significantly influenced by reflections. The beginning of the systolic upstroke in this case represents the level of pressure in the artery capable of opening the occluded segment. Therefore, at supra-diastolic cuff pressures the foot of the pressure pulse recorded in the midsegment region may be used as a measure of the effective transmural pressure being exerted on the artery by the cuff at that moment.

At a cuff pressure 50 mm Hg above K1 the nonpulsatile intra-arterial pressure in the midsegment region averaged 17.2, ±3.8 mm Hg, less than cuff pressure. The magnitude of the difference decreased as cuff pressure

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**Table 2**

Diastolic Pressure Gradients at Cuff Pressures of 20 mm Hg above Muffling and at Muffling

<table>
<thead>
<tr>
<th>Subject</th>
<th>Proximal to cuff</th>
<th>At mid cuff</th>
<th>Distal to cuff</th>
<th>Diff.</th>
<th>Proximal to cuff</th>
<th>At mid cuff</th>
<th>Distal to cuff</th>
<th>Diff.</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.W.</td>
<td>63</td>
<td>79</td>
<td>+16</td>
<td>45</td>
<td>-34</td>
<td>68</td>
<td>68</td>
<td>0</td>
</tr>
<tr>
<td>J.P.</td>
<td>69</td>
<td>92</td>
<td>+23</td>
<td>68</td>
<td>-24</td>
<td>73</td>
<td>79</td>
<td>+6</td>
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<tr>
<td>T.J.</td>
<td>45</td>
<td>65</td>
<td>+20</td>
<td>15</td>
<td>-40</td>
<td>50</td>
<td>53</td>
<td>+3</td>
</tr>
<tr>
<td>L.C.</td>
<td>73</td>
<td>88</td>
<td>+9</td>
<td>44</td>
<td>-38</td>
<td>70</td>
<td>77</td>
<td>+7</td>
</tr>
<tr>
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<td>64</td>
<td>80</td>
<td>+16</td>
<td>57</td>
<td>-23</td>
<td>65</td>
<td>68</td>
<td>+3</td>
</tr>
<tr>
<td>C.B.</td>
<td>65</td>
<td>83</td>
<td>+18</td>
<td>55</td>
<td>-28</td>
<td>66</td>
<td>68</td>
<td>+2</td>
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<tr>
<td>Mean</td>
<td>+17.0</td>
<td>-31.2</td>
<td>+3</td>
<td>66</td>
<td>+2</td>
<td>66</td>
<td>+2</td>
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<tr>
<td>SD</td>
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</table>

*MSP = midsegment nonpulsatile pressure.
†CP = cuff pressure.

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**Table 3**

Difference between Midsegment Nonpulsatile Pressure and Cuff Pressure at Supra-systolic Cuff Pressures

<table>
<thead>
<tr>
<th>Subject</th>
<th>30 mm Hg</th>
<th>30 mm Hg</th>
<th>20 mm Hg</th>
<th>10 mm Hg</th>
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<tbody>
<tr>
<td></td>
<td>MSP*</td>
<td>CP†</td>
<td>Diff.</td>
<td>MSP*</td>
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<tr>
<td>J.W.</td>
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<td>152</td>
<td>-12</td>
<td>120</td>
</tr>
<tr>
<td>J.P.</td>
<td>152</td>
<td>173</td>
<td>-21</td>
<td>131</td>
</tr>
<tr>
<td>T.J.</td>
<td>118</td>
<td>140</td>
<td>-22</td>
<td>107</td>
</tr>
<tr>
<td>L.C.</td>
<td>155</td>
<td>172</td>
<td>-17</td>
<td>136</td>
</tr>
<tr>
<td>W.W.</td>
<td>145</td>
<td>160</td>
<td>-15</td>
<td>125</td>
</tr>
<tr>
<td>C.B.</td>
<td>159</td>
<td>175</td>
<td>-16</td>
<td>141</td>
</tr>
<tr>
<td>Mean</td>
<td>-17.2</td>
<td>-15.3</td>
<td>-14.7</td>
<td>-12.0</td>
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</table>

*MSP = midsegment nonpulsatile pressure.
†CP = cuff pressure.

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Table 4

<table>
<thead>
<tr>
<th>Subject</th>
<th>At K1 (mm Hg)</th>
<th>CP</th>
<th>Diff.</th>
<th>Level of cuff pressure above K1 (mm Hg)</th>
<th>CP</th>
<th>Diff.</th>
<th>At muffling (mm Hg)</th>
<th>CP</th>
<th>Diff.</th>
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<tr>
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<td>105</td>
<td>102</td>
<td>-3</td>
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<td>87</td>
<td>87</td>
<td>0</td>
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<td>F.F.</td>
<td>118</td>
<td>123</td>
<td>5</td>
<td>110</td>
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<tr>
<td>T.C.</td>
<td>79</td>
<td>79</td>
<td>0</td>
<td>83</td>
<td>83</td>
<td>0</td>
<td>65</td>
<td>65</td>
<td>0</td>
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<td>112</td>
<td>122</td>
<td>10</td>
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<td>92</td>
<td>0</td>
</tr>
<tr>
<td>W.W.</td>
<td>101</td>
<td>111</td>
<td>10</td>
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<td>90</td>
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<td>84</td>
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<td>125</td>
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<td>94</td>
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<td>0</td>
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<td>0</td>
</tr>
<tr>
<td>Mean</td>
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<td>109</td>
<td>3</td>
<td>106</td>
<td>106</td>
<td>0</td>
<td>94</td>
<td>94</td>
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</table>

\( *MSDP = \text{midsegment diastolic pressure.} \)
\( +CP = \text{cuff pressure at same instant.} \)

approached K1 and at 10 mm Hg above K1 midsegment intra-arterial pressure averaged 12.0, \( \text{SD} 5.6 \) mm Hg, less than cuff pressure (table 3).

At the time of K1 midsegment intra-arterial diastolic pressure averaged 8.1, \( \text{SD} 1.9 \) mm Hg, less than cuff pressure. Between 30 and 10 mm Hg cuff pressures above muffling the midsegment diastolic pressure was approximately 10, \( \text{SD} 3.5 \) mm Hg, below cuff pressure, and at muffling the average difference was 7.3, \( \text{SD} 2.5 \) mm Hg (table 4). Since the midsegment region represented the area of maximum compression exerted by the cuff on the brachial artery, it was apparent that the cuff pressure was incompletely transmitted to the artery since in all stages of the deflation cycle prior to muffling, cuff pressure was higher than midsegment intra-arterial diastolic pressure.

**Difference between Auscultatory and Direct Indication of Blood Pressure**

In each of the control recordings of intra-arterial pressure preceding each cuff inflation the mean of the respiratory fluctuation in systolic and diastolic pressures were measured. The eight control intra-arterial pressure recordings of systolic and diastolic pressure taken prior to cuff inflation in each subject were averaged and are shown in table 5. Also, the recorded cuff pressurees at the time of the microphone recordings of K1 and of muffling also were measured and the eight readings for each subject were averaged (table 5).

The results indicated that there was close agreement between the average cuff pressure at the time of K1 and the average control level of systolic pressure, the deviation between the two estimates being \(-0.1, \text{SD} 2.2 \) mm Hg, in the six subjects. On the other hand, the average cuff pressure at the time of muffling provided an estimate of diastolic pressure that was significantly higher than the average control direct recording of diastolic pressure. The cuff pressure estimated the diastolic level was 8.7, \( \text{SD} 2.1 \) mm Hg, higher than the intra-arterial diastolic pressure.
DYNAMIC REACTIONS

Table 5

<table>
<thead>
<tr>
<th>Subject</th>
<th>Average* systolic pressure (mm Hg)</th>
<th>Average† cuff pressure at Ki (mm Hg)</th>
<th>Diff. (mm Hg)</th>
<th>Average* diastolic pressure (mm Hg)</th>
<th>Average† cuff pressure at muffling (mm Hg)</th>
<th>Diff. (mm Hg)</th>
</tr>
</thead>
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<tr>
<td>J.W.</td>
<td>103.6</td>
<td>102.3</td>
<td>-1.3</td>
<td>56.2</td>
<td>62.8</td>
<td>+6.6</td>
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<tr>
<td>J.P.</td>
<td>122.8</td>
<td>124.6</td>
<td>+1.8</td>
<td>71.5</td>
<td>83.0</td>
<td>+11.5</td>
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<td>T.J.</td>
<td>93.3</td>
<td>90.1</td>
<td>-3.2</td>
<td>50.0</td>
<td>57.4</td>
<td>+7.4</td>
</tr>
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<td>L.C.</td>
<td>122.3</td>
<td>125.4</td>
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<td>74.3</td>
<td>81.1</td>
<td>+6.8</td>
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<td>110.9</td>
<td>110.1</td>
<td>-0.8</td>
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<td>73.6</td>
<td>+9.9</td>
</tr>
<tr>
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<td>124.4</td>
<td>-0.4</td>
<td>73.5</td>
<td>83.7</td>
<td>+10.2</td>
</tr>
<tr>
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<td>-0.1</td>
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<td>73.6</td>
<td>+8.7</td>
</tr>
<tr>
<td>SD</td>
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*Average of eight recordings of systolic and diastolic pressure, respectively, taken prior to various cuff inflations.
†Average of eight recordings of cuff pressures at time of first Korotkoff sound taken during various cuff deflations.

Discussion

The possibility was considered that the insertion of the catheter may have produced local spasm of the arterial wall, which interfered with the production of normal Korotkoff sounds. Care was used in inserting the needle and catheter to avoid trauma to the artery. The needle was removed immediately after insertion of the catheter, and pressure was applied to avoid hematoma. After waiting 10 minutes the Korotkoff sounds recorded with the catheter in place appeared no different from those recorded prior to the arterial puncture.

There also was no evidence to indicate that the presence of the catheter interfered with normal closure of the compressed arterial segment. If such had occurred, K1 would have been recorded earlier, that is, at higher cuff pressures when the catheter protruded through the midsegment than when it was below it. However, there was no significant variation in cuff pressures at K1 when the catheter tip lay in the proximal or in the distal end of the cuff. As a further check intra-arterial pressures were recorded simultaneously from the contralateral arm in one subject before as well as after insertion of the catheter. The latter record failed to reveal any indication that the catheter altered the Korotkoff sound indications of systolic or diastolic pressure.

To our knowledge a study of the dynamic pressure changes in the compressed arterial segment has not been previously carried out. Bazett and associates recorded pressures proximal and distal to the compressed segment of the femoral artery of the dog but the artery was severed in order to place it within the compression chamber. They noted by inspection that the occlusion was conical and opened out both proximally and distally from the maximally compressed midsegment. This observation is consistent with the presently recorded pressure gradients during occlusion in the compressed segment of man.

The central closure of the compressed segment divides the proximal and distal parts into separate systems that equilibrate with the pressures existing in their respective vasculatures. The first recorded Korotkoff sound always occurs simultaneously with the first pulse that traverses the distal segment. When the first pulse traverses the midsegment at the time of K1 it passes into an arterial segment, which has a pressure drop of approximately 75 mm Hg over a length of only 4 or 5 cm. The steepness of this gradient must induce a marked acceleration of the blood at the moment that the occluded segment opens.

Compression of the brachial artery by an air-filled cuff produces a variety of dynamic reactions, some of which elevate and others reduce the apparent readings. Some of these
influences were recognized as early as 1901 by von Recklinghausen and were further amplified by Erlanger and Wiggers in later years. However, instrumentation was not sufficiently developed to measure their effects experimentally in man.

The present results indicate that the pressure in the cuff was incompletely transmitted to the underlying arterial segment. At suprasystolic cuff pressures the pressure within the compressed arterial segment was lower than the cuff pressure. This was the case even in the completely occluded midsegment region. Between Ki and muffling the beginning of the systolic upstroke in the arterial midsegment occurred at an intra-arterial pressure somewhat lower than the cuff pressure. This indicated that the occluded segment was opening at an intra-arterial pressure below that exerted by the cuff. The inefficient transmission of pressure energy probably was a consequence of the resistance imposed by the viscoelastic tissues of the arm.

A pressure loss occurring between cuff and artery would produce a falsely high indication of blood pressure by the indirect method. This appeared to be the case with regard to the muffling criterion for estimating diastolic blood pressure. At muffling the beginning of the arterial upstroke in the midsegment region occurred at an intra-arterial pressure averaging 7.3 mm Hg lower than cuff pressure. This approximated the discrepancy found between directly measured intra-arterial diastolic pressures taken prior to cuff inflation and that taken by the auscultatory method using muffling as the diastolic criterion.

Since transmission of cuff pressure also was incomplete at Ki, why was there good agreement between the direct and indirect measurements of systolic blood pressure? We believe that an additional phenomenon was operative at Ki (but not at muffling), which acted in the direction of lowering the indirect reading. At the time of Ki the pressure exerted on the artery was sufficient to produce occlusion throughout most of the pulse-wave cycle. Only the peaks of the pressure pulses were able to penetrate into the midsegment. Because they were transient and of small magnitude (top half of figure 1) they contained too little pressure energy to open the entire length of the collapsed segment until the effective pressure exerted by the cuff fell significantly below the level of the intra-arterial systolic pressure. Ki always occurred simultaneously with the passage of the first pulse wave which passed completely through the compressed segment. In order to cause such complete penetration a pulse pressure of sufficient amplitude to drive the pulse wave through the occluded segment was required. The extent of the delay in penetration will be dependent on the length of the occluded segment, which in turn is a function of cuff width. This compensatory phenomenon cannot occur at muffling when the cuff pressure is near the level of diastolic pressure and pulse waves of normal amplitude are passing through the compressed segment.

Another factor that possibly could influence the indirect readings of blood pressure is wave reflections, since the arterial occlusion acts as a closed end-reflecting point. Reflections would elevate the systolic peaks proximal to the occlusion but would have no effect on the diastolic portions of the pulse wave. The initial snapping portion of the Korotkoff sounds is associated with opening of the occluded segment. As muffling is approached the occluded segment opens near the foot of the pulse wave which is unaffected by wave reflections. However, at Ki reflections could influence the level at which the first pulse passes through the occluded segment since reflections elevate the systolic peaks and could lead to a falsely high estimate of systolic blood pressure.

Bazett and associates have indicated that the conical shape of the arterial occlusion probably would prevent major reflections from developing. At the time of Ki there was a slight positive systolic pressure gradient in the proximal half of the cuff (table 1 and fig. 3), which may have been caused by
wave reflections. However, if significant reflections were present, they were not reflected back for any considerable distance since the systolic pressure recorded just proximal to the cuff at the time of Kl was no higher than that recorded prior to cuff inflation. Whatever influence reflections might have had in elevating the systolic blood pressure at the time of Kl their effect on the indirect reading appeared to be compensated for by the delay in penetration of the diminutive pulsations through the occluded midsegment.

The present results indicate that when muffling is used as the criterion the diastolic pressure reads higher by the indirect method than the intra-arterial diastolic blood pressure measured directly. Simultaneous recordings of intra-arterial and auscultatory blood pressures have been carried out by numerous other investigators. A minority of observed muffling at cuff pressures 3 to 5 mm Hg lower than intra-arterial diastolic, whereas the majority found that muffling occurred at cuff pressures usually averaging 8 to 10 mm Hg higher than the intra-arterial diastolic pressure. For this reason some authors believe that disappearance of sound provides a better estimate of diastolic pressure.

Disappearance of sounds could not be assessed as a criterion of diastolic pressure in the present study because in some recordings sounds of low frequency and intensity persisted to levels far below diastolic pressure. It was apparent that the point selected for disappearance will vary with the acuity of hearing of the observer and possibly also with the type of stethoscope head used. In addition, it is known that following exercise or reactive hyperemia and in patients with aortic insufficiency clearly audible, post-muffling sounds may extend far below diastolic levels of intra-arterial pressure. Thus, muffling rather than disappearance seems to provide a more dependable end point for estimating diastolic pressure.

Although further investigation is needed to determine the point in the sequence of Korotkoff sounds that most accurately represents the diastolic pressure, the present study indicates that muffling is the acoustic representation of the disappearance of a diastolic pressure gradient through the compressed segment. However, because of the loss in transmission of pressure from the cuff to the artery, the indirect reading will be too high. The amount of correction needed requires further study. The present results as well as the majority of reports in the literature, comparing the auscultatory and direct methods, suggest that approximately 9 mm Hg subtracted from the manometric reading at muffling may provide the closest estimate of intra-arterial diastolic blood pressure using a standard cuff in nonobese adults.

References


100 Years Ago

Routine Temperature of Patients

For the last sixteen years my attention has been uninterruptedly directed to the course pursued by the temperature in diseases of various kinds. The thermometer has been regularly employed at least twice daily, and in febrile patients from four to eight times a day, and even oftener, in special circumstances, for all the patients in my wards. I have also experienced the applicability of this method of investigation in very numerous cases in private practice. In this way I have gradually got together a material which comprises many thousand complete cases of thermometric observations of disease, and millions of separate readings of the temperature. The more my observations were multiplied the more firmly rooted did my conviction become of the unparalleled value of this method of investigation, as giving an accurate and reliable insight into the condition of the sick.—C. A. WUNDERLICH: Preface of First Edition. On the Temperature in Diseases: A Manual of Medical Thermometry. London, The New Sydenham Society, 1871 [ p. v].

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Dynamic Reactions Produced by Deflating a Blood Pressure Cuff
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