3 Fat Arms, Cuff Size and Blood Pressure Reassessed

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Controversy continues over the effect of cuff size when recording blood pressures. Maxwell *et al.* (Lancet 1982, **II**:33-35) claimed to show substantial errors due to cuff size in a large series without the use of random zero sphygmomanometers. Our study attempts to answer the question 'what systematic difference, if any, in systolic (SBP) or diastolic (DBP) blood pressure readings is produced by a standard compared with a large cuff on arms of various circumferences (AC)'?

Analysis problems include regression to the mean, order effects from different cuff sizes, the height of the BP etc. In our study two subject groups were studied: (a) hospital outpatients and (b) from general practice. Combined results were analysed, based on AC, using the large cuff first in groups 1 and 3, and the small cuff first in groups 2 and 4. Four sequential readings were made, two with each cuff, small and large alternately. Power estimates of 90% suggested 80-120 subjects were required. Results (mean ± s.d.) are shown in Table 1.

Hence, overall readings with the small cuff in whatever sequence were consistently higher than with the large cuff. Data from analysis of covariance including age, heart rate, AC, order of use, cuff used and level of BP are presented.

In conclusion, as intra-arterial BP levels are generally lower than indirect records, to approach such levels these results suggest that cuff dimensions for routine sphygmomanometry should be revised upwards.

Table 1.

	Age (years)	Arm circumference (cm)	Difference (small > large cuff reading; mmHg)		Mean of two blood pressure readings with each cuff	
			SBP	DBP	Small	Large
Group 1 (n = 19)	45.9 ± 9.5	36.8 ± 2.1	+2.76 ± 8.4	+5.35 ± 5.9	SBP 148.7 ± 20.4	145.9 ± 18.6
					DBP 95.7 ± 12.6	90.1 ± 15.2
Group 2 (n = 21)	54.2 ± 7.5	36.2 ± 2.2	+5.6 ± 5.7	+6.0 ± 7.1	SBP 150.9 ± 16.8	145.9 ± 18.9
					DBP 97.4 ± 9.8	91.5 ± 9.9
Group 3 (n = 28)	52.4 ± 8.5	31.3 ± 1.4	+1.81 ± 7.9	+4.04 ± 5.48	SBP 151.1 ± 26.1	150.1 ± 25.8
					DBP 91.3 ± 11.1	87.4 ± 9.8
Group 4 (n = 23)	57.5 ± 9.7	31.1 ± 2.5	+5.47 ± 7.1	+3.8 ± 4.2	SBP 155.9 ± 18.3	148.7 ± 16.8
					DBP 88.6 ± 8.1	83.2 ± 10.3

4 Adrenaline and Hypertension in Man

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Three years ago, the adrenaline hypothesis of hypertension was presented to the Society by (Brown MJ, Macquin I: Lancet 1981, **II**:1079-1082.) They proposed that uptake of circulating adrenaline into sympathetic nerve endings led to the 'conversion' of adrenaline from hormone to neurotransmitter; and that the resulting concentration of adrenaline in synaptic clefts (when adrenaline is re-released by sympathetic nerve firing) is sufficient to facilitate noradrenaline release by activation of the positive feedback loop through the presynaptic β-adrenoceptor. Proposed investigations of the hypothesis included (a) experiments to show whether adrenaline can increase noradrenaline release in man, and (b) a comparison of adrenaline secretion in hypertensive and normotensive subjects. These investigations have now been completed.

In (a), adrenaline infusions were performed for 60-80 min at 0.05 μg/kg/min in six healthy volunteers. Infusion of adrenaline alone causes no change in plasma noradrenaline concentration and this was postulated to be due to the simultaneous stimulation by adrenaline of the inhibitory and facilitatory feedback loops through presynaptic α_2 and β_2 receptors, respectively (Majewski H, Rand MJ: Eur J Pharmacol 1981, **69**:493-498.) After administration of the selective β_2-receptor antagonist, ICI 118551 5 mg p.o., adrenaline reduced plasma noradrenaline from 0.20 ± 0.020 to 0.15 ± 0.018 ng/ml ($P < 0.01$). After administration of the selective α_2-receptor antagonist, idazoxan, 0.2 mg/kg i.v. adrenaline elevated plasma noradrenaline from 0.25 ± 0.03 to 0.37 ± 0.05 ng/ml ($P < 0.01$). Idazoxan had no effect on adrenaline induced changes in blood pressure, so therefore this rise cannot be baroreceptor mediated.

For (b), four consecutive 24-h urine samples were collected from 270 untreated hypertensives who were participating in the MRC mild blood pressure trial, and from 270 age/sex matched normotensive subjects registered with the same general practitioners. Aliquots of these urines were analysed blind by the double-isotope enzymatic technique for adrenaline and nor-