BORDERLINE HYPERTENSION—A CRITICAL REVIEW

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INTRODUCTION

THIS REVIEW explores a poorly-defined area of borderline abnormal blood pressures. Different definitions as to what is normal and abnormal blood pressure traditionally apply sharp cutting lines, but they differ from study to study. No one would question that consistent readings below 130/80 represent normal blood pressure, and those always above 160/100 at any age require medical attention. However, the significance of readings falling between these limits may be questioned. The situation becomes more confused when the readings oscillate between the entirely normal and quite abnormal range.

The medical significance of this condition is even more uncertain. Evidence from the literature based on heterogeneous data ranges from a tentative indictment of borderline hypertension as a precursor of hypertensive disease [1] to a view supporting a more favorable outlook [2].

This report will review some of the evidence on the epidemiology, clinical characteristics, and pathophysiology of borderline hypertension. Since the nomenclature and type of measurement used in different studies are variable, we will define borderline hypertension as broadly as possible. We will review papers describing subjects in whom:

A. Blood pressure readings over 150/90 were intermittent and occasional normal readings were obtained.

B. Average or single readings were below 160/100 but above 150/90.

C. There was no evidence of target organ damage.

Exceptions will be made for some important papers not adequately characterizing the population but dealing with subjects we believe had borderline hypertension.

The traditional division into 'established' and 'labile' blood pressure will not be pursued in this review, chiefly, because the lability can not be defined accurately and it depends greatly on the number and circumstances of the determination.

If the subjects described in a paper fit our criteria, we will call them 'borderline hypertension' irrespective of the original nomenclature. To facilitate this review data on definitions, characteristics of the population and methods of measurement in the major papers are not presented in the text.

TABLE 1. PREVALENCE OF BORDERLINE HYPERTENSION (PERCENTAGE)

Author	Blood pressure definition	Below 30		4th Decade	5th Decade	6th Decade	Over 60
		(15-19)	(25-29)	(35-39)	(45-49)	(55-59)	(61-69)
[4] Boe et al							
Females	150-160/90-95	1.04	3.62		11.63	16.69	10.61
Males	150-160/90-95	3.01	7.78	10.72	12.89	13.91	13
		(20-29)		(30-39)	(40-49)	(50-59)	(60-79)
Tecumseh Study	Diastolic 90-99						
Males		8.5		10.0	17.4	10.1	13.6
Females		2.6		5.5	10.6	14.0	13.5
		(14-29)		(30-44)			
[6] Mathewson et al	Systolic 140-159						
Males		10.8		13	12.5	—	—
[7] Ostfeld	Systolic 140-159				(40-44) 21	(50-55) 29	—
Males	Diastolic 90-99	—		—	(40-55) 26	—	—
[8] Kooperstein et al	(a)	(20-24)					
Males	Systolic 140-159	24.6		—	—	—	—
	(b)						
	Diastolic 90-94	6.1					
	Combined (a) & (b)	3.2					
[9] Atanassowa	Systolic 150-165	(20-25)					
Predominantly Males	Diastolic 90-105	1.3		—	—	—	—

Table 1 continued—

Author	Blood pressure definition	Age				
		Below 30	4th Decade	5th Decade	6th Decade	Over 60
[10] Thomas et al Males	Diastolic 90–99 after 15–30 min rest during a single exam	(21–31) 1	—	—	—	—
[11] Thomas Males	Results of all observations over 4 yr. 140–159/90–95 at one or more occasions.		(39 av.) 7.9	—	—	—
	160–95 or more but occasionally lower		25.9			
[12] Heyden et al Both sexes	Above 140/ 90 Av. 145/ 90	11	—	—	—	—
[13] Moeller et al Females	Above 140/ 90 followed by lower readings	—	—	—	(av. 51) 29.4	—
Males	Above 140/ 90 followed by lower readings				25.8	—

Table 1 continued—

Author	Blood pressure definition	Below 30	4th Decade	5th Decade	6th Decade	Over 60
				Age		
[3] Glock et al	Diastolic above and below 90 never over 100 in daily readings over a period of 3 weeks	—	—	(21–51) 23	—	—
[14] Eich et al Males	Above 150/90 followed by normal readings	(19–39) 4.1	—	—	—	—
[15] Diehl Males	Mean of 6 systolic readings 140–145 a.m. p.m.	4 10	— —	— —	— —	— —
[16] Harburg et al Female	140–159/90–94	—	—	(Av. 43) White High stress 14 Low stress 14 Black High stress 16 Low stress 13	—	—
[16] Harburg Male	140–159/90–94	—	—	White High stress 16 Low stress 19 Black High stress 21 Low stress 12	—	—

Table 1 continued

Author	Blood pressure definition	Age				
		Below 30	4th Decade	5th Decade	6th Decade	Over 60
[17] Thomson Both sexes	Yearly observations over 15 yr Blood pressure above 140/90	—	—	—	Calculated av. age 52 Occasional peak Systolic 16.8 Diastolic 16.3 Frequently elevated Systolic 13.8 Diastolic 14.4	—
[18] Kannel *et al* Males Females	First casual 140/90–160/95		(30–49) 38.8 27.2	—	(50–62) 36.6 42.2	—
[19] Levy Males	Systolic >150 or diastolic >90 followed by readings ≤150 or ≤90	(25–29) 5.9 (30–34) 9.0	(35–39) 10.4 (40–44) 12.7	(45–49) 16.2 (50–55) 18.6	(55–59) 18.5	—
[20] Rogers *et al* Males	Systolic: varying from 140–160 Diastolic: varying from 95–110	(21–45 mostly in 20s) 14	—	—		—
[21] Boynton & Todd	Systolic >140 males Systolic >140 females Diastolic >90 males Diastolic >90 females	(16–30) 7.36 1.12 5.87 2.18	—	—	—	—
[1] Stamler	Diastolic 90–99	—	(30–39) 11	—	(50–59) 14	—

EPIDEMIOLOGY OF BORDERLINE HYPERTENSION

Prevalence

The data on prevalence of borderline hypertension are beclouded by numerous interacting variables—among these are age, definition of the condition, sex, anthropometric measures, number and method of blood pressure readings. Glock, for example [3], took daily pressures over 3 weeks and found that as much as 23 per cent of the subjects had at some time borderline diastolic readings. If the first casual blood pressure in field studies is used [18] the prevalence of borderline hypertension can be as high as 42 per cent. Table 1 is an attempt to summarize prevalence data found in the literature. The majority of papers allow for a few general conclusions.

1. The prevalence of systolic borderline hypertension across all ages above 20 is 10 per cent or more.
2. Diastolic borderline hypertension is less prevalent than the systolic.
3. Prevalence of borderline hypertension appears to increase with age.
4. Prevalence of borderline hypertension among females below 50 is smaller than in males.

It appears therefore, that borderline hypertension is by no means a rare condition. By age 60, the prevalence may be quite close to 40 per cent. If indeed, as it was frequently suggested, borderline hypertension represents 'prehypertension', then one has to agree with Fejfar [22] that: "the high prevalence and obvious public health importance of this syndrome requires intensive clinical and epidemiological studies which should preferably begin before (age) 20".

Incidence

The information available for incidence of borderline hypertension is sketchy at best. As can be noted from Table 2, the incidence rate in the middle-aged population is around 1 per cent per year of observation. There is also some indication in these data that the incidence of borderline hypertension increases with age. Again, if borderline hypertension is taken seriously, and there is a consistent yearly recruitment of new cases involving 1 per cent of the total population, this would represent a sizeable public health problem.

Natural History

In this portion of the paper most of the remarks on the natural history will relate to the question of whether patients with borderline hypertension tend subsequently to develop established hypertension.

As Table 3 indicates, a certain portion of the population with borderline hypertenson does become hypertensive. For the majority of the studies listed, the per cent of patients with borderline hypertension becoming hypertensive can be compared with a normotensive group followed simultaneously. Care must be used in interpretation, since a variety of populations by age, length of follow-up, and definition of conditions is shown.

Definitions of hypertension at the follow-up are quite different. While some studies require clinically established hypertension with evidence of target organ damage [1, 8] others will be satisfied with a single 'abnormal' blood pressure reading at the end [26, 32]. Occasionally the cutting line from 'borderline' at the onset and 'hypertension' on follow-up are quite close, and only 1 mm of blood pressure rise over the years will bring the former 'borderline' into the hypertensive category [28,31].

TABLE 2. INCIDENCE OF BORDERLINE HYPERTENSION

Author	Definition	Av. length of follow-up (yr)	Age	Incidence of borderline hypertension (%)
[23] Mathewson Males	Initial 139/89 or less Final 140–159/90–94	17 17	Initially 15–29 30–44	13.3 20.3
[24] Chapman et al*	140–159/90–94	12	21–39	17.8
[8] Kooperstein et al Males	Initial systolic <140 Final systolic 140–159	10+	Initially 20–24	8.1 systolic
	Initial diastolic <90 Final diastolic 90–94			3.3 diastolic

Table 2 continued—

Author	Definition	Av. length of follow-up (yr)	Age	Incidence of borderline hypertension (%)
[17] Thomson* Both sexes	Previously normotensive. Later diastolic, frequently >90	15	At the appearance† 30–34 35–39 40–44 45–49 50–54 55–59 60+	0.36 1.44 1.76 4.34 4.72 3.07 1.73
[12] Heyden et al Both sexes	Initial normal Final 140–159 or 90–94	7	Initially 15–25	13
[19] Levy et al Males	Reaching > 150/90 followed by a normal value	5	At the appearance 25–29 30–34 35–39 40–44 45–49 50–54 55–59	5.9 9.0 10.4 12.7 16.2 18.6 18.5

*From normal status to mild hypertension status.
†Of borderline hypertension.

TABLE 3. NATURAL HISTORY OF BORDERLINE HYPERTENSION

Author	Initial pressure		Final pressure ('Hypertension')	Av. length of follow-up (yr)	Av. initial age (yr)	Per cent hypertensive	
	Normal	Borderline				Initially normal	Initially borderline
[25] Paffenbarger et al	Systolic <140	Systolic 140–149	'Doctor diagnosed high blood pressure'	22–31	19	7.3	20.2
Males	Diastolic <79	Diastolic 90–99	'Doctor diagnosed high blood pressure'			6.6	20
[26] Vancura Sex unknown	None	Systolic 100+age transient	Systolic: 120+age sustained	20	15–24 25–34 35–44	None	43.1 60 57.8
[27] Diehl Males	Systolic <130	Systolic (a) ≥130 on one occasion only (b) intermittently ≥130	Systolic ≥140	7	20	0 0	5 8
[28] Vancura et al Sex unknown	—	Av. casual: 180/102	'Permanent hypertension'	10	25	—	31.7
	—	Av. resting 156/181	'Permanent hypertension'	15 20		— —	38.25 34.24
	—	Av. casual: 180/102	'Permanent hypertension'	10	25–34	—	49.9
	—	Av. resting 141/84	'Permanent hypertension'	15 20		— —	50.7 62.4
	—	Av. casual: 178/98	'Permanent hypertension'	10 15	35–44	— —	39.8 48.0
	—	Av. resting 156/85	'Permanent hypertension'	20		—	57.8

Table 3 continued—

Author	Initial pressure		Final pressure ('Hypertension')	Av. length of follow-up (yr)	Av. initial age (yr)	Per cent hypertensive	
	Normal	Borderline				Initially normal	Initially borderline
[28] Vancura (continued)	— — —	'Transient hypertension'	Diastolic >110	10 15 20	15–44	— — —	5.1 10.4 18.9
[29] Evans Males	<180/100 (later accepted for military service)	>180/100	Diastolic 100 or more	10	17–40	4	18
[30] Palmer Males	Systolic <140	Systolic >140	Systolic >150	10	21	2	10
[31] Hines Sex unknown	Systolic <140/ <85	Systolic 140/85– 160/100	>160/>100 One or several times	10 20	20–80	2.3 3.8	59.7 82.5
[32] Julius et al Males	<140/<90	>140/>90	Systolic >150 and/or diastolic >90 at age 40	20	20	12	26
[23] Mathewson Males	<139/89	140/90 to 159/94	>160/95	15–20	15–29 30–44	6.1 12.8	10.8 25.5

Table 3 continued

Author	Initial pressure		Final pressure ('Hypertension')	Av. length of follow-up (yr)	Av. initial age (yr)	Per cent hypertensive	
	Normal	Borderline				Initially normal	Initially borderline
[8] Kooperstein *et al* Males	Systolic <140 Systolic <140 Diastolic <90	Systolic 140–159 Systolic 140–159 Diastolic 90–94	'Clinical essential hypertension' Systolic >160 Diastolic >95	10+	20–24	1.0 1.2 2.3	5.0 8.7 7.5
[1] Stamler *et al* Males	Diastolic <90	Diastolic >90	Diastolic >95 at age 50	20–30	20–30	9.5	24.6
[12] Heyden *et al* Both sexes	'Normotensive'	>140/90 Av. 145/90	3 readings >160 or 95	7	21	0	40
[19] Levy *et al* Males	Always <150 and <90	Systolic >150: Systolic >150 or Diastolic >90 or Diastolic >90 sustained followed by reading ≤150 or ≤90		5	25 30 35 40 45 50 55	0.05 0.09 0.17 0.26 0.57 0.97 1.49	0.24 0.25 0.56 1.12 2.20 3.32 4.80
[90] Volkov	<129/89 age 39 <139/89 age 59	Elevated casual followed by a normal resting pressure	'Hypertensive disease'	6 months to 4 yrs	majority 20–49	0.6	8.7

In essence, this means that a subject with borderline hypertension had only to maintain his previous reading to be later called hypertensive.

Another peculiarity of the data in Table 3 is that the majority of papers report on males.

With all reservations, it is safe to say that the risk of 'hypertension', as defined by different authors, in the initially borderline group is at least twice (and in some studies much higher) that of the normotensive group. However, the absolute level of risk is not very great since the rate of development of hypertension is small in the general population. Studies claiming exceedingly high risk use either an unacceptable definition of hypertension [26], or lack control on sex and age [31]. If indeed 82 per cent of patients with borderline hypertension later develop 'sustained' hypertension, as Hines claims, other studies would have no difficulties in confirming such evident and overwhelming risk.

Mortality and morbidity

If borderline hypertension really leads into established hypertension this should be reflected by mortality and morbidity data since clearly established hypertension carries an increased mortality. Review of the existing evidence is given in Table 4. The papers are not too numerous and fall into 2 quite different categories. The first group are studies based on individual follow-up of a group of subjects with borderline hypertension. Some of these utilize their own control group [12, 17, 18, 36], whereas, one study utilizes mortality rates for the general population as a standard of comparison [33]. Another group of reports stems from the life insurance industry and treats borderline hypertension in terms of excess mortality over the standard actuarial risk [34, 35]. Out of 5 studies utilizing longitudinal individual follow-ups, 4 demonstrate an excess mortality among patients with borderline hypertension and this excess appears somewhat higher in the middle age group. Mathisen's study is the only one to find mortality rates among patients with borderline hypertension to be of the same magnitude as in the general population [33]. Nevertheless, the cumulative survival rates in this study are minimally below those of the normal population.

From the life insurance data, borderline hypertension uniformly appears to carry a higher risk. This data might be evaluated with a grain of salt, since there are at least 3 important sources of error.

1. The blood pressure measurement at entry is not properly standardized.
2. The physicians may tend to report the lowest possible pressure in order to enroll their subjects at lower rates.
3. The validity of the actuarial calculations of the standard risk may be questioned.

However, the consistency and reproducibility of this material in different life insurance studies is impressive, and the risk in all age groups appears to be double that of the normal.

The specific morbidity from cardiovascular disease in patients with borderline hypertension is increased as can be seen from Table 5. All reports reviewed appear to be in agreement in this respect.

Rate of blood pressure rise with age

Another investigative approach to the question of whether subjects with borderline hypertension tend to develop established hypertension is to observe the blood pressure

TABLE 4. MORTALITY IN BORDERLINE HYPERTENSION

Author	Blood pressure definitions	Age at entry	Yr of observation	Deaths Initially borderlines	Deaths Initially normotensive	Borderline/ normal ratio	Remarks
[33] Mathisen et al Males	Previous hypertension. Diastolic ≤95 at entry	Av. 37 all under 46	10 16 22	10.7* 20.3 21.4	5.4* 14.6 23.8	2.0 1.4 0.9	*Deaths/1000 yr of observation
Mathisen Females	See above	Av. 38 all under 46	10	7.7	4.5	1.7	**Compared to av. mortality in the general population
[34] Lew Males	BP 152/95	35	10 15 20	9.1 17 27.6	3.4* 6.5 11	2.7** 2.6 2.5	*No normotensive controls. Compared to standard risks mortality
	BP 152/95	45	10 15 20	16.9 29.1 43.9	7.9 14.1 22.5	2.1 2.0 1.9	**Per cent annual deaths.
	BP 152/95	55	10 15 20	32.5 53.1 73.7	15.9 28.2 44.0	2.0 1.9 1.7	
[35] MacKenzie, Shepherd et al	Av. systolic previous systolic 145–154 followed by 135–144	29 30–39 40–49 50–59	Maximum 16 yr expressed as yr of observation	101 201 177 196	100* 100 100 100	1 2.0 1.7 1.9	*Calculated from tables of expected death rates for standard risk
MacKenzie, Shepherd et al	Diastolic 91–95	30–39 44–49 50–59	See last entry	111 79 186	100* 100 100*	1.1 0.79 1.86	
	Diastolic 96–100	30–39 44–49 50–59		147 216 192	100* 100 100	1.47 2.16 1.92	*Calculated from tables of expected death rates for standard risk

Table 4 continued—

Author	Blood pressure definitions	Age at entry	Yr of observation	Deaths			Remarks
				Initially borderlines	Initially normotensive	Borderline/ normal ratio	
[36] Levy et al Males	BP>150 or 90 followed by normal readings	25–59	1–20 expressed as person-years observed			2.2	Cardiovascular deaths. Age adjusted ratio of expected and observed deaths based on deaths in the whole population at study
[19] Levy et al Males	BP>150 or 90 followed by normal readings	25–29 30–34 35–39 40–44 45–49 50–54 55–59	1–5 in each age group accounted as years of observation	0.0 0.2 1.2 1.6 2.5 4.9 8.9	0.2* 0.1 0.1 0.4 1.2 2.1 5.2	0.0 2.0 12.0 4.0 2.1 2.3 1.7	These are deaths only from cardiovascular renal causes. *Deaths/1000 yr of observation

Table 4 continued—

Author	Blood pressure definitions	Age at entry	Yr of observation	Deaths — Initially borderline	Deaths — Initially normotensive	Borderline/normal ratio	Remarks
[17] Thomson Both sexes	More than 50% of diastolics >90	40+	15 yr	14.5	5.2	2.8	Deaths as per cent of the population at risk
Blood pressure study (1939)	BP $\dfrac{143\text{–}147}{94\text{–}98}$	?	Maximum 13 yr	165	100	1.65*	*Calculated from tables of expected death rates for standard risk.
quoted by Lew	BP $\dfrac{148\text{–}157}{94\text{–}98}$	?		190	100	1.90	
Build and BP study (1959) quoted by Lew	BP $\dfrac{138\text{–}147}{93\text{–}97}$	?	Maximum 19 yr	199	100	1.99	
	BP $\dfrac{148\text{–}157}{93\text{–}97}$	?		224	100	2.24	
[12] Heyden et al Both sexes	>140/90 Av. 145/90	21	7	9.1	0		Death as per cent of the population at risk
[18] Kannel et al Both sexes	141–159/91–94	30–62	14	100	53	1.99	Sudden deaths only. Age adjusted ratio of expected and observed deaths based on deaths in the whole population at study.

TABLE 5. MORBIDITY IN BORDERLINE HYPERTENSION

Author	Blood pressure definition	Type of morbidity	Length of follow-up	Age initially	Border-line/ normal ratio
[12] Heyden et al Both sexes	BP > 140/90 Av. 145/90	Sustained hypertension with cardiovascular complications	7 yr	21	20%–0%
[19] Levy et al Males	BP > 150 or 90 followed by normal readings	Disability and retirement for cardiovascular causes	5 yr periods	30–34 35–39 40–44 45–49 50–54 55–59	3.5 3.8 3.7 2.3 2.0 1.7
[1] Stamler et al Males	Diastolic > 90 (20% readings > 100)	Hypertension heart disease	20 yr 30 yr	30–39 below 30	3.0 4.0
[17] Thompson Both sexes	Frequently, but not always, diastolic > 90	Cardiovascular compli-cations (CHF, angina, myocardial infarction, ECG changes)	15 yr	below 55 above 55	2.0 1.8
		Strokes	15 yr	below 55 above 55	4.0 3.0
[6] Mathewson Males	140/90 to 159/94	Coronary heart disease	Av. 10 yr exposure	15–44	1.35
[18] Kannel et al Males Females	First casual 140/90–160/95 First casual 140/90–160/95	Coronary heart disease Coronary heart disease	14 yr 14 yr	below 50 above 50 below 50 above 50	1.69 1.56 1.63 1.95

trends over periods of years. Average blood pressure in the USA increases with age. If patients with borderline hypertension, already close to abnormal values, were to stay at the same level, or become normal, their blood pressure over the years should actually increase less than in the rest of the population. However, the opposite seems to be the case. Oberman et al [37] have shown that normotensive subjects with initial blood pressure in the upper quintile were found 24 yr later to maintain high readings. This underscores the importance of the initial level in determining future blood pressure readings. In the same vein are data recently presented by Thomas [11]. At the beginning subjects were divided into quintiles of the distribution of the resting systolic and diastolic blood pressure. The observation ranged from 8 to 21 yr; 1.4 per cent developed clinical hypertension and 7.9 per cent transitory-non-established hypertension. Eighty-one per cent of those with clinical hypertension and 68 per cent of those with later transitory hypertension had initial readings in the upper quintile. Since the mean value for the upper quintile was rather low, this study again indicates that higher initial readings, even within the so-called normotensive range, predict a later, steeper blood pressure rise. Miall et al [38] measured blood pressures of a random sample of 2 populations in South Wales and re-examined them after 4 and

10 yr. Their individuals with borderline systolic blood pressure between 160–180 had a steeper blood pressure rise than subjects with lower values.

Harris [39] studied records of annual health exams of employees of a life insurance company. The upper tertile subjects with a mean systolic pressure of 135 at age 25–29, had a steeper blood pressure rise with aging than those with lower systolic pressures. By age 49, av. readings of this group exceeded 150 mm Hg. The same was true for the upper tertile of diastolic blood pressure at age 25 (82 mm Hg). By age 40, they reached an av. diastolic reading of 90, continued to climb until age 45, and then leveled off at values around 95.

Conclusions

1. Borderline hypertension is not a rare condition, and its prevalence and incidence increases with age. The prevalence among young and middle aged females is lower than in males.
2. The risk of developing future hypertension in subjects with borderline hypertension is higher than normal. However, the absolute level of this risk is not extreme.
3. The mortality and cardiovascular morbidity among subjects with borderline hypertension significantly exceeds the rates in normotensive subjects. Though significant, this difference is not dramatic.
4. There is some evidence that over a period of years, subjects with borderline hypertension tend to show a bigger than normotensive rise of the blood pressure.

BLOOD PRESSURE LABILITY AND VASCULAR REACTIVITY IN BORDERLINE HYPERTENSION

Excessive blood pressure lability should be taken to mean that there are wide spontaneous fluctuations of the blood pressure in a person as compared to the naturally occurring changes in a group of normal subjects.

On the other hand, vascular reactivity in a clinical sense may be defined as the change in blood pressure in response to a defined stimulus. It is widely believed, but clearly a misconception, that persons who are 'hyperreactors' respond excessively regardless of the kind of stimulus applied. In line with this belief, Hines [40, 41] implied that vascular hyperreactors also have a higher spontaneous lability of the blood pressure due to the excessive response to 'internal' and 'external' stimuli. This concept was actually never tested. Therefore, the evidence on blood pressure lability and vascular reactivity in borderline hypertension in this review will be examined separately.

Thacker [43] found a higher casual to resting difference in the blood pressure in college students with borderline hypertension. Robinson and Brucer [44] reported increased blood pressure variability in repeated annual examinations of policyholders when those entering who had a systolic less than 120 were compared to those with a higher pressure. In both studies, findings of higher spontaneous blood pressure variability among patients with borderline hypertension are subject to serious objections. In the first study [43], the time between the 'casual' and 'resting' measurements has not been defined and the subjects with 'high' readings may have been given longer periods to achieve the resting condition. In the second study [44], in the lower pressure group the upward variability of the blood pressure was limited by definition

(not above 120), whereas, no such restrictions were imposed on the high pressure group.

Conclusions to the contrary were reached by Diehl [15] who obtained morning and evening readings over 6 days in 100 students. There was no correlation between the mean systolic blood pressure and variability of the blood pressure oscillations. Similar results can be construed from a study by Glock et al [3] based on daily readings over a period of 3 weeks.

Hines frequently stated that subjects with 'labile' blood pressure were also 'cold pressor hyperreactors' [45, 42, 41] but did not offer convincing data thereby opening doors for a long standing controversy. Thacker [43] found higher cold pressor responses in young students with mild and predominately transient blood pressure elevations. These findings were not substantiated in later studies. Thomas et al in 1961 [10], actually found an inverse relationship between baseline blood pressure readings and cold pressor response in college students. Much in the same vein are the results by Eich and Jacobsen [14]. The distribution of casual and resting blood pressure among the normo and hyperreactors to cold pressor were practically identical. The same results were obtained by Cuddy et al [46] in patients with 'labile' hypertension. Recently, Frohlich et al described a group of subjects with hyperreactive beta adrenergic receptors [47]. A substantial proportion had a borderline blood pressure elevation. Their cold pressor response did not differ from the normals. A further extension of these negative studies could be found in papers dealing with normotensive populations [48, 49] where again no relationship between baseline blood pressure levels and cold pressor responsiveness were found.

It was earlier proposed that young subjects who are prone to later hypertension may show an exaggerated blood pressure response to exercise [50]. Newer exercise studies with intraarterial pressure recordings are now available [51–54]. In none of these studies did subjects with borderline hypertension respond to the stimulus of exercise with an exaggerated rise of blood pressure.

Vascular reactivity to the stimulus of upright tilting was also investigated [55]. Patients with borderline hypertension showed a rise whereas in normotensive controls the blood pressure decreased.

Conclusions

Though subjects with borderline hypertension are frequently described as 'labile' there is no good evidence that they indeed exhibit wider spontaneous blood pressure oscillations than normal people.

If exposed to different pressure raising stimuli, patients with borderline hypertension do not show a stereotype 'hyperreactive' response.

PSYCHOSOMATIC CHARACTERISTICS OF PATIENTS WITH BORDERLINE HYPERTENSION

The popular concept that blood pressure 'lability' and 'nervousness' go hand in hand has been evaluated in a couple of studies. Ayman, in 1933, published the first report on psychosomatic traits of patients with borderline hypertension [57]. A set of specifically worded questions pertaining to the patient's personality and his behavioral traits were asked and compared to normotensive controls. Subjects with borderline hypertension described themselves as being more high strung, quick-

tempered, excited within themselves, unusually sensitive, unusually active and prone to shyness or blushing in public places.

Later, Thacker reported on college students with borderline hypertension [43]. He analyzed medical records for routine notes on the patients emotional state and divided the whole spectrum of psychological characteristics into 'emotionally stable', and 'nervous and excitable'. Subjects with systolic borderline hypertension had a 4-fold prevalence of 'nervousness and excitability'.

Next to conduct extensive psychological investigation in students with borderline systolic hypertension was Hamilton [58]. All the tests were numerically scored and statistically analyzed in a blind fashion. Groups with elevated systolic readings tended toward less physical and social activity. They were somewhat less dominant and self-assertive, but prone to anger. Blushing was the principal physical symptom.

Harris *et al* in 1953, started a continuous study of a group of 40 patients with borderline hypertension and 40 control undergraduate women [59]. Twenty patients with borderline hypertension and 22 controls were re-examined 4 yr later [60, 61]. Finally 11 yr later, Harris and Singer reported on the third exam in 11 patients with borderline hypertension and 13 controls [39]. Though in 1953, Harris *et al* promised "the actual incidence of hypertension for the 2 groups will be checked by later studies", subsequent reports deal only with the consistency of psychological characteristics. Blood pressure data are flagrantly missing. The first report utilized psychodrama, whereas in the other 2 reports the subjects were recalled for an interview. In all instances, the performance was rated by independent observers who had no knowledge of the subjects' blood pressures. In these papers, students with borderline hypertension initially demonstrated and later maintained a typical personality type. They exhibited less of a mature feminine pattern, showed less acceptance of self in a conventional role in society and seemed to derive less pleasure from their life experience. They acted with a certain motoric unrest and were quite prone to overt responses to inner and outer stimuli. Therefore, they appeared tense and pressured. At the same time they were trying to control themselves and hide their emotional feelings. The borderline hypertensives were also hostile and sensitive to perceiving hostility in other people.

Harburg *et al* [62] studied young males with systolic borderline hypertension and compared them to students with low readings. Subjects with higher systolic readings had significantly higher scores on 'submissiveness', 'sensitivity', 'sociability', and 'suspecting'. To further check on the alleged submissiveness of subjects with borderline hypertension, a special test of experimental yielding was devised. Subjects with systolic blood pressure elevation anticipated that they would not yield, but actually yielded to their counterparts. Later they reported yielding not only "for the compromise sake", but actually truly changing their opinions. Julius repeated a similar study with medical students in Yugoslavia [63]. The results were strikingly similar to previous findings by Harburg *et al* in Ann Arbor. The 'sociability' and 'submissiveness' were again associated with the systolic borderline hypertension.

Further evidence that blood pressure variability may have psychological correlates was presented by Harburg *et al* [64]. Subjects with a more variable blood pressure tend to have different early life experiences. They had a negative father image and remembered a stern and dominant father who was concerned with upward social mobility.

Until now, we have reported only papers supporting an association between certain psychological traits and borderline hypertension. There are no published works to the contrary. However, one group of investigators has devoted many yr to the investigation of 'precursors of hypertension' and still considers the findings in regard to psychosomatics inconclusive. Though their definition of precursors of hypertension is based on family history and only a few of the subjects have borderline blood pressure elevation, the results are relevant to the general question of early phases of hypertension. Bruce and Thomas [65] investigated students with hypertensive and non-hypertensive parents using the Rorschach Test. There was some suggestion that aggression, hostility, obsessive compulsive trends and feelings of inadequacy were more prominent among offspring of hypertensives and coronary patients. A later analysis of Rorschach Test responses with particular emphasis on aggressivity and hostility responses failed to show striking difference [66]. Again in [67], psychological variables failed to contribute to differentiation based on parental history of hypertension. Anxiety scores developed from a special questionnaire discriminated between three groups of students with different parental history of coronary disease, but were not helpful for parental hypertension. In 1967, Dr. Thomas concluded: "We have not yet been able to demonstrate a fundamental difference for any psychological factor in the offspring of hypertensive parents compared to offspring of negative parents".

Conclusion

The possible association of borderline hypertension with certain psychosomatic characteristics in young subjects was repeatedly investigated. When using similar instruments and within the framework of individual studies, authors are able to show consistent and reproducible personality patterns. Since they use different methodology and nomenclature, a comparison of psychological findings across studies is difficult. In the broadest terms, there is an agreement that subjects with borderline blood pressure elevation have difficulties in self-assertiveness, may be submissive, are tense, unusually sensitive and prone to uncontrolled impulsiveness. Sometimes they are described as showing signs of motoric unrest. None of the studies has succeeded in positively relating the described psychological traits to the subsequent development of hypertension.

HEMODYNAMICS IN BORDERLINE HYPERTENSION

Uncomplicated established essential hypertension is characterized by increased peripheral resistance and normal cardiac output [68]. A different hemodynamic picture is emerging in the borderline hypertension. Wezler and Böger in 1939, already recognized that in occasonal young patients with hypertension the elevation of blood pressure is maintained by an increased cardiac output with "normal, at least not elevated values of their elastic and peripheral resistance" [69]. Almost 20 yr later, Widimsky and his co-workers [70] reported such elevation of cardiac output in a substantial group of juvenile hypertensives, a majority of whom did not have established hypertension. Since then, hemodynamic data on over 400 subjects with borderline hypertension are available. For better comparison, we summarize the results in Table 6. Cardiac output and heart rate in this table are considered elevated if they were more than 2 standard deviations above the mean of control subjects. In all series, a substantial proportion of the subjects had elevated cardiac

TABLE 6. HEMODYNAMICS OF BORDERLINE HYPERTENSION*

Author	No. of cases	Age range	Cardiac output	Heart rate	Remarks
[71] Bello *et al* 1965	11	18–60	72	0	Cardiogreen recumbent
[72] Bello *et al* 1967	10	15–21	2	0	Cardiogreen. Mean output of borderlines significantly above normal recumbent
[73] Eich *et al* 1962	52 21	50 50	40 24	? ?	RISA recumbent Mean pulse +12 in young labiles—no difference in older ones
[74] Finkielman *et al*	17	25–69	76	0	Evans blue
[51] Johansen	19 17 25	17–29 30–39 40–49	31.5 17 4	20 38 24	Sitting Cardiogreen
[75] Konig *et al*	5	?	40	20	Direct Fick recumbent
[52] Julius *et al*	44 21	35 35–55	6.8* 9.5	40	Cardiogreen— unpublished data added recumbent *Mean of borderline significantly elevated
[54] Levy *et al*	20	19–41	15	?	Cardiogreen
[53] Sannerstedt	14	17–58	35	50	Bromsulphalein sitting
[70] Widimsky *et al*	25	16–30	70	45	Evans blue recumbent

*Per cent of cases 2 std. dev. above control.
For further data on hemodynamics in borderline hypertension which could not be presented in this tabular form see references: 81–84.

output. The difference of the means of the cardiac outputs between the 2 groups is significant in the majority of studies. Some discordant notes are struck by Johansen [51] who finds 'almost' significant elevation in the younger age group, but no difference in older subjects, and by Levy and coworkers [54] who do not find a difference at all. Johansen's finding that differences in cardiac output between normals and patients with borderline hypertension decreases with age is supported by Julius and co-workers [52], but we could still demonstrate significant elevation in older subjects.

In Levy's study, the mean cardiac index for patients with borderline hypertension is 200 ml higher than for the controls, but did not reach significance because one control subject had a very high cardiac output. Three subjects with borderline hypertension out of 20 in Levy's study had cardiac outputs more than 2 standard deviations above the control mean. Their data actually are not substantially different from other reports.

On the whole, the evidence points to an increase of cardiac output in patients with borderline hypertension. Not all subjects have a high flow at rest. Between 1/3 and 1/2 of the group loads around a higher mean which is usually at least 1 standard deviation above the mean of the control subjects, but there is also a wide overlap. The distribution of cardiac outputs in patients with borderline hypertension is continuous and one can not distinguish separate 'normal' and 'high' subgroups. The elevation is recognizable in the recumbent [52], tilted [55], and sitting positions [51]. However, during exercise, particularly with high levels of work, the difference between normals and patients with borderline hypertension decreases or disappears [51–53].

Elevation of cardiac output is described in overt anxiety [77]. Presence or absence of anxiety, could substantially influence the reproducibility of the measurements of the cardiac output. However, the elevation of the cardiac output in patients with borderline hypertension is fairly reproducible. Eich and co-workers [78] repeated measurements in 10 min. Only 1 out of 8 subjects with previously high cardiac output had normal readings the second time. On 2 subsequent days, 1 of the 4 patients failed to maintain his elevated output. Long term month-to-month reproducibility of the elevation of cardiac output in an individual depends on whether he is hypertensive at the time of the study. However, as a whole, the group with previously high cardiac outputs continued after 50 months to maintain a significant elevation [78]. We have recently shown that after 50 min of rest patients with borderline hypertension continue to have a significant elevation of cardiac output [55].

Whether the elevation of the cardiac output in borderline hypertension is maintained by increased heart rate or stroke volume is somewhat controversial. Finkielman et al and Bello and co-workers do not find increased heart rates and consequently the stroke volume is elevated [74, 71]. Eich et al [73] have a significant increase of the heart rate, but the cardiac output is even more elevated so that the stroke volume increases. Widimsky et al [76] find a combined elevation of heart rate and the stroke volume in 58.33 per cent of all cases, an elevated stroke volume with normal heart rate in 37.5 per cent, and in only 4.17 per cent was the stroke volume normal. On the other hand, Sannerstedt, Johansen, Julius, et al [51–53] report normal stroke volumes and increased heart rates. Frohlich et al [56] in their group of mild hypertensives, also find a faster heart rate, but the elevation of the cardiac output was minimal. Those finding higher heart rates are able to demonstrate them also during exercise [51–53] after 10 min of 45° of tilt [55], and even after blockade with propranolol [55]. The reason for some discrepancies in regard to the heart rate could conceivably be found in the age of studied subjects, since the increase in heart rate among patients with borderline hypertension decreased with age [52].

The underlying mechanism for the increase in cardiac output remains obscure. An apparent normal regulation of the cardiac output in its relationship to the oxygen consumption is maintained both at rest and during the exercise [51–53]. Increase of the cardiac output after exercise [51–53] or after infusion of dextran [79] among patients with borderline hypertension is of the same magnitude as in normal subjects.

Conversely, the decrease of the cardiac output to tilt in borderline hypertension is also not abnormal and is comparable to the decrease in control subjects [55]. Therefore, the cardiac output in borderline hypertension appears regulated in a normal fashion but maintained at a higher level. Whether this is achieved by an increased sympathetic drive of the heart, increased venous return through a higher tone in the capacitance system, or whether the defect lies in the intrinsic propensity for forcible cardiac contraction has hardly been explored. Johansen [51] has not been able to establish any systemic difference between control subjects and patients with borderline hypertension in the rise of the central venous pressure after plasma volume expansion; an indication of a normal venous tone. Finkielman et al [74] also believed that patients with borderline hypertension have a normal capacitance tone, since their pletismographic flow cessation pressure is normal. These results are at variance with the results of Ulrych et al [80] who found in borderline hypertension a shift of the blood volume to the thorax. This shift presumably is a consequence of the smaller capacity of the peripheral vessels.

Whether the increased cardiac output is mediated by an overactive beta adrenergic system was investigated by Sannerstedt et al [55]. After blockade with propranolol, the difference in cardiac output between normals and subjects with borderline hypertension decreases, and the remaining elevation in patients with borderline hypertension is not statistically significant. Consequently it would appear that the sympathetic beta adrenergic nervous system has an important role in maintaining elevated flow in borderline hypertension,

Levy et al [54] investigated the possibility of an increased intrinsic myocardial contractility in borderline hypertension. Three out of their 20 patients had indications of more forcible myocardial contraction. Unfortunately, it is not shown whether these subjects had elevated cardiac outputs at rest.

Measurements of the volume of the blood in borderline hypertension are scarce, Finkielman et al [74] found normal values, Bello et al [72] slightly lower readings whereas, we [79] were able to find considerably lower values in borderline hypertension.

The distribution of the increased flow in subjects with borderline blood pressure is essentially unexplored. Bello and co-workers find elevated renal blood flow in their subjects [71].

In regard to total peripheral resistance, the majority of investigators do not find significant differences between controls and patients with borderline hypertension [51–54,70]. Eich et al [73] did choose patients with extremely high cardiac outputs and compared them to normotensive controls. Under such circumstances, the total peripheral resistance in patients with borderline hypertension is significantly lower. However, the real question is what would have been the peripheral resistance of normal individuals at those high levels of cardiac output. This question was raised by Fejfar [22] and recently by Julius et al [79]. When the cardiac output is taken into consideration and comparison made at similar levels of cardiac output, the patients with borderline hypertension always show elevated peripheral resistance. The normal adjustment to increased flow is a corresponding decrease in the resistance resulting in normal blood pressure. In patients with borderline hypertension the peripheral resistance does not adjust to the increased flow, and consequently the pressure rises.

The appearance of a typical high-output hemodynamic pattern in borderline hypertension raises the question whether this is a specific form of hypertension or an

earlier phase in the development of the established hypertension. Eich *et al* [73] believed that patients with increased cardiac output may have a better, more benign course. However, their next paper [78] gave a somewhat different assessment. One third of patients who originally had an elevated cardiac output 50 months later maintained elevated blood pressure, but their cardiac output decreased. Whether this really means a change from high output to high resistance hypertension is not yet clear and depends on whether the 'new' pattern of higher resistance is a permanent state or not. It is important to remember that for some subjects in Eich's series the reverse was also true; those with previously normal cardiac output and higher resistance, later exhibited a higher cardiac output.

Conclusions

A substantial proportion of subjects with borderline hypertension has an increased resting cardiac output in the recumbent, sitting, and tilted positions. This is a fairly reproducible finding. Changes of cardiac output induced by exercise, plasma volume expansion or tilting are similar in degree and direction in patients with borderline hypertension to those observed in normal subjects. Also the cardiac output in borderline hypertension remains regulated in a normal fashion to the oxygen consumption. There is some controversy whether the output is increased by elevated heart rate, or a high stroke volume. The mechanism for the elevation of the cardiac output and the relative role of the venous return, autonomic nervous control and the intrinsic myocardial contractility are not fully explained.

The peripheral resistance in patients with borderline hypertension with elevated cardiac output is either low or normal. However, if the comparison is made at equal levels of cardiac output, subjects with borderline hypertension have high peripheral resistance.

DISCUSSION

Terminology

Many terms have been proposed to describe borderline hypertension. Generally, the definition includes some discrimination between the severity ('borderline', 'mild', 'benign') and the stability of the blood pressure ('labile', 'occasional' ,'transient'). Almost uniformly, the term 'hypertension' is utilized. Confusing nomenclature and various definitions seriously interfere with meaningful comparison of results of different investigators. Therefore, some basis for a unified nomenclature and methodology will be proposed.

1. 'Prehypertension' is not an applicable term. The incidence of subsequent essential hypertension is more frequent in this group of subjects, but not at a level justifying the term. As in 'precancerous' 'perhypertension' should indicate a very high risk for subsequent occurrence of the fully developed disease. In a strict sense as in 'prediabetes', 'prehypertension' would imply that this condition is a phase in the development of essential hypertension. Whether this is the case remains to be proven.

2. The distinction between 'labile' and 'stable' blood pressure is artificial and not meaningful. It usually involves the relationship of the blood pressure to an arbitrary cutting line and does not offer a measure of the blood pressure varia-

bility. Furthermore, the classification of a subject very much depends on the number of measurements and the manner in which they are taken.

3. Though the term hypertension has a serious connotation, it has traditionally been applied to the condition of borderline blood pressure elevation.

For all these reasons, we endorse the World Health Organization nomenclature and propose that the condition be uniformly called *borderline hypertension.*

In addition to the desirability of a unified nomenclature, there is a need for some agreement in regard to the ways of collecting data. We would like to propose the following principles:

1. It is clearly important to differentiate between studies based on only one blood pressure and those utilizing more readings.

2. One reading will most likely be preferred in epidemiological studies. This could be an entirely casual reading.

3. Another standardized method of measurement is to take repeated casual readings on different days and call this *usual* blood pressure. Average of 3 readings should be the minimum requirement. One reading should be below and 2 above the upper limits of normotension. The av. of 3 blood pressures should fall in the borderline range.

Under these conditions 2 categories would emerge:

1. Casual borderline hypertension.
2. Usual borderline hypertension.

It is relatively easy to set limits for these 2 categories, since there is a multitude of data on casual blood pressure distribution in populations. A rational approach would be to take as the upper limits of normal blood pressure levels delineating the upper 10 per cent of the total distribution and then remove the clearly hypertensive cases. Table 7 shows some relevant data from different studies.

An alternate approach is to define the borderline as cases falling within the range of upper 85–95 per cent of the actually observed blood pressure distribution. This method is applicable only in big populations; it will be necessary for clinical studies to rely on preset ranges of blood pressure.

Borderline hypertension has traditionally been diagnosed from casual readings. One would be entirely at a loss to define what the range of borderline resting blood pressure should be. If resting readings are preferred, the investigators may want to use the upper 90–95 per cent of the observed distribution in their population and define this as borderline hypertension.

Earlier we expressed the opinion that division into 'labile', and 'stable', is artificial and useless under ordinary circumstances. If, however, the blood pressure variabilty *per se* is to be observed and analysed, specific methods should be developed. We are proposing the principles for such a methodology.

1. There must be a substantial number of random blood pressure readings under varying circumstances. The use of a portable blood pressure recorder over a period of hr at different days is recommended.

2. Lability should be defined as the standard error of deviation about an individual's mean (in Mm Hg).

3. It is advisable that the *range* of the observed variability be expressed as the *percentage* of the average of all blood pressure readings of an individual.

TABLE 7. REVIEW OF DATA ON LIMITS OF NORMAL BLOOD PRESSURES

Author	15–19 U.L norm	15–19 U.L borderline	25–29 U.L norm	25–29 U.L borderline	35–39 U.L norm	35–39 U.L borderline	45–49 U.L norm	45–49 U.L borderline	55–59 U.L norm	55–59 U.L borderline	65–69 U.L norm	65–69 U.L borderline	Remarks
[91] E.P. Fedorova	129/84	139/90	129/84	139/90	129/89	139/94	139/89	145/94	139/89	145/94	149/89	159/94	Analysis of blood pressure distribution in Moscow. Criteria for upper limits of normal—not given
Federal Aviation standard			140/88		145/92		155/96		160/98				Basis for the criteria—not given
Stanley in handbook of Circulation FASEB, 1959 Males			140/90	150/95	145/92	160/100	155/95	170/104	165/98	180/108	170/100	190/110	Construed from a graph
Females			138/85	140/92	140/90	150/98	155/95	175/105	170/100	185/108			
[4] Boe, et al Males	137/78	153/91	144/85	162/94	146/87	164/98	152/90	173/103	164/94	198/110	178/96	220/115	Upper limits of normal defined by upper limits of the 3rd quintile. Upper limits of borderline = 95% of total distribution
Females	131/77	144/89	135/81	150/92	141/85	162/95	156/91	183/103	175/95	207/112	194/99	232/119	

* U.L = Upper Limit

   4. The term 'labile' blood pressure should be reserved for the upper quintile of the observed distribution of the lability as defined in 2 and 3.

Implication for research

   The whole literature in the field of borderline hypertension leaves much to be desired. In this part of the discussion, we will outline the most evident deficiencies and try to catalogue areas which require answers through research.

   Though there is a body of evidence indicative of a poorer prognosis for patients with borderline hypertension, the whole question of the natural history of the disease is still unclear. Almost all studies are retrospective and therefore, suffer from a poor description of the subjects at the onset of the study. Good prospective studies should answer many unclear questions: What is the relative importance of one casual elevated reading? Is the av. level of blood pressure, the amount or the range of blood pressure variability more predictive of future morbidity? Is there some way to recognize the future hypertensive amongst patients with borderline hypertension? What is the importance of family history, age of onset of borderline hypertension, overweight and later history of weight gain in this group of patients? What is the relative importance of the blood pressure when divorced from other risk factors?

   Another extremely important area in the natural history of borderline hypertension is the relationship of the hemodynamic abnormalities to the later development of established hypertension. Is it really true that the elevated cardiac output leads into increased peripheral resistance? Under which circumstances does the transition occur? There is some evidence that increased heart rate predicts to later hypertension and cardiovascular disease [25, 86]. Some patients with borderline hypertension have increased heart rates [51–53, 73, 76]. Is this important for the development of established hypertension and is the effect of the heart rate independent from the cardiac output?

   The role of modifying factors in the natural history of borderline hypertension has practically not been explored. It has been suggested that life experience and socio-psychological factors play an important role, but this has never been tested by serial measurements in prospective studies. Far more important is the scarcity of reliable information on whether treatment of borderline hypertension offers protection from the development of cardiovascular hypertensive disease. It is generally accepted that treatment of the established hypertension reduces the so-called pressure related complications but does not change the rate of the development of atherosclerosis [86]. Could early treatment in the borderline phase reduce the later vascular disease? Would positive effects outweigh the side effects of the drug treatment? Finally, can one effectively control the blood pressure in borderline hypertension? To our knowledge there is only 1 paper showing positive effects of treatment in very mild hypertension [87] but the patients in that study did not have borderline hypertension as defined in this review.

   In the area of hemodynamics, the basic animal experiment whether increased systemic flow really leads to a rise in the peripheral resistance has not been attempted. In clinical hemodynamic studies, one would expect the investigations to move from the phase of cataloguing and documenting abnormalities to explorations of the mechanisms by which these abnormalities are perpetuated. Are elevated cardiac output and heart rate maintained through increased sympathetic outflow, by a lack

of vagal inhibition, by a circulating hormone or by some intrinsic increase in the myocardial contractility? Does increased venous return through a rigid and small capacitance system play a role?

In conclusion, while borderline hypertension appears to carry increased risk for the individual and a better understanding of this condition may shed some important light on the development of the established hypertension, information is lacking in many basic areas. It is hoped that in the future more emphasis will be given to this potentially rewarding investigative field.

Practical implications

Whereas one can argue on both sides of the Pickering–Platt argument whether hypertension as a separate disease [88] or extension of normal blood pressure distribution [89] the practising physician has clear guidelines—moderate to severe hypertension should be medically treated and normotension does not require professional attention. But with borderline hypertension, the management of a patient becomes a problem. The prevalence of borderline hypertension is substantial and a practising physician can expect to face this therapeutic dilemma in at least 10 per cent of all his patients.

Unfortunately, there is not enough reliable information to set firm guidelines for the management of a patient with borderline hypertension. Some principles, however, could be proposed: (i) a person with borderline hypertension needs medical attention in view of the increased risk for the development of cardiovascular disease and its complications. This medical attention always implies a regular follow-up with the same physician or institution, but the specific type of care may vary from drug treatment to only regular physical exams, depending on the individual case. (ii) Since the blood pressure in this condition does fluctuate, it is of paramount importance to establish a reliable baseline by repeated casual and resting measurements. Whenever possible, a series of readings over weeks in the patients' home (by self-determination or by the members of the family) should be utilized. These readings are then used to evaluate the effects of the treatment or make a later decision for treatment if the blood pressure starts to rise. (iii) The amount of the initial search for secondary causes of hypertension must be left to the physician's discretion, but a complete assessment of the hypertensive target organ damage should be compulsory (eyegrounds, left ventricular hypertrophy, evidence of vascular damage, renal status). A very thorough history of familial tendencies toward hypertension and atherosclerosis should be taken. (iv) The decision whether or not a patient should be treated must be individualized. This review indicates that the risk for the development of sustained hypertension in a patient with borderline hypertension is double than in normotensives. However, one should remember that the prevalence of hypertension in the general population is about 5 per cent so that in patients with borderline hypertension it would be around 10 per cent. If one takes for granted, and that has by no means been proven, that treatment in the borderline phase prevents the development of established hypertension, treating all patients with borderline hypertension means that 90 per cent who would stay normotensive are unnecessarily exposed to side effects and expense of the treatment. The rational approach to the treatment in borderline hypertension must be based on some main considerations;

(a) In view of the family history [11], body wt. [92], blood sugar [93], cholesterol

[93], resting heart rate [85], and the av. level of the blood pressure is the individual patient at a higher risk for vascular disease?

(b) Could some factors be modified before treatment with drugs is recommended— in particular sodium intake, overweight, and a stressful environment?

(c) Is the patient symptomatic?

(d) Is there any evidence of target organ damage?

(e) Finally, did the applied treatment produce any perceptible change in the blood pressure levels or in patients' subjective symptoms?

If in the future a well-organized prospective study provides the answers, the task of the practising physician will be simpler. Until then, we believe the evidence warrants a cautiously active approach to patients with borderline hypertension.

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ADDENDUM

The following papers in the field of borderline hypertension came to our attention after this review has been completed.

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