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EDITORIAL

BLOOD PRESSURE READINGS

What prognostic significance can be attached to raised blood pressure readings? And why is a raised blood pressure serious in one patient and not in another? These questions cannot be answered in the present state of our knowledge, yet they are of such everyday importance that every medical practitioner must hold some views upon them.

Measurement of the blood pressure is the pivotal point of the medical examination—no less to the average practitioner than to the examiner of service recruits, immigrants or university students. In this age of popular medicine, the examinee expects it; the doctor—be he an uncertain young fledgling or a tired specialist physician—often finds refuge in it when he does not know what else to do, for it is a concrete test that impresses the patient and therefore serves to gain his confidence. The value and importance of the test is magnified—perhaps *ad absurdum*—by the insurance companies, who employ doctors virtually to measure the blood pressure of their applicants and thereafter to check that there is nothing else wrong with them.

Anyone who has examined healthy young individuals will know how often the readings are above the traditionally normal levels of 140 mm. Hg systolic pressure and 90 diastolic. In a recent investigation carried out by a London cardiologist, Dr. William Evans,[1] 182 out of 400 army recruits (45%) were found to have systolic readings above 160, and 258 (64%) diastolic readings of 90 or over. The hearts of all these men were investigated by careful clinical examination, screening and electrocardiography, and pronounced normal, and they were accepted into the British army. About 10 years later 50 were re-examined, and in only 8 (16%) did the readings 'exceed slightly' the initial pressures. Both sets of examinations were conducted under as nearly ideal conditions as could be achieved: a quiet room, an unrushed consultation, the lowest of 3 readings being accepted as the basal value. There is nothing new in this type of work. Most medical officers engaged in it will bear out Dr. Evans' experience. Yet it would be a rash doctor who diagnosed essential hypertension in an able-bodied young man of 21 at a single examination. As Allbright emphasized more than 50 years ago, when he showed that hypertension could occur in the absence of renal disease, essential hypertension can be compatible with long life and freedom from serious disease.[2] On the other hand, no

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BLOEDDRUK-LESINGS

Van watter belang is verhoogde bloeddruk-lesings by die prognose? En hoekom is 'n verhoogde bloeddruk ernstig by die een pasiënt en onbelangrik by die ander? Met ons hedendaagse kennis kan ons nog nie hierdie vrae beantwoord nie, maar die saak is van alledaagse belang, sodat elke geneesheer wel die een of die ander mening daarop nahou.

Die meet van die bloeddruk is die spil waarom 'n mediese ondersoek draai—ewe belangrik vir die gemiddelde praktisyn as vir die ondersoeker van diensrekrute, immigrante of universiteitstudente. Die pasiënt verwag dit in hierdie eeu van leke-medisyne; die geneesheer, of hy nou 'n onsekere jong doktertjie of 'n moeë spesialis-internis is, neem dikwels sy toevlug daartoe as hy nie weet wat anders om te doen nie, want dit is 'n tasbare toets wat 'n indruk maak op die pasiënt en dus sy vertroue wen. Die waarde en belangrikheid van hierdie toets is baie vergroot—miskien *ad absurdum*—deur versekeringsmaatskappye, wat feitlik dokters huur om die bloeddruk van hul applikante te meet en om daarná na te gaan of hul iets anders ook makeer.

Enige dokter wat reeds gesonde jong mense ondersoek het weet dat die lesings dikwels bó die normale hoogtes van 140 mm. Hg sistoliese druk en 90 diastolies is. In 'n onlangse ondersoek deur 'n Londense kardioloog, dr. William Evans,[1] was dit bevind dat 182 uit 400 (45 persent) rekrute vir die leër sistoliese lesings bo 160 gehad het, en 258 (64 persent) diastoliese lesings van 90 of meer. Al hierdie mans het noukeurige kliniese hartondersoeke ondergaan, met fluoroskopie en elektrokardiografie, en dit is bevind dat hulle normaal was. Hulle is in die Britse leër opgeneem. Omtrent 10 jaar later is 50 van hulle weer ondersoek, en by slegs 8 (16 persent) het die lesings die oorspronklike syfers ‚effens oorskry'. Beide ondersoeke is onder feitlik ideale toestande uitgevoer: 'n rustige kamer en 'n ongejaagde mediese ondersoek; en die laagste uit 3 lesings is aanvaar as die basale waarde. Daar is niks nuuts in hierdie soort werk nie. Die meeste mediese beamptes wat daarby betrokke is, sal dr. Evans se ondervinding kan bevestig. Maar die dokter wat tydens 'n enkele ondersoek essensiële hoë bloeddruk by 'n gesonde jong man van 21 jaar oud diagnoseer, is heeltemal te haastig. Soos Allbright meer as 50 jaar gelede benadruk het, toe hy bewys het dat hoë bloeddruk *sonder* niersiekte kan voorkom, kan essensiële hoë bloeddruk gepaard gaan met 'n lang lewe sonder ernstige siekte.[2] Aan die ander kant hoef geen dokter daaraan herinner te word nie dat egte slagaarontaarding, met sy onvermydelike nasleep van ernstige komplikasies, een van

doctor need be reminded that true arterial degeneration, with its inevitable train of grave complications, ranks high in the list of causes of human mortality and morbidity. Organic hypertension is due to arterial degeneration—the relatively malignant type with arteriolar necrosis, the relatively benign type with atheromatous changes—and the problem is this: How does one know whether a patient with a clinically demonstrable high blood pressure is going to develop irreversible arterial changes that will shorten his life, or whether he is of the type of human being that normally shows a rasied blood pressure in the consulting room and will live to a ripe age?

Evans seeks to separate the condition of 'labile and intermittent hypertension which may manifest itself under the stress of medical examination' (which he calls 'hypotonia') from the text-book disease of hypertension. While most people will commend his notion of 'hypotonia', Evans will find many critics of his assertion that it can actually be distinguished in advance from true hypertension. As his own series showed, cardiac pain was not confined to the true hypertensives (nearly 50% of his healthy group complained of it 10 years later) and some of his cardiac 'hypotonics' died of coronary infarction. Moreover, American work has shown that sustained hypertension is commoner in persons with previous transient hypertension than in those whose blood pressure has never been raised.[3] One wonders whether Evans' follow-up period of 10 years is sufficient; perhaps the passage of a further decade will alter the picture entirely and render the fate of his 'hypotonic' group nearer to that of his hypertensives.

It is clear that there is as yet no solution to this problem. Experience and common-sense remain, in the absence of other signs and symptoms, the best guides to when a raised blood-pressure reading should be regarded as significant. The best place for your sphygmomanometer when examining healthy young adults—according to one prominent physician—is the back seat of your motor car, but sooner or later a coarctation of the aorta, a hydronephrosis, or some other remediable condition will be missed. And when dealing with patients the blood pressure must undoubtedly be taken—for 3 reasons, viz. the patient expects it, the examination may help to establish a confidence and, finally, it may be useful in the diagnosis. It is particularly useful in reassurance, i.e., as a means of enabling one to tell the patient that his pressure is 'normal'; with the present-day lay ideas of the meaning of 'low blood pressure' and 'high blood pressure' it is probably unwise ever to tell him anything else. Certainly lesser mortals than Dr. Evans will have difficulty in persuading any selection board, let alone an insurance company, to accept a young man with a blood pressure of more than 140/90 as a perfectly fit individual.

1. Evans, W. (1957): Lancet, **2**, 53.
2. Editorial (1957): *Ibid.*, **2**, 81.
3. Levy, R., Hillman, C. L., Stroud, W. D. and White, P. D. (1944): J. Amer. Med. Assoc., **126**, 829.

die mees voorkomende oorsake van menslike sterftes en siekte is. Organiese hoë bloeddruk word veroorsaak deur slagaarontaarding—die betreklik kwaadaardige soort met endslagaar-ontaarding, en die betreklik goedaardige soort met slagaarvervetting—en die probleem is dit: Hoe weet 'n mens of 'n pasiënt met 'n klinies bewysbare hoë bloeddruk later onherstelbare slagaarveranderings gaan ontwikkel wat sy lewensduur sal verkort, en of hy van die soort is wat gewoonlik 'n verhoogde bloeddruk in die spreekkamer toon en tog 'n lang lewe sal geniet?

Evans trag om die kondisie van ‚onvaste en onderbroke hipertensie wat onder die stremming van 'n mediese ondersoek na vore kom' (hy noem dit ‚hipotonie') te onderskei van die hoë bloeddruk wat in die handboeke beskryf word. Die meeste mense sal wel Evans se teorie van ‚hipotonie' goedkeur, maar daar sal baie wees wat krities sal staan teenoor sy bewering dat dit werklik vooraf kan onderskei word van die egte hoë bloeddruk. Sy eie reeks toetse het bewys dat pyn in die hart nie beperk was tot lyers aan egter hipertensie nie (byna 50 persent van sy gesonde groep het 10 jaar later van hartpyn gekla) en sommige van sy ‚hipotoniese harte' het aan hart-infarksie gesterf. Ook is dit in Amerika bewys dat volhoue hipertensie meer dikwels voorkom by pasiënte wat tevore verbygaande hoë bloeddruk getoon het as by dié wat nooit hoë bloeddruk gehad het nie.[3] 'n Mens wonder of Evans se opvolgperiode van 10 jaar lank genoeg was; miskien sou die beeld ná nog 'n dekade heeltemal anders lyk, en sal die lot van sy ‚hipotoniese' pasiënte meer op dié van sy lyers aan hoë bloeddruk lyk.

Dit is duidelik dat hierdie probleem nog nie opgelos is nie. Wanneer ander tekens en simptome uitbly, moet ons maar op ons ondervinding en gesonde verstand staat maak om te bepaal wanneer 'n verhoogde bloeddruklesing as veelbeduidend beskou moet word. Die beste plek vir die bloeddrukmeter is agter in jou motor, maar vroeër of later sal 'n saamgeperste aorta, 'n geval van hidronefrose, of die een of ander geneesbare siekte oor die hoof gesien word. En wanneer die dokter met pasiënte te doen het, moet die bloeddruk altyd gemeet word—om 3 redes: die pasiënt verwag dit, die ondersoek dra moontlik by om sy vertroue in die dokter op te wek, en laastens kan dit behulpsaam wees by die diagnose. Dit is veral nuttig aangesien dit die dokter in staat stel om die pasiënt gerus te stel dat sy bloeddruk ‚normaal' is. Maar met die hedendaagse leke-opvatting van ‚lae en hoë bloeddruk', is dit miskien raadsaam om maar altyd aan die pasiënt te sê dat sy bloeddruk normaal is. Dit staan vas dat minder belangrike mense as dr. Evans dit moeilik sal vind om enige keurraad—om nie eers van versekeringsmaatskappye te praat nie—oor te haal dat 'n jong man met 'n bloeddruk van meer as 140/90 heeltemal gesond is.

1. Evans, W. (1957): Lancet, **2**, 53.
2. Van die Redaksie (1957): *Ibid.*, **2**, 81.
3. Levy, R., Hillman, C. L., Stroud, W. D. en White, P. D. (1944): J. Amer. Med. Assoc., **126**, 829.

HEPATIC FAILURE

An important function of the liver is the detoxication of products of intestinal protein digestion that reach it through the portal vein. The break-down of this mechanism is believed to be the cause of the characteristic syndrome of hepatic coma.[1] Entry of undesirable protein metabolites into the systemic circulation may take place in two ways; firstly as a result of massive hepatocellular destruction (as, for example, in fulminating viral hepatitis) these substances are

allowed to traverse the liver unmodified by normal metabolism, and secondly through the presence of an extensive collateral circulation which enables them to by-pass the liver.

The exact nature of the harmful products remains to be settled. Ammonia has been blamed, but blood-levels correlate poorly with the clinical state.[1] However, neurological changes are always present when the level exceeds twice the normal upper limit of 1 μg./ml.[4] The technique of estimation is too cumbersome for routine use.

Prominent early manifestations of this 'portal-systemic encephalopathy' are personality changes and constructional apraxia. The patient often has difficulty in reproducing simple designs in matches, and the handwriting deteriorates and becomes irregular (a daily handwriting chart is useful in following slight fluctuations in the status of the convalescent patient). Non-specific electro-encephalographic changes are seen. When present, associated signs, e.g., flapping tremor, foetor hepaticus, spider naevi and palmar erythema, not to mention jaundice, help in diagnosing coma or the earlier neurological changes as being due to liver failure.

In cirrhosis the patient is often in a precarious state of balance, easily upset by an increase in the nitrogenous content of the blood. This is usually due to a large protein load in the bowel, e.g., a high-protein meal or a gastro-intestinal bleed. Methionine is known to precipitate coma in the susceptible, and ammonium chloride, given as a diuretic, is dangerous in these people. Acetazolamide (Diamox) may also induce coma, but does so by inhibiting tissue uptake of ammonia. The dangers of operations and acute infections in cirrhotics are also well recognized.

In the emergency treatment protein should be banished from the diet, and enemas and saline purges used to flush nitrogenous material from the gut. An attempt should be made to inhibit bacterial digestion of protein by bowel sterilization with oral chlortetracycline or neomycin.[2] If gastro-oesophageal varices continue to bleed, tamponade with a Sengstaken balloon should be attempted.

At least 1,600 calories should be provided daily, as glucose; if the oral route is impractical, a 20% solution can be infused into the superior vena cava through a polythene catheter. When the patient recovers protein is slowly reintroduced into the diet, but a cirrhotic who has had a bout of coma should not take more than 50 g. per day. The well-compensated cirrhotic with ascites who has not had coma falls into a different category; a high-protein diet plays an important part of the management in this type of case.

It has been stated that various 'specific' substances alter the course of hepatic coma. Glutamic acid combines with ammonia to form glutamine; arginine,[3] ornithine and thioctic acid are supposed to stimulate the tricarboxylic acid cycle. Their advantages are mainly theoretical and there is not enough evidence to recommend any of them for routine use. Sherlock[4] considers that there is no place for steroids in the treatment of hepatic coma due to cirrhosis, where the principles outlined above usually achieve their purpose; fulminating hepatitis has such a high mortality that they should be tried, in addition to the standard regime, if only for want of something better to do.

1. Annotation (1957) : Lancet, **1,** 623.
2. Annotation (1957) : *Ibid.*, **2,** 280.
3. Najarian, J. S. and Harper, H. A. (1956): Amer. J. Med., **21,** 832.
4. Sherlock, S. (1957) : Amer. J. Digest. Dis., 2, 353