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CONTRAINDICATIONS TO THERAPY IN*
HYPERTENSION AND ANEMIA

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In an era of ever-increasing medicaments for the ills of mankind, any appeal for consideration of less treatment must indeed sound anachronistic. Nonetheless, an occasional reaffirmation of the ageless hippocratic warning "primum non nocere" is felt to be justified in this hectic period of therapeutic innovations. In our enthusiasm for the new, a calm appraisal of what we are treating and what we can expect to accomplish by our treatment must continue to be the basis of all therapeutic thinking. All of us are intrigued with the unusual entities; the rare disorder is the challenge and the spice of medicine. However, the proper management of such ordinary conditions as hypertension and anemia may just as truly distinguish the capable physician as his handling of the obscure, involved diagnostic problem.

There is little doubt that man has always been endowed with a blood pressure. Whether the relatively recent ability to measure this particular physical fact has been a universal blessing may be seriously questioned. The morbidity engendered by the sphygmomanometer undoubtedly ranks high with that of other alleged boons of the machine age. The latter was supposed to free man — but this particular product, the blood pressure cuff, has probably harnessed more people to more grief and expense than we will ever know. As with all potentially useful tools, the failure has not been in the instrument but in the user, the physician himself.

The distinction between high blood pressure or arterial hypertension and hypertensive vascular disease is a vital and most important one. The former indicates an apparent abnormality requiring consideration and guarded follow-up; the latter represents an outright disease state necessitating active treatment whatever its etiology. High blood pressure is common and represents a threat, primarily because of the patient's awareness of having it. Hypertensive vascular disease is far less common and represents a true organic threat to the individual inasmuch as it portends a shortened life span. The responsibility for the confusion of the two conditions must be laid at the feet of the medical profession. Needless to say, the resolution of the confusion is our duty.

Thus, the question of whether we are dealing with simple high blood pressure or true hypertensive disease has much more than academic significance. Aside from the psychological burden to the patient which the diagnosis of hypertensive disease entails, the program of treatment with the agents currently available commits him to an indefinite period of considerable expense and no little hazard. The undesirable side effects of even the simplest antihypertensive medication — the Rauwolffia compounds — let alone the treacherous vagaries of the more potent Apresoline and ganglion blocking agents — should spur caution in our approach to treatment of what may be simple arterial hypertension.

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What factors then should guide us in deciding this question of treatment, or to put the question another way, how do we decide whether the patient has high blood pressure or hypertensive vascular disease?

Studies have indicated that blood pressure is influenced by age, sex and weight. Authorities differ about the limits of systolic pressure but most are in agreement that a diastolic pressure of 110 is abnormally high.

Master\(^1\) has made an important contribution in his study of the effects of sex and age. He has concluded that for a man 20 years old, upper normal blood pressure limits are 140/88 mm Hg. Hypertension at 20 years of age exists with a systolic over 150 mm Hg and diastolic over 95 mm Hg. The significance of the range between 140-150 mm Hg systolic and between 88-95 mm Hg diastolic can only be determined with careful follow-up observations. At the age of 40, hypertension is said to be present with a systolic of 158 mm Hg and diastolic of 100 mm Hg. At 60 years of age upper limits are set at 180 mm Hg systolic and 110 mm Hg diastolic. Master and associates found that blood pressures for women are in general higher than in men after the age of 20.

The influence of obesity on hypertension and the frequent beneficial effects of simple weight reduction are familiar facts to all of us. It should be kept in mind that considerable error may exist in blood pressure readings taken on the obese arm. Recently, the taking of a blood pressure with the cuff on the forearm and auscultating over the radial artery at the wrist has been recommended as much more accurate in the obese patient. A limited personal experience would seem to bear this out.

Pickering\(^2\) differs with most authorities in doubting that a definite dividing limit between normal and pathological blood pressure levels can be set. When one further considers the observations of Wakefield\(^3\) in older women, this doubt is given further substance. He has studied some 500 female patients in the age group 70-100 over a 20 year span.

Seventy-five per cent of these patients at all times had essential hypertension with ranges of systolic pressures to 260 mm Hg and diastolic to 150 mm Hg. Many began with elevated blood pressure in the third and fourth decades and ran courses of 30 to 60 years duration. One lady, observed to her ninety-sixth year, carried a blood pressure of 260/140 mm Hg. The only treatment these ladies received during this twenty year period was encouragement and reassurance. Not a single patient manifested renal failure or severe nitrogen retention and careful study of the eye grounds revealed, for the most part, only low grade arteriosclerosis. Wakefield could find no correlation of symptoms or blood pressure levels nor did hypertension seem to affect the rate of progression of other disorders or the subsequent development of complications.

One of several cases of this type under our observation is an 83 year old lady who was first seen in 1941 at the age of 67. Her blood pressure at that time was 200/100 mm Hg. Through the years it has been recorded as high as 260/140 mm Hg, averaging 200-220/100-110 mm Hg. In the past 16 years she has survived surgical fixation of a fractured hip, small bowel resection for obstruction due to a fibroma with intussusception, and, sad to relate, her original physician, who was
many years her junior. During these years there has been no change in her retinal findings, cardiac size, electrocardiograms, urinalysis or nitrogen retention. Her main setbacks, aside from the foregoing surgical procedures, have occurred when well meaning but futile drug therapies were attempted. In one instance, digitalis was given because of the development of basal rales which subsequently proved to be due to chronic bronchitis, not heart failure. With the advent of Rauwolfia drugs, it was inescapable that she be given a trial; the only effect was repeated bouts of diarrhea. At the present time she is leading an active, useful life and gradually recovering from her long-standing fear of high blood pressure.

It is apparent, then, that elevated blood pressure in itself is not an indication for drug therapy. In the absence of overt signs of cerebral, retinal cardiac or renal abnormality compatible with disease due to hypertension, no treatment is justified beyond simple reassurance for those patients who are unfortunately aware of it and premeditated failure to inform the others of its presence.

If criticism is to be leveled at our management of blood pressure, how much more vulnerable is our approach to that which is under pressure, the blood itself. In this realm, of course, anemia is the commonest stumbling block. No other entity subject to objective evaluation is probably as frequent in occurrence nor as often maltreated or overtreated.

In the problem of anemia, as with blood pressure, we are faced first of all with the same task, i.e., interpreting physical measurements with regard to the patient, his symptoms and his findings. Do the figures presented to us for RBC counts, hemoglobin and hematocrit constitute a pathological deficiency and if so, what is the cause?

It may be said that “anemia”, in many instances, has about as much validity as so-called low blood pressure. Not uncommonly one sees patients with red cell and hemoglobin determinations in the range of fifteen per cent below the accepted lower normal limits; i.e., in women a red cell count of approximately 3.7 million and hemoglobin of 10 grams, with corresponding figures slightly higher in the male. These patients may have been observed over long periods and despite multiple functional complaints (including usually tiredness, weakness, etc.) and multiple hematinic treatments, their sound physical status and hematologic data remain unchanged. Extensive laboratory examinations disclose no other abnormalities.

An example in point is the case of a 59 year old colored female who has been observed and extensively examined for the past 15 years. On the basis of hemoglobin values consistently between 10 and 12 gm per 100 c.c., she has been plied continuously with iron, liver, vitamins and thyroid. Her many functional complaints of the neurasthenic variety have remained unaltered, as have her hematologic values. The morphology of her red cells, it should be stressed, is entirely normal.

Is this patient and many others we encounter in this category truly anemic? Should they be treated? Considerable light has been thrown on this type of problem by such studies as those of Nelson and Boyle4. They have shown in a group of 13 patients, some of whom — the females — had hemoglobins as low as 9.5 gms per 100 c.c., hematocrits as low as 34 percent, that the total circulating red cell mass was
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normal but that the plasma volumes were relatively or absolutely increased. Thus, these patients were not actually anemic but by virtue of an increase in the fluid portion of the blood, an apparent anemia was suggested as judged by routine blood determinations. Obviously, no amount of hematinics can alter this deceptive, but in reality, normal condition. Various designations have been applied to these patients. Dameshek³ terms them "anemia of neurasthenia"; others refer to it as dilution anemia or anemia of hypervolemia. The basic mechanism involved in the production of this disproportionate increase of plasma volume is not known but an awareness of this pseudo-anemia will help to avoid much totally unnecessary treatment.

Parenthetically, it is most interesting that the converse of this situation, that is, a pseudopolycythemia, equally subject to much confusion, exists on the basis of a decrease in plasma volume.⁴

Inasmuch as determinations of total blood volume and red cell mass are not as yet routine laboratory procedures, how can we avoid the pitfall of bland treatment of such patients for an "anemia" which is essentially an artefact? It goes without saying that the cornerstone of a careful and complete history and physical examination must be negative for pertinent abnormalities. Evidences of occult nitrogen retention, hypothyroidism, drug intoxications and chronic infections must be sought and excluded. With respect to the laboratory data, more reliance on hematocrit than hemoglobin is indicated, as the former will usually be less altered. A most careful analysis of the peripheral blood with particular reference to the morphology of the red cells is invaluable. The morphology of the red cells, I feel, remains entirely normal in those patients with pseudoanemia and if this is accurately ascertained by an experienced observer, it excludes most of the true anemias. I should like to underline this statement because this most basic examination of the blood smear for size, shape, hemoglobin content and hemoglobin maturity of the red cells properly done will give the clue to true or erroneous anemia in most instances. Unfortunately, few physicians are being trained these days to interpret the wealth of information available in the blood smear. Paradoxically, untold hours are given to mastery of the electrocardiogram waves — which is right and proper — but blood smears are too often relegated to technician personnel.

Granting, then, that treatment of pseudoanemia is contraindicated, what of the treatment of true anemia? Oddly enough, the answer should be the same — one never treats anemia. All of the current pharmaceutical huckstering for this or that multipurpose hematimic notwithstanding, anemia is a symptom, not a disease. Until the precise etiology is disclosed by logical step by step investigation, no treatment of the anemia is permissible. The one exception is that of acute hemorrhagic anemia where transfusions are sometimes required before the exact nature of the gastrointestinal lesion is disclosed.

Examples of the dire results of treating anemia as a symptom are too well known to this group to justify detailed recording. The cases of occult gastrointestinal neoplasms who are ineffectually treated by the "shotgun" method with subsequent tragic delay in the diagnosis of the underlying lesion; or the anemias due to bleeding from diaphragmatic hernias given the same all purpose hematinics made up of in-
sufficient amounts of the one needed element — iron; the pernicious anemia patients given preparations containing too much folic acid and inadequate B-12 who respond hematologically but relapse neurologically; anemias due to hemolysis, chronic infections, uremia, hypothyroidism or marrow replacing lesions carried unsuccessfully for months on all types of expensive drugs, delaying definitive diagnosis and management — all of these are well known to us and bear no further comment.

In the final analysis, both hypertension and anemia are symptoms. Failure to consider them as such may frequently lead to ineffective or unnecessary treatment. Knowledge of the physiological variations and pathophysiology involved with each will insure that specific therapy will be instituted only when indicated on the basis of an established disease state manifested by either of these symptoms.

BIBLIOGRAPHY