pulmonary artery: With some remarks upon the effect of this anomaly in producing cirrroid dilatation of the vessels. J. Anat. 20: 26, 1886.


Medical Eponyms

By Robert W. Buck, M.D.


“A systolic tug of the left false ribs posteriorly communicated by the diaphragm may be conspicuous. The recoil from the drag may be so distinct as to look and feel to the hand like pulsation, and in the first case in which I observed it, now more than 20 years since—a case of left empyema—it was taken for pulsation, and it was supposed that a pulsating tumour of some kind underlay the empyema. A post-mortem examination showed that the cause was adherent pericardium. I have often seen this tugging since, and in some cases it can be made to affect the right false ribs by causing the patient in the sitting position to lean over to the left so as to throw the drag of the heart upon the right half of the diaphragm. It must be added that this indication is not infallible, as the tugging has been observed when the heart was hypertrophied without adhesions.”


Ten dogs received hydralazine in varying dosages orally. The pattern and rate of toxic reactions varied directly with the dosage schedule. A progressive normocytic anemia, fever, anorexia, and cachexia appeared, often with terminal convulsions. Leukopenia was not apparent. LE cells were not found. Autopsy findings were compatible with a diagnosis of hemolytic anemia (i.e., congestion of the liver and spleen with hemosiderosis of both). No renal or other changes suggested systemic lupus erythematosus (SLE). From these experiments the authors concluded that hydralazine did not give rise to the SLE syndrome, and that in human beings there are 2 types of hydralazine reactions. The most common is a drug sensitivity in a patient with essential hypertension, the other type being a patient with latent SLE whose underlying disease is exacerbated by the use of hydralazine. In this latter type, the picture is typical of the underlying disease, and LE cells are found. The disappearance of symptoms and laboratory changes after withdrawal of a noxious agent in SLE is common.

MAXWELL
ta in iste effecto es discutite. Le risce de inducere edema pulmonar per le administration de diureteticos osmotic a patientes cardiac es recognize. Le valor clinic de diureteticos osmotic debe esser determinate per studios additional.

REFERENCES

It is with the living that medicine has to do. The living man must be studied in health as in disease; to the physician or surgeon the sick or wounded man is as the mineral to the geologist, as the star to the astronomer.—William Stokes His Life and Work (1804-1878) by his son WILLIAM STOKES. London T. Fisher Unwin, MDCCCXCVIII, p. 162.


When first I applyed my mind to observation from the many dissections of Living Creatures as they came to hand, that by that means I might find out the use of the motion of the Heart and things conduceable in Creatures; I straightwayes found it a thing hard to be attained, and full of difficulty, so with Fracastorius I did almost believe, that the motion of the Heart was known to God alone.—William Harvey. De Motu Cordis, 1628.


In these experiments a venous reflux wave was not demonstrated in left or right atrial pressure pulses at the time of the gallop sound. On this basis it is probable that the upward deflection in the jugular venous pulse that follows the V wave depends upon some components of the arterial pulse that are unavoidably included in the venous pulse. Atrial pressure is uniformly higher than that of the ventricle at the time of the gallop. It is concluded that sudden impact or stretching of the ventricle is a better explanation for the genesis of gallop sounds.

OPPENHEIMER
studio esseva evalutar le utilitate de Manvene como agente capace a modificar le stato lipidic sin producere grados significative de estrogenicitate in masculos con morbo cardiac coronari. Pro objectivos de comparasion, un grupo de patientes simile a illes tractate con Manvene esseva subjucite a un curso de Premarina que es un potente estrogeno conventional. Studios del lipidos seral e evalutationes clinic esseva effectuate pro omne le patientes durante un periodo de controlo ante le tractament e post 2 e 6 menses de medication.

A dosages supra 2,5 mg per die, Manvene producera significative alteraciones del lipidos seral, abassante tanto le proportion C/P como etiam le proportion lipoproteinie beta/alpha. Tamen, 50 mg de Manvene esseva require te pro attinger le alteraciones lipidic producere per 10 mg de Premarina.

In certe subjectos Manvene produciera alteraciones simile a illos producere per adeque doses de estrogenos conventional, sed in altere subjectos le effectos de Manvene esseva leve. Tamen, significative alteraciones del lipidos seral con minimal effectos lateral estrogenic esseva observate in 5 del 29 patientes. Iste constatation justifica le continuation del cerca de un vermente "non-estrogenic" derivat de estrogeno in que le effectos super le metabolismo lipidic remane intacte.

REFERENCES


At the outset I would like to emphasize the fact that the student of internal medicine cannot be a specialist. The manifestations of almost any one of the important diseases in the course of a few years will box the compass of the specialities.—William Osler, M.D. Internal Medicine as a Vocation. Med. News (N.Y.), 1897.

The environmental factor that correlates best with coronary heart disease is total fat consumption when expressed as a per cent of the total calories. This relationship between total fat intake and coronary heart disease rates for men below 65 may be expressed as follows: populations with fat intakes approximating 40 per cent of their total calories have high death rates; populations with total fat intakes below 20 per cent of total calories have low death rates; populations with intermediate fat intakes have intermediate death rates. Although there are degrees of susceptibility mediated by such unalterable factors such as sex, race, heredity, and body constitution and other environmental factors such as physical activity, obesity, and excessive tobacco, it is clear that the amount and type of fat intake is a major etiologic factor in the pathogenesis of coronary heart disease. Several groups of observers have conclusively demonstrated that feeding diets consisting of highly saturated fats results in high levels of blood cholesterol. Substitution or addition to the diet of certain oils, all naturally rich in unsaturated fatty acids, results in a consistent fall of serum and B-lipoprotein total cholesterol. A deficiency of unsaturated fatty acids, particularly of the essential linoleic, linolenic, and arachidonic acids, results in the formation of a saturated fatty acid-cholesterol complex. This is deposited to a greater extent in the intima of high-pressure arteries. These deposits in the aorta or other large vessels are relatively innocuous but when they are deposited in a strategic position in a coronary vessel, an occlusion or thrombosis may occur. The demonstration that feeding certain marine and vegetable oils containing unsaturated fatty acids causes a fall in serum and B-lipoprotein total cholesterol levels constitutes a major breakthrough for public health that may prove of practical value.

Harris
NONPENETRATING INJURY OF THE AORTA


31. DeBakey, M. E.: Personal communication to Storey.30


A new method has been developed to determine the unesterified fatty acids in blood plasma. This technic has been used in these experiments. During a fast arteriovenous differences were observed. These indicated a net transport of fatty acid from fat tissue to heart, voluntary muscle and viscera. Amino acids given during a fast had a similar smaller effect. The suggestion is made that adipose tissue releases more or less unesterified fatty acids into the blood to preserve "caloric homeostasis." A mechanism that is sensitive to the availability of nonfat calories seems to exist, which controls the release of unesterified fatty acids.
has had, but we have had no deaths in our obstetrical ward ever since Dr. Robert Wilson took over as professor of obstetrics about 9 or 10 years ago. So we know no reason for going ahead with mitral valvotomy during pregnancy.

The other important thing is that when you do get a death in pregnancy, if you should have one with rheumatic heart disease, it is important to ask yourself at what stage of pregnancy did it occur and why. Dr. Metcalfe showed this morning that the extra load of pregnancy occurs somewhere around the eighth month. If you are to attribute the death to mitral stenosis itself and not pregnancy, one should expect death at that time, but patients with rheumatic heart disease and mitral stenosis do not die at that time. If there is any death at all that I have been able to find in the recent literature, it is mostly in the immediate postoperative period. In other words, about 12 to 14 hours after the delivery. So I think that there is perhaps, with extremely rare exception, no indication for mitral valvotomy during pregnancy.

Dr. Metcalfe: I want to make this point. The success of the pregnancy after mitral valvotomy, either during the pregnancy or the year before, does not mean that the mitral valvotomy was indicated or successful.

Moderator Sprague: The final question as we end this discussion once again goes to Dr. Wolf. Every time a prominent national or local person dies of a heart attack, many anxious heart patients visit the office. Is it not important that their electrocardiograms are normal even with anxiety?

Dr. Wolf: Yes, I think it is important, but I think that we should be alert to the possibility that the changes we might detect at this time might be transitory changes associated with anxiety.

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The authors present the results of studies in man of early aortic atherosclerotic lesions. Structures with the general form of capillaries were demonstrated in the superficial layers of the intima by use of the alkaline phosphatase staining technique for endothelium. In 1 instance, red blood cells were found within a structure that showed enzyme activity. These vascular structures were interpreted as representing an extremely early feature of the disease. The foam-cell accumulations, the fibrous-tissue proliferation, and the deposition of hemosiderin, which are all found in the earliest atherosclerotic lesions, may be explained by recurrent hemorrhages from the rupture of “high pressure” intimal capillaries.

Maxwell


Observations on 6 patients with penicillin-susceptible streptococcal endocarditis who were treated with at least a 4-week course of large doses of penicillin V (2,000,000 units every 4 hours by mouth) and a streptomycin dihydrostreptomycin parenterally are presented. Clinical and bacteriologic cures were observed in all patients with no signs of relapse during follow-up periods ranging from 3 to 10 months. In 5 patients the serum penicillin levels were maintained above 1.6 units per ml. throughout the 4-hour interval between doses. Penicillin V serum levels, however, were consistently lower and less uniform than those obtained in other patients receiving equal doses of aqueous penicillin G intramuscularly, but were higher than those obtained with equal doses of penicillin G orally. Higher penicillin levels were found when the penicillin V was taken after meals. Simultaneous administration of aluminum hydroxide gel was found to depress the subsequent penicillin levels. Two patients developed nausea and vomiting of sufficient severity to interfere with therapy. One patient failed to obtain an adequate penicillinemia until probenecid was administered concurrently. Because the absorption of penicillin V taken orally is not predictable nor uniform enough to assure an adequate penicillinemia in this disease, the authors conclude that parenteral administration of penicillin still remains the preferred treatment for the majority of patients with subacute bacterial endocarditis.

Sagall