CIRCULATION
OFFICIAL JOURNAL OF THE AMERICAN HEART ASSOCIATION

EDITOR-IN-CHIEF
Herrman L. Blumgart, Boston, Mass.
Edith E. Parris, Assistant Editor

EDITORIAL BOARD

E. Cowles Andrus, Baltimore, Md.
Benjamin M. Baker, Baltimore, Md.
Janet S. Baldwin, New York, N. Y.
Richard J. Bing, St. Louis, Mo.
Stanley E. Bradley, New York, N. Y.
Howard B. Burchell, Rochester, Minn.
A. C. Corcoran, Cleveland, Ohio
André Cournand, New York, N. Y.
Jefferson M. Crismon, Stanford, Calif.
Clarence de la Chapelle, New York, N. Y.
Albert Dorfman, Chicago, Ill.
Charles T. Dotter, Portland, Ore.
Jesse E. Edwards, Rochester, Minn.
A. Carlton Ernstene, Cleveland, Ohio
A. Stone Freedberg, Boston, Mass.
Charles K. Friedberg, New York, N. Y.

Harry Goldblatt, Cleveland, Ohio
Robert E. Gross, Boston, Mass.
Hans H. Hecht, Salt Lake City, Utah
Louis N. Katz, Chicago, Ill.
Aaron Kellner, New York, N. Y.
Charles E. Koossman, New York, N. Y.
Walter F. Kvale, Rochester, Minn.
Jere W. Lord, New York, N. Y.
H. M. Marvin, New Haven, Conn.
Dickinson W. Richards, New York, N. Y.
Fredrick J. Stare, Boston, Mass.
Helen B. Taussig, Baltimore, Md.
James Watt, Bethesda, Md.
Earl H. Wood, Rochester, Minn.
Irving S. Wright, New York, N. Y.

ASSOCIATE EDITORS

Edgar V. Allen
Rochester, Minn.

Stanford Wessler
Boston, Mass.

Paul M. Zoll
Boston, Mass.

PUBLICATIONS COMMITTEE AMERICAN HEART ASSOCIATION

J. Scott Butterworth, Chairman
New York, N. Y.

Stanley E. Bradley
New York, N. Y.

Ludwig W. Eichna
New York, N. Y.

Irving B. Hexter
Cleveland, Ohio

Clifton D. Howe
Houston, Texas

James Watt
Bethesda, Md.

George E. Wakerlin, Medical Director, American Heart Association, New York, N. Y.

Herrman L. Blumgart
Boston, Mass.

Howard P. Lewis
Portland, Ore.

Carl F. Schmidt

Interlingua Summaries by Science Service, Division de Interlingua, New York, N.Y., Alexander Gode, Chief.


The authors analyzed 1950 electrocardiograms to determine the significance of depression of the T-U segment and of the presence of natural Q waves in certain leads. A trough-like deformity of the T-U segment extending below the level of the U-P segment was not found in 500 electrocardiograms of healthy adults. It was observed once (Fallot's tetralogy) in 250 patients with nonecoronary heart disease and among 15 with cardiac pain it was the only abnormality in 7. A natural Q wave in adults is defined as one not over 0.04 second wide and between 1 to 3 mm. deep except in lead 3 where it may be deeper. Among 500 electrocardiograms in healthy adults natural Q waves were not isolated to leads 1, 2, 3 or CR, or confined to the following combinations of leads: 1 and 2; 1 and CR; 2 and CR; CR and CR; 1, 3 and 3R; 1, 2 and CR; 2, CR, and CR; 1, 3, 3R and CR; 2, 3, 3R and CR; 3, 3R, CR, and CR; 1, 2, 3, 3R and CR; 1, 3, 3R, CR, and CR. For this reason, natural Q waves so occurring were regarded as evidence of myocardial injury. Although natural Q waves in leads 1, CR, and CR, are found in healthy adults, the combination occurred so much more frequently in older persons and those who later developed myocardial infarction that this combination may likewise prove to be an early electrocardiographic sign of a myocardial injury.

Soloff
2. Il existe differentias inter le magnitudes relative del reductiones postural e non-postural del pression de sanguine effectuate per IN 292, mecamylamina, e pentolinio. In 292 produceva forte reductiones del pression de sanguine non dependente del prender un postura erecte, durante que pentolinio produciva principalmente effectos postural.

3. Paradoxemente, pacientes con augmentos postural del pression sanguinee sub conditiones de controlo ante la administration de drogas a blocage ganglionic tende a exhibir forte reductiones postural del pression sanguinee post le action del agentes mentionate. Un explication possibile de iste phænomeno es discutite.

REFERENCES


One hundred sixty-eight patients with blood pressures of over 200/120 mm. Hg were subjected to partial or total adrenalectomy alone or in combination with various forms of sympathectomy. One hundred eighteen of the patients have lived and 50 died over the observation period from 3 months to 8 years. Forty-eight patients have responded with blood pressures in the range of 150/100 mm. Hg and 27 with blood pressures below that. Improvement in funduscopy examination has occurred in these patients. In 55 there was improvement in the electrocardiogram and decrease in heart size during the follow-up period. Blood urea nitrogen determination was a reliable index for renal function. In 70 of the 118 patients headache was a major complaint before operation, and in 62 patients the headache was relieved or disappeared entirely. Thirty-six of the survivors of adrenalectomy developed some pigmentation of the Addisonian distribution. Eight patients developed recognizable peptic ulcer. All patients with total adrenalectomies were treated with replacement cortisone and desoxy cortisol acetate. In hot weather supplementary salt was given.

The authors believe that fully 50 per cent of individuals with severe hypertensive disease subjected to adrenalectomy and sympathectomy exhibit a total improvement, that this type of operation is quite justified, and that the results obtained by these procedures are rarely equaled by the administration of anti hypertensive drug therapy. The authors point out that there is no successful procedure for selecting patients capable of a highly favorable response to the operation. At the present time they limit the operation to those in urgent need of control of hypertensive disease or those who respond poorly to antihypertensive drugs or who are unable to tolerate antihypertensive drugs.

Harvey

So Nature being perfect and divine, and making nothing in vain, neither gave a heart to any where there was no need, nor made it before there was any use for it, but by the same degrees in the forming of all animals passing through the constitutions of all creatures (as I may in the egg, Worm, and birth) it acquires its perfection in them all.—WILLIAM HARVEY. De Motu Cordis, 1628.
del agentes therapeutic (judicate in retrospecto). Le manifestaciones del periodo post-trac-tamental es satis commun sub apparentemente bon conditiones de providentia. Le studio de iste periodo debe esser includite in tentativas de definir optimos therapeutic pro febre rheumatic.

REFERENCES
3. FISCHEL, E. E., FRANK, C. W., AND RAGAN, C.: Observations on treatment of rheumatic fever with salicylate, ACTH, and cortisone. I. Appraisal of signs of systemic and local inflammatory reaction during treatment, the


Medical Eponyms

By ROBERT W. BUCK, M.D.

De Musset Sign. The de Musset sign of aortic regurgitation was described not by a physician, but by Paul de Musset, brother of the poet, in his "Biography of Alfred de Musset" (Biographie de Alfred de Musset, sa Vie et ses Oeuvres) Paris, 1877. The quotation is from chapter 14, pages 274-275.

"The illness so well cared for by Sister Marcelline had left him with a troublesome tendency to affections of the chest. . . . We called the doctors twice during the course of the winter; they bled him too often.

"Whatever they may say, I am convinced that their lancets caused him irreparable harm. At breakfast one morning in March, I noticed that my brother's head was bobbing involuntarily with each pulse beat. He asked my mother and me why we were looking at him with such a startled air. We told him what we saw, and he said, 'I did not think you could see it; but I will reassure you.'

"He made some sort of pressure on his neck with his index finger and thumb, and in a moment his head stopped marking his pulse. 'You see,' he then said to us, 'that this dreadful illness can be cured by simple and inexpensive means.'

"We were reassured, being ignorant, for we had just observed the first symptom of a grave malady to which he was to succumb fifteen years later."


72. White, P. D., and Glendy, R. E.: Trauma and Heart Disease. In Brady, L.; and


“Pulseless Disease”—Description by Robert Adams, 1827

An Historical Note

James H. Currens, M.D.

In reading some of the literature on Cheyne-Stokes breathing in the Dublin Hospital Reports, I have come upon an account by Robert Adams of an early description of “pulseless disease.” This happens to be in the same report that contains the classical description of Robert Adams of the Adams-Stokes syndrome in 1827. It is probable from the case report that the patient suffered a dissecting aneurysm of the aorta to account for the pulseless state rather than being due to the “Complete Ossification of the Coronary Arteries of the Heart and of the Aortic Valves,” which was Adams’ interpretation of the case. An autopsy was done, but the examination of the aorta was not described.

“A gentleman, etat. 68, of a pallid countenance, yet full and corpulent, while exerting himself in arranging some books on a high shelf in a library, suddenly felt severe pain in his chest, extending down his right arm, accompanied by a sensation of numbness: his sight became dim, he had vertigo, but did not fall. From that moment his breathing became oppressed, and in a little time he discovered that his pulse, which was unaccountably weak in his left arm, was altogether imperceptible in the right.

“On the following day, the 18th of October, he had still further grounds for alarm; the most careful examination could not detect the least pulsation in any artery in the body; nor was the movement of the heart sensible to the hand laid over the breast; an obscure undulating motion could alone be heard when the ear was for some moments attentively applied to the side of the thorax.

“His breathing was high and laborious, and could only be performed when the body was nearly erect, inclined a little backward or forward. At night he became worse, and enjoyed no sleep; he occasionally turned on his side with a wish to rest, but this posture increased the dyspnoea, and could be preserved only for a few moments.

“Although this gentleman was perfectly aware of the alarming nature of his symptoms, being himself a physician, he was cheerful, and his countenance was but little disturbed. His appetite was not good; but he was able to eat some chicken or fish for his dinner daily. His digestive organs performed their functions but imperfectly; he suffered great distress from flatulence; thus he remained for six weeks, with little alteration in his symptoms, except that his strength was observed declining daily and his breathing becoming more difficult; his rest during the night was still more imperfect; during the entire of this distressing period, no pulse was to be felt in any artery in the body. Although I daily made the most careful examination, it was in vain.”—Robert Adams. Cases of Disease of the Heart, Accompanied with Pathological Observations. The Dublin Hospital Reports and Communications in Medicine and Surgery 4: 443, 1827.


An occasional observation of improvement of psoriasis in a patient being treated with Tromexan because of myocardial infarction is reported. In order to learn whether the improvement was coincidental or had other meaning, 17 patients with psoriasis have been treated with Tromexan or with Sintrom or Dicumarol; 3 of these did not respond to treatment, 8 showed regression, 6 became free of lesions. It was noted that the anterior surfaces cleared more rapidly than the posterior, and that the scalp cleared first while the legs were last. The time required for complete regression varied between 2 and 6 months. It was found that the therapeutic effect was obtained when a “threshold” dose was given; lower doses were ineffective, while larger single doses did not produce better results; repeated “threshold” doses (limited in number only by their anticoagulant effect) were more effective. Tromexan was commonly effective in single doses of 300 mg. and was better suitable for this purpose than comparably effective doses of Sintrom or of Dicumarol, because of its lower anticoagulant effect. There was no relation between the prothrombin level and the therapeutic result obtained. However, the use of antagonists to the anticoagulant (menaphthone), together with the drug, inhibited its beneficial effect.
consistente de 200 g de crema de lacte con un contorno de 40 pro cento de grasa. Le lipemia del plasma es cesaba mesurato a intervalos de un hora per determinar photometricamente le densitate optic del plasma. Electrocardiogrammas esese obtenate in stato jejun e a intervalos de un hora post le ingestion del repasto grasse. Le patientes qui exhibiva alterationes electrocardiographic post le repasto grasse esesessubmitte al mesme test, sed nunc illes recipeva additionalmente 250 mg de mucina gastric, administrate simultaneamente con le repasto de grasia. Mucina gastric inhibiva invariabilemente le hyperlipemia alimentari, e in un mediate del casos illo reduciva le alterationes electrocardiographic associate con le hyperlipemia.

REFERENCES


Measurements of the cardiac output by the direct Fick principle utilizing cardiac catheterization in control subjects and patients with chronic congestive heart failure showed that exposure to a hot and humid environment increased the cardiac output, stroke volume of the ventricle, mechanical, and physiologic cardiac work, and tension upon the walls of the ventricles in both groups. There was less of an increase in the patients with congestive heart failure than in the control subjects. The increase in cardiac work and output was found to occur more as a result of a larger stroke volume than greater cardiac rate. The therapeutic implications of the observed stressful cardiac responses to a hot and humid environment are discussed.


Twenty-nine deaths were recorded in a series of 1,706 thoracic aortographic examinations. Use of a 70 per cent concentration was fatal 8 times more frequently than when a 30 or 35 per cent concentration was employed. Retrograde carotid injection was attended by a higher mortality than brachial or catheter injection. No deaths followed the use of retrograde brachial aortography with 30 to 35 per cent media. Most of the deaths and severe reactions appeared to be associated with cerebral damage. Less frequent were renal, cardiac, and respiratory complications. The author recommends that in children under the age of 4 lesser concentrations be employed; in adults 20 to 30 ml. of a 70 per cent agent is relatively safe. The use of a general anesthetic increases the hazards. The roles played by catheter positioning, patient's state of hydration, electrocardiographic monitoring and sensitivity testing are discussed.

SCHWEDEL
DEEP S WAVE IN RIGHT VENTRICULAR HYPERTROPHY

pertrophia dextero-ventricular, e in omnes profunde undas S esseva presente in V1, V2, o V3.

Proportiones R/S de minus que 1,0 in V1 esseva presente in 3 casos. Le voltage de R in V1 esseva infra 0,5 millivolt in 3 casos. Undas R in aVR esseva absent in 3 casos.

Le vectocardiogrammas de iste patientie indicava hypertrophia dextero-ventricular e illustrava le rationes pro le constataiones electrocardiographic.

REFERENCES


Atrial fibrillation appeared following mitral valvulotomy in 47 per cent of 77 patients who had a normal sinus rhythm prior to surgery. Fibrillation appeared most frequently on the second postoperative day and rarely on the day of surgery. Spontaneous resumption of sinus rhythm occurred in 10 patients, and quinidine restored sinus rhythm in 16 patients, leaving 10 who had persistent postoperative atrial fibrillation. Since the ventricular rate is high in patients not receiving digitalis, and since additional digitalis was needed to slow the ventricular rate of most patients who developed postoperative atrial fibrillation, all patients should probably be fully digitalized prior to valvulotomy. Since spontaneous resumption of sinus rhythm occurs in many patients, quinidine may be withheld until the seventh postoperative day. It may also be more effective at that time, since factors responsible for the onset of atrial fibrillation have by then apparently diminished.

Simon
treatment should not be employed in patients who have been getting progressively worse or in those who recently have had myocardial infarction. The best results are obtained in persons whose condition has remained more or less stationary or has progressed only slightly over a period of 6 months or more.

MODERATOR KATZ: Does anybody else have a last word before I close this meeting?

DR. BURCH: I would like to come to the support of Dr. Parker about bleeding the patient with polycythemia in association with cor pulmonale. Several of our patients have obtained dramatic benefit from bleeding, which is too frequently neglected in the management of such patients. We bleed them gradually, depending on the clinical state, until the hematocrit level becomes normal; our results have been impressive almost invariably.

MODERATOR KATZ: You see, we have had the traditional kind of panel, each panelist expressing his own viewpoint—sometimes this was in accord with the views of others and sometimes there were disagreements. I am reminded of what a psychoanalyst said about tranquilizer drugs. This psychoanalyst said, "You know peace of mind is a fine thing but what would happen to the world if everyone was agreeable. After all progress in this world has been made by neurotics, by those who disagree."

I regret that time will not permit consideration of the many questions sent up by the audience. But I think your patience has been tested long enough.

I think you will agree that the purpose of the panel has been fulfilled in that it has stressed what we do not know. It has raised many questions still unsettled. It has shown the practicing physician how careful he must be in applying new knowledge and accepting old dogmas as valid. This is not to say that we must chide ourselves for doing so badly in medicine. Actually we are doing exceedingly well, progress has been rapid, but we must not become smug and believe we know everything.

I wish to thank the panelists for their contributions and for so agreeably accepting the manner of conducting the panel that your moderator suggested. And finally may I thank all of you for listening so quietly and patiently to what we have had to say. I hope you have found this session profitable, illuminating, and thought provoking.


Ten patients with unexplained pulmonary hypertension are reported. Six presented the characteristic picture of an apparently healthy, young female who developed progressive dyspnea with substernal discomfort and fatigue. Effort syncope was common. Physical signs of pulmonary hypertension and a malar flush were found, but there was little cyanosis. Electrocardiogram, fluoroscopy, and cardiac catheterizations were confirmatory. Primary contraction of terminal muscular pulmonary arteries was usually found. In 2 other patients pulmonary hypertension was due to a generalized lung arteritis. Such patients showed, in addition, digital arterial involvement. In 2 patients, cyanosis and hemoptysis were noted. In these bronchopulmonary anastomoses were found. Therapy was largely ineffective.

KURLAND