ABSTRACTS
Editor: STANFORD WESSLER, M.D.

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BLOOD COAGULATION AND THROMBOSIS

In a 10 year period, pulmonary embolism was demonstrated in 118 of 943 autopsies on adults. Fatal pulmonary embolism occurred in 22 patients postoperatively. Four of these died in the 24 hours following operation. In these patients a low grade fever and tachycardia associated with reduced physical activity (at least 1 week of hospital confinement prior to surgery) were noted. This combination of fever, tachycardia, and activity restriction, and fatal pulmonary embolism suggested that thrombosis was present prior to surgery. Patients with activity restriction, fever, and tachycardia should be regarded with suspicion of thrombosis, and pre-operative prophylaxis is of equal importance to postoperative prophylactic care.

KITCHELL

HYPERTENSION

Thirty-eight patients with malignant hypertension, who did not react to other antihypertensive drugs, were given 10 mg. of camphidonium, 0.2 mg. of reserpine, and 1 mg. of serpentine once a day for 3 to 4 weeks. It was found that the hypotensive effects of camphidonium and reserpine were additive, so that the combined use of both drugs allows smaller doses of the ganglionic blocking agent to be used, resulting in less unwanted side effects. Serpentine was shown to increase cardiac output and to decrease peripheral resistance. Addition of serpentine to camphidonium and reserpine prevents major orthostatic hypotension, which ordinarily results from ganglionic blocking agents, and thus allows ambulant treatment with these agents.

LEPESCHKIN


Dietary treatment of rats with propylthiouracil (PTU) prevented the development of elevated blood pressure usually accompanying kidney encapsulation with latex envelopes. Within 4 weeks after dietary administration, PTU also reduced to control levels the elevated blood pressure of rats whose kidneys were encapsulated 9 weeks previously. With regard to dosage, 0.1 per cent PTU appeared to be more effective than 0.06 per cent. This drug (0.1 per cent) also reduced the blood pressure of normal rats. When PTU was administered to growing rats, body weight remained at the level at which administration began. Older rats generally lost weight on the drug, although the extent of weight loss was not consistent among 3 separate lots of PTU used. The reduction in blood pressure of the "encapsulated" rats could not be attributed solely to the weight loss produced by administration of PTU.

WENDKOS

622 Circulation, Volume XIX, April 1959

The intragastric administration of either licorice or ammoniated glycyrrhizin for 50 days to male rats, unilaterally nephrectomized and given 0.87 per cent sodium chloride solution to drink, caused a blood pressure elevation consistently higher than that induced by desoxycorticosterone acetate (DCA) under similar conditions. Renal and cardiovascular lesions, consisting essentially of arteriolar necrosis and hyalinization, were very severe in the DCA and licorice-treated groups, while they were milder in the glycyrrhizin group. Licorice or DCA treatment caused a significant polydipsia and growth impairment, whereas the curves of fluid intake and body growth of the glycyrrhizin-treated group remained approximately like those of the controls. These results support some previous reports on the steroid-like activity of licorice. It seems that the hypertensive properties of licorice could be attributed to its glycyrrhizin content, while polydipsia and some other effects may be due to other constituents.

WENDKOS

METABOLIC EFFECT ON CIRCULATION


The total and trichloracetic acid soluble fractions of glycogen were determined in various portions of the rat heart. The amount in each portion was compared on the basis of the nitrogen content of the same sample. The total glycogen concentration was highest in the atria, lowest in the left ventricle, and intermediate in the septum and right ventricle. A similar distribution was found for the trichloracetic acid soluble glycogen. The possible role of glycogen in cardiac energetics and structure is discussed.

WENDKOS


The oxygen tension in the myocardium of 19 dogs was recorded by means of the platinum polarograph during and following a period of rapid systemic arterial bleeding. The oxygen tension in the myocardium fell sharply with the fall in blood pressure, rose promptly with the rise of blood pressure when 1-norepinephrine was injected intravenously, fell again when the 1-norepinephrine was discontinued and returned nearly to the control levels when the reservoir blood was transfused. In 3 animals, a brief drop in blood pressure preceded the rise in myocardial oxygen tension when the 1-norepinephrine was injected.

Survival experiments were conducted in 17 pairs of dogs. Each pair was bled rapidly at the same time. One animal received a constant infusion of 1-norepinephrine beginning 5 minutes or less after the start of bleeding and severe hypotension and the other served as a control. Animals surviving 2 hours were transfused and returned to their cages and were subsequently autopsied 7 days later. Only 2 of the 17 treated animals died and these before the end of 2 hours of the experiment. Eleven of the 17 untreated controls succumbed, 7 during the 2 hours of the experiment and 4 within the next 12 hours. These experiments indicate that 1-norepinephrine corrected the myocardial ischemia which occurred during severe hypotension from bleeding and increased the chances of survival provided it was given less than 5 minutes from the beginning of severe hypotension. There was other evidence to suggest that if administered 10 minutes or longer after the onset of severe hypotension, 1-norepinephrine did not have a beneficial effect and might be injurious.

BROTHERS

PHARMACOLOGY


The role of Fluothane, a potent, volatile, non-explosive anesthetic agent, was studied in epinephrine-induced arrhythmias of the dog heart. The dogs were divided into 4 groups: (1) 9 dogs received an average total dose of 74 mg. per Kg. of thiopental over an average period of 313 minutes, forty-two minutes after the drip was stopped Fluothane was started; (2) 17 dogs were given minimal thiopental, maintained on open drop Fluothane and then tested with intravenous epinephrine; (3) 7 dogs were given intramuscular doses of epinephrine or norepinephrine after the arrhythmic threshold for intravenous doses were determined; (4) 11 dogs were treated as in (2), then the respiratory pump was turned on, and the dogs were passively hyperventilated. The Fluothane concentration was reduced and the trials with intravenous epinephrine were carried out. The results showed that Fluothane increased the sensitivity of the dog heart to the effect of epinephrine and norepinephrine. Under similar
conditions, thiopental did not. Intramuscular epinephrine and norepinephrine produced serious, but never fatal, arrhythmias in dogs under Fluothane anesthesia. It required 159 times the intravenous dose to produce a similar effect by intramuscular injection. There does not seem to be an absolute contraindication to using Fluothane and epinephrine in normal, healthy patients, but it is suggested that epinephrine be omitted in the presence of cardiovascular disease.

RINZLER


Substances such as 1/6 M sodium lactate, 5 per cent glucose, and Locke’s solution, exhibited no ectopic excitatory effect by intracoronary injection in the uninfarcted dog heart. After infarction, the same substances produced high-frequency ventricular tachycardia and fibrillation. It is conceivable that the injected fluid mobilized stagnant extravasated fluid containing substances, that were excitatory to live tissue. Application of these observations to the management of infarction when such substances are injected intravenously remains to be explored.

AVIADO


The intracoronary administrations of epinephrine and norepinephrine in beating, fibrillating, or potassium-arrested dog hearts indicated that the primary action of both amines was coronary vasoconstriction. Their vasodilator action was secondary to their stimulating effect on myocardial metabolism, particularly to the induced hypoxia of the heart muscle.

AVIADO


The subjects were 28 women who were normal except for complaints judged to necessitate dilation of the cervix and curettage of the uterus. The ages ranged from 22 to 54 years. The investigation dealt with the effect of hypercarbia on the production of ventricular arrhythmias during cyclopropane anesthesia. Intravenous infusions of epinephrine or norepinephrine at rates of 4 to 26 μg. per minute produced similar arrhythmias in 6 of 8 subjects. The concentration of the amines in arterial plasma during periods of arrhythmias were much greater when the arrhythmias were produced by infusion than when they were caused by hypercarbia, suggesting that an increase in catechol amines was not the cause of arrhythmias. Atropinization did not affect the ability of hypercarbia to initiate ventricular arrhythmias to any extent. Bilateral blockade of the stellate ganglia with a local anesthetic made hypercarbia ineffective in producing ventricular arrhythmias, but did not change the ability of infused amines to do so. The conclusion is that hypercarbia increases the rate of liberation of catechol amines from sympathetic nerves terminating in the myocardium and that this causes ventricular arrhythmias.

RINZLER

PHYSIOLOGY


The blood flow, the aortic and pulmonary artery pressure, the pressure in the right and left atrium, the ventilatory volume, and the carbon dioxide content of end-expiratory air were recorded in cats under chloralose anesthesia, with open chest and fixed volume of artificial ventilation. In another series of animals, with spontaneous respiration and closed chest, only the pulmonary artery, or the femoral artery pressure, the carbon dioxide content and the ventilatory volume were recorded. In the experiments in artificial respiration, carbon dioxide was added to oxygen; in the series in spontaneous breathing, carbon dioxide was suddenly added or gradually increased by rebreathing. The mean arterial pressure, the effective pressure difference, the resistance, and the ‘relative vascular diameter’ were calculated from the data recorded. In the animals in artificial, fixed volume respiration, increase in carbon dioxide up to 20 per cent failed to induce hemodynamic changes; only at such carbon dioxide concentration did the pulmonary artery pressure increase in 4 experiments out of 25; the aortic pressure increased in 2 observations. In the animals breathing spontaneously the respiratory volume and the pulmonary arterial pressure increased with the increasing concentration in carbon dioxide; the circulatory effect was probably due to the increased work of respiration. The possible significance of increased carbon dioxide alveolar tension as a regulatory mechanism of alveolar perfusion was
briefly discussed. It was concluded that the results of acute experiments in the animal cannot be applied to the interpretation of physiopathologic mechanisms in chronic disease in man.


In 73 cardiac patients the pressure in the superior vena cava or in the right atrium was recorded synchronously with the intraesophageal pressure: the height of the a wave was measured in different phases of respiration; using these data, and adopting the intraesophageal pressure an estimate of the intrathoracic pressure, the 'transmural' pressure was obtained. Similarly, the pressure in the intra-abdominal inferior vena cava was compared with the infragastric pressure. The velocity of the pulse wave in the venae cavae was also recorded in some cases through a double lumen catheter. It was found that the transmural pressure tracing differed from the atrial or venous pressure directly recorded. In inspiration the filling pressure increased in the intrathoracic superior vena cava and in the right atrium; the intra-abdominal inferior vena cava was constricted by the increased abdominal pressure. These observations support Donder's theory of respiratory suction of blood into the chest. The velocity of the pulse waves increased with the increase in transmural pressure. It was pointed out that these data had been obtained in patients with nearly normal intrathoracic pressure and lying horizontally; the effect of standing and of abnormal intrathoracic pressure was surmised.


 Arrest of the dog heart by potassium chloride appears to weaken the connective tissue elements so that the myocardial fasciculi may become separated upon fixation. The erythrocytes become swollen and show more rouleaux formation, probably due to the osmotic effect of the potassium chloride solution. The density of erythrocyte-filled capillaries is shifted from the epicardium (in the normal heart) to the endocardium. Arrest of the heart by potassium chloride for the purpose of yielding a dry field for cardiac surgery makes the heart more sensitive to stress, so that reperfusion of the coronary system at the conclusion of a period of cardiac arrest causes profound tissue hemorrhage.


In the presence of a normal heart rate, cardiac index, and systemic blood pressure, the oxygen uptake is 8 to 10 ml. per minute per 100 gm. of left ventricle. In the open chest dog, the corresponding oxygen usages of the left ventricle whose external work has been reduced to zero are as follows: 2 ml. in complete arrest with vagal stimulation or with intracoronary potassium injection; 3.8 ml. in fibrillation; and 3.4 ml. in the empty but beating heart. The experiments with vagal stopping of the heart lay the groundwork for calculation of the oxygen debt of the heart. A constant finding is the apparent increase in calculated usage of oxygen (1.5 ml.) above the control level immediately after induction of vagal asystole. This increase is believed to be caused by the preceding cardiac activity and could be called oxygen debt.


In anesthetized dogs, electrically induced atrial tachycardia induced a very temporary initial decrease in arterial blood pressure, cardiac output and coronary flow, then a return of all parameters to the control level. Ventricular tachycardia essentially caused the same phenomena but the initial decrease in all 3 measurements was more marked than that of atrial tachycardia of the same rate. The mechanisms underlying these changes were discussed.


In dogs anesthetized with pentobarbital, with the heart denervated, the adrenal glands ligated, and the sinoatrial node crushed, pairs of stimuli (S1-S2) were applied to the atrium or ventricle, varying the delay between S1 and S2. When the atrium was stimulated, the following relations were measured: S1-S2: A1-A2; A1-V1; A2-A2; V2-V2. The corresponding curves for ventricular stimulation were also plotted. Calling R1 and R2 the responses at a given site, the S1-S2:R1-R2 curves usually showed a horizontal branch. This branch indicated that impulses stimulated at different moments in the refractory cycle could reach the recording site at the same moment of the cycle. A theorem is demonstrated which
proves that the constancy of the R-T-R interval cannot be explained merely on the basis of slowed conduction rate of R, but implies that the impulse stops at some point in the conducting path. For A-A or V-V propagation, this stop is due to delay in the initiation. For A-V or V-A propagation, the stop occurs at some site in the intermediate conducting tissues. The stop in these cases is due to the existence of a prolonged functional refractory period.

**Wendkos**


Experiments performed on 2 species of monkeys showed that they possess reflex mechanisms similar to those of other mammals. The carotid sinuses are provided with baroceptors that regulate systemic arterial pressure. The adjoining carotid bodies contain chemoceptors that are sensitive to lobeline and cyanide ion, and that induce reflex stimulation of respiration.

**Aviado**


It has been postulated by numerous investigators that intraventricular diastolic suction somehow contributes to ventricular filling. This postulate has been based mainly on reasoning from very meager evidence until 1952, when evidence in favor of this concept started to appear. The existence of ventricular diastolic suction has been only established when the ventricle contains a more nearly normal residual volume due to ejection against the resistance of a fluid column. The quantitative contribution of suction to ventricular filling at various residual volumes and various levels of cardiac activity is still unknown.

**Aviado**


Coronary blood flows (electromagnetic) and peripheral coronary pressure (distal to occluded branch) were measured in anesthetized dogs. Electrical stimulation of the sympathetic cardiac nerves, increased myocardial contraction, as did levaterenol, and caused a slight degree of coronary arterial dilatation. Parasympathetic fibers exerted no significant effect, although acetylene diminished coronary arteriolar tone.

**Aviado**

**REVIEWS IN CARDIOVASCULAR DISEASE**


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Circulation. 1959;19:622-626
doi: 10.1161/01.CIR.19.4.622

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1959 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/19/4/622.citation

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