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# Transfer of $Cd^{115m}$ across the pericardium of dogs

The rates of transfer of radiocadmium, $Cd^{115m}$, across the intact pericardium of dogs were measured. This isotope was studied because of its many interesting features, and because it is an important and interesting trace element found in mammals, including man.[1-3] Cadmium is essential for certain enzyme functions.[4] Chronic cadmium-poisoning has been reported to produce cardiomegaly.[5,6] However, cadmium is not a useful experimental tool for the production of cardiomegaly, as originally anticipated.[7]

$Cd^{115m}$ was instilled in the pericardial cavity in Ringer's solution in a concentration of approximately 5.5 mg. per 50 ml. or 110 mg. per liter, which is far greater than the concentrations normally found in mammalian tissues.[3] This concentration did not change the electrocardiogram. By methods and mathematical analyses previously described[8,9] the mean rate of transfer was 0.176 μg per square centimeter per minute; the range was 0.03 to 0.36 μg/cm.²/min. This represents a daily bidirectional rate of transfer of about 150 mg. across the pericardium of the dog. Of the ions studied in this laboratory by this technique ($D_2O$, $H^3$, $Cl^{36}$, $Na^{22}$, $Mg^{28}$, $Cd^{115m}$),[8,9] $Cd^{115m}$ had the slowest rate of transfer. This rate of disappearance of $Cd^{115m}$ from the pericardial test fluid probably represents not only loss by transfer through the pericardium but also $Cd^{115m}$ bound to protein and epithelial cells of the pericardium. Thus, like other serous membranes there is an extremely rapid transfer of many elements and complex substances across the pericardium. Because the pericardium is normally relatively dry in spite of the rapid and constant exchanging of fluid and electrolytes, the accumulation of fluid and solutes in pericardial diseases remains a great mystery.

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# A plea for standardization of definitions in cardiovascular functions

In 1950, a group of pulmonary physiologists[1] in the United States met to standardize their symbols and terms with the view of avoiding the confusion that existed in the English literature due to semantics. At present, there is a dire need for a similar agreement in the cardiovascular field, not so much on symbols as on definitions. To illustrate the state of confusion, the following terms might be considered.

The terms "hypoxia" (anoxia), "hypoxemia" (anoxemia), and "ischemia" are used synonymously by some authors. In general, when tissue cells do not consume sufficient oxygen to meet their *requirement under a given set of conditions*, they are said to be suffering from hypoxia (anoxia).[2,3] The term "hypoxemia" denotes a reduced oxygen *content* of arterial blood,[2] irrespective of the adequacy of the supply of oxygen to tissue cells. The low oxygen content may be the result of low saturation of hemoglobin (low $pO_2$) or low hemoglobin content of blood (anemia). Here, the importance of blood flow to the tissue must be borne in mind. Thus, a mild degree of hypoxemia may not result in hypoxia of a tissue if the blood flow and/or venous desaturation are proportionately increased. Some investigators[4,5] have used the term "hypoxia" to describe hypoxemia due to low arterial saturation. Berne, Blackmon, and Gardner[6] have used the term "hypoxia" for a reduction in the oxygen *content of tissue* rather than its oxygen *consumption*, presumably on the assumption that the two decrease proportionately. However, it is conceivable that under certain circumstances the oxygen content and oxygen consumption of a tissue may vary independently (e.g., hypothermia, cyanide poisoning), and, since there are no methods to quantitate tissue oxygen content, this concept of hypoxia does not seem to be a useful one. Some investigators[7] have used the term "anoxia" to describe arrest of blood flow (complete ischemia). This is not justified on the grounds that in complete ischemia, although anoxia is the most prominent disturbance, there are other derangements that may be functionally significant. In complete ischemia the tissue is deprived of *all* the nutrients that are brought by plasma (e.g., glucose, etc.), whereas in anoxia it is deprived only of oxygen. Besides, the metabolic products ($CO_2$, lactic acid, etc.) which are not washed away accumulate to very high concentrations. These other changes may contribute significantly to "wreck the biochemical machinery" of tissue cells. Hence, anoxia and ischemia are not synonymous.

Another example of confusion is the concept of "work" and "power" of the heart. External mechanical "work" as calculated conventionally by physiologists has always been expressed *per unit of time*, whereas in physics "work" does not involve time. For physicists, work per unit of time is "power." Some investigators[8] have complicated physiologic semantics by introducing the physicists' definition of "work" and "power." This is unfortunate in that these physical definitions tend to aggravate the confusion among physiologists, and, furthermore, the term "power" for a long time in physiology has been associated, somewhat loosely, with "force" of muscle contraction (be it in heart or skeletal muscle). According to Klip,[9] some authors[10] have also defined myocardial "power" as the quotient of diastolic aortic pressure × blood volume in the left ventricle divided by duration of isometric contraction. Klip has pointed out that this is not physically justified because the energy needed to bring the left ventricular pressure up to the value which can open the aortic valves is *not* equal to the product of diastolic pressure in the aorta and the volume of blood in the left ventricle. In our opinion, one cannot speak of "power" during this period for the simple reason that practically no external "work" is done during the period of isometric contraction. The volume of intraventricular blood is practically unchanged during this period.

In view of these and other considerations, we believe that some of the terms used by physical scientists are not entirely appropriate in physiology. Nevertheless, a judicious selection of physical definitions is highly desirable.

The term "work capacity" of the heart has also been used in different ways. This is discussed in a separate communication.[11]

A further illustration of misleading terminology is related to the expression "mean or average systolic pressure" in arteries. Wiggers,[12] and Sarnoff and associates[13] have used this term for the mean pressure *during* the period of ventricular ejection. But conventionally, "systolic" pressure refers only to the momentary *peak* pressure during ejection. Hence, the term should refer more appropriately to the average of peak pressures which exist in the arteries over a period of time. Although the conventional definitions of "systolic" and "diastolic" arterial

0024171