crease in automaticity once again related to a change in the slope of phase 4 of the action potential and increase in automaticity occurred only after frank loss of cellular integrity as evidenced by a loss of resting potential.

Recently, in a study of the electrical response to high concentrations of certain fluoroalkane aerosol propellants, the surviving animals demonstrated a higher Pco2, a greater base deficit and a lower pH than did the nonsurvivors to a statistically significant degree. In this latter study the derangements were not drastic, but were sufficient to make possible a highly reliable separation of survivors and nonsurvivors.\textsuperscript{11}

A service has been rendered if the paper by Rogers et al has nudged us out of our complacency and made us critically examine the variables in the clinical situation of respiratory insufficiency. If we take the authors' advice and become wary of glibly blaming the occurrence of ventricular arrhythmias on derangements of blood pH and oxygen tension, and if we continue to search for other causal aspects in the patient's internal environment, then we may discover and deal with the more clinically relevant variables in his problem.

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\textbf{Left Ventricular End-diastolic and Filling Pressures in Assessment of Ventricular Function}

Cardiac function can be assessed by examining the performance characteristics of individual muscle fibers or of the overall pumping characteristics of the whole ventricle. In man it is often difficult to obtain the parameters that assess cardiac muscle directly and this is particularly true in the acutely ill. Instead, left ventricular function has usually been examined on the basis of the Frank-Starling principle which relates the force of ventricular contraction to the myocardial fiber length immediately before the onset of ventricular systole. Since neither of these is easy to measure, cardiac output, stroke volume, developed pressure and left ventricular (LV) stroke work have been used as indices of the force of contraction and LV end-diastolic volume (EDV) and end-diastolic pressure (EDP) have been used as indices of fiber length.

In subjects with normal cardiovascular systems, pressures in the pulmonary artery, left atrium and left ventricle are equal at end-diastole (ED). Similarly, left atrial pressure at ED equals mean left atrial pressure, and LVEDP is very close to mean left ventricular diastolic pressure (LVDP).\textsuperscript{1,2} Acute increases of LVEDP by hypervolemia result in a proportionate increase of mean left atrial pressure.\textsuperscript{3} However, LVEDP exceeds LV diastolic pressure prior to atrial contraction (LVDP pre “a”) by an average of 1.6 mm Hg.\textsuperscript{2} Thus, in normal subjects, pulmonary artery EDP, mean and end-diastolic left atrial pressures, mean LVDP and LVEDP are usually the same and can be used interchangeably.

Left ventricular filling pressure may be a convenient term to refer to all of these parameters.

The situation is different in patients with heart disease. For example, in some patients with acute myocardial infarction pulmonary artery EDP is higher than mean left atrial pressure due to an increased pulmonary vascular resistance.\textsuperscript{4} In mitral stenosis left atrial pressure is higher than LVDP. In patients with left ventricular disorders such as hypertrophy, myocardial diseases or myocardial infarction,\textsuperscript{44} powerful atrial contraction may produce a LVEDP significantly higher than mean left atrial pressure, whereas pulmonary artery EDP and mean left atrial pressure are the same as LVDP pre “a” wave. Unfortunately, in these patients altering LV performance acutely by atrial pacing, increasing aortic pressure or by administration of digitals may result in alterations of LVEDP that are different in magnitude and direction from the changes seen in
the indirect estimates of LVEDP. Therefore, to use the term left ventricular filling pressure interchangeably between LVEDP and the other pressures just enumerated seems inappropriate. A powerful atrial contraction that elevates LVEDP without a proportionate increase of mean left atrial pressure has the advantage of minimizing the consequences of an elevated LVEDP on the pulmonary circulation. Thus, an elevated LVEDP helps to maintain the force of ventricular contraction and since the mean left atrial pressure does not increase to the same degree the risk of developing pulmonary edema is reduced.

In patients with elevated LVEDP, a presystolic wave may be seen in the pulmonary arterial pressure pulse. It is felt that this wave represents retrograde transmission of pressure resulting from left atrial contraction and has been called pulmonary arterial "a" wave. A good correlation has been demonstrated between LVEDP and the pressure at the peak of this wave. Although this wave was identified in 60 to 93 percent of patients with LV dysfunction when fluid filled catheters were used, it was present in only 7 percent of patients when high fidelity catheter tip micromanometers were utilized. The constancy of the relation of the peak pressure of this wave to LVEDP under varying clinical situations has not yet been defined. In many patients, the identification of this wave will be difficult because pulmonary arterial pressure pulses recorded by using fluid filled catheter systems often have motion artifacts induced by the beating heart.

Pulmonary artery EDP and mean LA pressure have been reported to be the same or lower than LVEDP in patients with "coronary artery disease" or with angina pectoris. Patients with angina but without a previous myocardial infarction usually have a normal LVEDP at rest and many, but not all, patients with previous myocardial infarction have an elevated LVEDP at rest. Thus, it appears that in these patients under resting conditions the discrepancy between pulmonary artery EDP and LVEDP is usually related to an elevated LVEDP resulting from a previous myocardial infarction; however, this inequality is not invariably present.

Recently, LVDP prior to atrial contraction has been termed LVEDP pre "a" or just LVEDP. Ventricular diastole ends after atrial systole and before ventricular contraction has started. End-diastolic pressure can only occur at one point in diastole. Therefore, LVDP at end-diastole is LVEDP, is measured at the "z" point and occurs in an average time of 0.052 sec after onset of the QRS of the electrocardiogram. Left ventricular diastolic pressure prior to atrial contraction could most appropriately be referred to as LVDP pre "a."

Both in experimental studies and in patients with LV dysfunction, it has been recognized for a long time that increases in LVEDP may not reflect predictable increases in LV diastolic volume because of alterations of LV compliance. Thus, there are limitations to the use of LVEDP as an index of cardiac failure. However, it can be accurately measured with relative ease and there is widespread appreciation of its limitations. LVEDV would be superior to LVEDP in the assessment of ventricular function because it is more closely related to myocardial fiber length. In man, LV volumes can be measured by angiocardiography, dilution techniques, radioisotope angiography and by echocardiography. These methods have limitations, and routine and frequent accurate determinations of LVEDV, under varying clinical situations, is not yet possible, although echocardiography holds promise. For these reasons, LVEDP continues to be a frequently utilized parameter in the assessment of ventricular performance. In patients with LV dysfunction, if ventricular filling pressure is utilized to assess LV performance, it is better to use LVDP rather than an indirect estimate of LVEDP for the following reasons: 1) by using an indirect estimate of LVEDP the contribution of atrial contraction to EDP and EDV is ignored because the indirect estimates of LVEDP are often the same as LVDP pre "a. The importance of atrial systole to ventricular function has been known for a long time and myocardiab fiber length after atrial contraction is known to influence ventricular contraction. While admittedly atrial contraction may result in a greater increase of EDP than EDV, the influence of atrial systole on LV fiber length is important in many of these patients because an appropriately timed atrial contraction contributes significantly to LV stroke volume which averages 25-39 percent. 2) It has not yet been demonstrated that LVDP other than LVEDP influences ventricular performance.

In conclusion, it is important that data from studies performed by various investigators be utilized maximally. Use of an accurate and standard terminology is crucial. The term left ventricular end-diastolic pressure should be restricted to LVDP at the "z" point, that is, after atrial contraction and left ventricular diastolic pressure prior to atrial contraction be referred to as LVDP pre "a. In addition, when presenting data in patients with left ventricular dysfunction, it would seem appropriate not to use the general term left ventricular filling pressure, but to mention the specific parameter that was...
measured. If ventricular filling pressure is used in the assessment of left ventricular function in patients with LV disease, it is preferable to use LVEDP rather than an indirect estimate of LVDP. Pulmonary artery EDP, mean left atrial pressure and LVDP pre "a" are good indicators of pulmonary venous pressure and help to assess the risk to the patient of pulmonary edema.

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An ACCP Compilation of Clinical Conferences in Pulmonary Disease:
A Foreword

The Department, Clinical Conference in Pulmonary Disease, has been enthusiastically endorsed by the readers of CHEST. A compilation of contributions to this section has been prepared as a special ACCP publication. The Department Editor, Dr. Baum, prepared a sprightly foreword for the manual and this foreword is presented in these editorial pages, as well as in the compilation.

The Editor

Clinical Conferences in Pulmonary Disease has been a department of Chest for five years. The premise upon which this presence is based is that physicians learn most about medicine from patients. This is my feeling whether I am in the hospital, the clinic, on a junket as visiting potentate or sitting quietly in the late hours of the evening being seduced by a provocative title in my favorite medical journal. I acknowledge that this was also...