Importance of Elevated Jugular Venous Pressure and a Third Heart Sound in Asymptomatic Left Ventricular Dysfunction

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In this issue of the *Journal,* Drazner et al. (1) sought to determine whether elevated jugular venous pressure and a third heart sound (S_3) in patients with asymptomatic left ventricular dysfunction predicted the development of overt heart failure. Jugular venous pulse and S_3, which are established physical signs (2), and "asymptomatic," a judgment derived from the medical history, were pitted against left ventricular dysfunction (ejection fraction $\leq$0.35), a quantifiable technology-based measurement (3).

Sophisticated laboratory methods now provide clinicians with remarkable diagnostic information, but their use may also de-emphasize the bedside examination upon which previous generations of clinicians were so much more dependent and therefore more adept. In the Drazner study, the manner in which physical examinations were performed was unavailable; nonetheless, the authors rightly concerned themselves with the decline in physician skills, although noting that nonstandardized physical examinations probably reflect clinical practice and thus supporting the wide applicability of their findings. Hence, emphasis on the history and cardiovascular physical examination was not simply a symbolic gesture to the past, but a valid method of modern clinical investigation.

Samuel Gottlieb Vogel (1796) described the merits of the medical history (4):

> The physician must listen to what the sick person says patiently and attentively, no matter how confused, garbled, or jumbled that story might seem. He must then order and connect everything in his own mind by addressing to the patient appropriate questions that clarify what is still vague. . . . This procedure puts the physician in a very favorable and desirable light in the patient's eyes. The sufferer comes to believe that he can place all his trust in a man who has taken such great pains to comprehend his illness.

Can we assume that patients in the study who were classified as asymptomatic (New York Heart Association class I or II) based on their histories were indebted to Vogel's principles despite having left ventricular filling pressures sufficient to elevate pulmonary venous and pulmonary arterial pressures enough to increase jugular venous pressure?

Similarly, Sir James Mackenzie, while focusing on the diagnosis of heart affections in 1916, described the benefits of the physical examination, which includes the reassuring physical contact between physician and patient—the laying on of the hands: "I hope to demonstrate to you that knowledge essential to the understanding of the patient's complaint can be made out by our unaided senses, and that artificial and mechanical aids can, in the nature of things, only be accessory aids" (5). Perhaps Drazner et al.'s use of elevated jugular venous pressure and an S_3 as markers was also indebted to these principles.

In 1867, Pierre Carl Potain attributed the S_3 to sudden cessation of ventricular distension during the rapid filling phase of the cardiac cycle (6), a phase that is not a passive event in which inflow merely expands the recipient ventricle (1). Instead, ventricular distension (relaxation) is a complex, active, energy-dependent process. Drazner et al. correctly related the S_3 to ventricular dysfunction, stating that "[s]everal acoustical characteristics favor the audibility of an S_3, including increased chamber stiffness and filling pressures, and early diastolic filling rates." Also favoring audibility is the auscultatory technique used to elicit an S_3, which is a low-frequency event best heard in a quiet room when the bell of the stethoscope is electively applied over the left or right ventricular impulse with just enough pressure to form a skin seal (1).

Third heart sounds that originate within the left ventricular cavity are heard over the left ventricular impulse; those that originate within the right ventricular cavity are heard over the right ventricular impulse (2). The authors, however, did not distinguish a left from right ventricular S_3, thus it is not known whether the distinction might have affected the prognostic value of the S_3. However, it is reasonable to assign a left ventricular S_3 to ischemic left

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ventricular dysfunction, as it is to assign a coexisting right ventricular S_3 to the tall right atrial V wave and a rapid Y descent implicit in an elevation of jugular venous pressure.

The authors recognized that audibility of an S_3 is improved by maneuvers such as passive leg raising, isotonic exercise, and the abdominojugular reflux, all of which increase venous return (1). It is not known whether these maneuvers were employed in the study. Likewise, details regarding criteria for establishing an elevated jugular venous pressure were not provided, so assumptions regarding mean jugular venous pressure or the heights of A and V waves can only be inferred. This lack of detail, however, does not detract from the interesting and informative background of the jugular venous pulse as a physical sign.

The cervical venous pulse was first described in 1728 by Giovanni Maria Lancisi as "systolic fluctuation of the jugular vein in a patient who at necropsy had tricuspid regurgitation" (7). Rhythmic pulsations of the cervical veins were subsequently described in 1794 by John Hunter, who wrote: "The larger veins, near to the heart, have a pulsation which arises from the contraction of the heart preventing the entrance of blood at that time" (8). Almost a century elapsed before Chauveau and Marey published their classic graphic records of the wave forms (9), and in 1902 Mackenzie underscored the value of bedside examination of the jugular pulse (3). About 30 years later, Carl Wiggers published his landmark study of the pressure pulses in the cardiovascular system, stating that "[i]t became increasingly more obvious to many physiologists that records of the venous pulse might be of service in the interpretation of the dynamic events in the heart" (10). In the 1950s, Paul Hamilton Wood rekindled interest in the meticulous observation of the jugular venous pulse, emphasizing that the wave forms in the right internal jugular vein permitted relatively precise anatomic and hemodynamic inferences (2).

Proper examination of the jugular venous pulse requires the patient to be supine on a bed or examination table that permits adjustable elevation of the trunk above the horizontal to a level corresponding to the maximum visible oscillations of the right internal jugular vein (2). The higher the central venous pressure, the higher the required elevation of the trunk. With the patient's head in a neutral position to avoid tension on the right sternocleidomastoid muscle, and with the examiner's right thumb palpating the contralateral carotid pulse for timing purposes (3), a light source (e.g., a small pocket flashlight) is directed tangentially to highlight the fluctuating shadows cast by the right internal jugular vein. The valveless external jugular, which may not be visible unless mechanically distended by digital pressure at the root of the neck, is recognized as a static nonpulsatile column that provides an estimate of mean right atrial pressure. With the sternal angle of Louis as a convenient reference, the heights of the A and V waves and the height of the column of the external jugular vein above the sternal angle can be established. A simple rule of thumb is that the A and V crests and the crest of the external jugular vein are normally not more than 3 cm above the sternal angle, with the patient's trunk 30° above horizontal (2).

Braunwald wrote that "[i]ntelligent selection of investigative procedures from the ever-increasing array of tests now available requires far more sophisticated decision-making than was necessary when the choices were limited to electrocardiography and chest roentgenography. The clinical examination provides the critical information necessary for most of these decisions" (11). This point of view reflects Drazner et al.'s study, which convincingly demonstrated the association between an elevated jugular venous pressure and an S_3 and the subsequent development of overt heart failure in patients with asymptomatic left ventricular dysfunction.

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