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Defining, Quantifying, and Classifying Acute Renal Failure

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The syndrome of so-called "acute renal failure" (ARF) is common in the intensive care unit (ICU) and may affect from 1% to 25% of patients [1–6], depending on the population and the criteria that are used to define its presence. In many ways, its nature and epidemiology resemble those of other ICU syndromes, such as severe sepsis, septic shock [7], or acute respiratory distress syndrome (ARDS) [8]. Like any other clinical entity, it is an invention of man. There is no such thing as ARF like there is a table or a chair; there are only people who have malfunction of the kidney for several reasons.

Because it is an artificial concept, it can neither be proven nor denied that someone has ARF unless one agrees ahead of time on what the term means. It is a bit like saying that a person is Chinese. Unless one agrees on what Chinese means, one cannot say that a given person is or is not Chinese (born in China? has a People's Republic of China passport? has a particular phenotype?). Although these "philosophical" observations might seem to be futile, unnecessary, and even counterproductive to the clinician who "knows" what a patient who has ARF is "when he/she sees one," they might be important to the researcher. The researcher needs clear consensus definitions to describe and understand epidemiology, to randomize patients in controlled trials, to test therapies in similar groups of patients, to develop animal models, to validate diagnostic tests and so on, including subdividing patients into objective types of ARF. Furthermore, if research is to generate evidence to change clinical practice, practitioners will need to adopt definitions from studies to apply their results.

Definitions cannot be arbitrary (the researcher likes them but other researchers do not). They need to be based on at least some widely accepted foundations of

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physiology, clinical behavior, response to treatment, histopathologic features, and prognosis. They need to be the same worldwide: a concept that moves the issue of definition from the scientific or clinical world to the political (''my definition is better than yours syndrome'').

In this regard, ARF is no different from ARDS, systemic inflammatory response syndrome, severe sepsis, or septic shock [7,8]. All of these syndromes have no gold standards of diagnosis, no specific histopathologic confirmation, and no uniform clinical behavior, just like ARF, because they do not ''exist.''

What exists are patients who have a spectrum of conditions that we choose to lump together for operative purposes until further information emerges that allows us to be more specific and to create other useful artificial entities. Yet, these imperfect, highly flawed, man-made definitions are useful. This usefulness is demonstrated nicely by the ability to conduct trials in patients who have sepsis or ARDS that would be impossible without definitions.

Thus, patients can and should be lumped together as having ''ARF'' for operative purposes. Such ''lumping'' of patients is done to facilitate the diagnostic process, clinical assessment, and, hopefully, therapeutic intervention. Once this cognitive process is accepted as useful, then it becomes necessary to define what ARF actually is and when someone does or does not have ARF. After a definition is provided, it also becomes necessary to quantify and classify the entity that is created by such a definition for the same utilitarian principles of diagnostic and therapeutic usefulness.

In this article, these issues are discussed in detail with a particular focus on the critically ill patient.

Approaching the issue of definition

A logical approach to organ ''failure'' is to start by defining what an organ does. In the case of the kidney, the list is long; however, many of its functions are shared with other organs (eg, acid-base control with lung) or require complex neuro-hormonal interactions, which also involve other organs (renin–angiotensin–aldosterone or vitamin D–calcium–calcitonin–parathyroid hormone axis). Other functions, which are not shared, are not measured routinely (small peptide excretion, tubular metabolism, hormonal production) in the ICU.

Accordingly, the kidney may ''fail'' in several of these tasks (eg, erythropoietin release may fail before changes in glomerular filtration rate [GFR] are detected); however, the clinician may be unable to detect such failure because of lack of measurement or because of compensation by other organs. Thus, the intensivist is left with limited information to help him/her assess renal function in the critically ill patient.

Only two ''functions'' that are ''unique'' to the kidney are measured routinely and easily in the ICU: the excretion of water-soluble waste products of nitrogen metabolism (of which we routinely measure only urea and creatinine) and the production of urine. Thus, clinicians have focused on these two aspects of renal