Anesthesiology
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Allen's Test—Positive or Negative?

To the Editor:—Edgar V. Allen, M.D., in 1929, first described "the compression test" for localizing occlusion of arterial blood flow to the palmar arch of the hand in patients with thromboangiitis obliterans.[1] This test has been named after Allen and is applied routinely on each patient in our hospital prior to percutaneous arterial puncture.

Confusion arises, however, in what constitutes a positive or a negative Allen's test. Reports in the literature are contradictory, and these can lead to problems in documenting the results of this proper test.

Shapiro described Allen's test as positive when the ulnar artery adequately supplies the entire hand.[2] Greenhow agrees in his description of a "false-negative" result as a delay in return of the arterial blush due to full extension of the patient's hand.[3] Abadir and Ung described this same delay in arterial blush as a "false-positive" result.[4]

Allen did not define his compression test as positive or negative. To do so can lead not only to miscommunication but also to poor documentation. To be more accurate, we believe an Allen's test should be described according to the length of delay in return of the arterial blush.

For example, the Allen's test was performed on the patient's right hand with a three-second delay. We consider a delay of five seconds or more to be abnormal.

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Averaging *p*H *vs.* H^+ Values, an Irrelevant Debate

To the Editor:—The lively correspondence provided by Giesecke[1] and Pace *et al.*[2] cannot, alas, continue forever. However, before editorial wisdom curtails this entertainment, you might perhaps allow a practical and statistical comment.

Before converting numbers to a statistic, such as a mean, one question is to be satisfied: "Why do it?" The answer must be "To provide the reader with useful information." The mean is the value around which the data are grouped, the standard deviation represents the scatter, and the standard error represents the accuracy of the mean. These values are useful when data are scattered with approximately a normal distribution. Giesecke creates five solutions of differing *p*Hs and mixes them; Pace *et al.* postulate mixing two such solutions. Neither example has a normal distribution and a mean is therefore unlikely to provide useful information; as both letters laboriously demonstrate, a mean *p*H will certainly not represent the *p*H of the mixture. If in practice any of us needed such information we would simply mix and measure!

Mean, standard deviation, etc., are appropriately applied to *p*H, or to H^+, when one, or the other, representation of the acidity has a reasonably normal distribution; healthy blood-gas values provide an example with a familiar mean *p*H of 7.4. However, gastric acidity shows no simple gaussian distribution, whether expressed as *p*H or as $[H^+]$. Indeed, consideration of the stomach's physiologic function permits us to anticipate high acidity during active hydrochloric acid formation and reduced acidity at other times. Representing such a complex distribution with a single mean is inappropriate.

Of clinical concern in the original paper was the

incidence of *p*H values less than 2.5. As summarized in Cohen's recent editorial,[3] this criterion is still used when identifying patients at risk. With "incidence of low *p*H" in mind both mean *p*H and mean H^+ appear irrelevant. The real question is "Does the therapeutic regimen significantly alter the *incidence* of patients 'at risk'?" In Stoelting's original paper,[4] nonparametric statistics (chi-square) were appropriately used and demonstrated a different incidence only when antacids were administered. His tables of mean acidity in the groups above and below *p*H 2.5 (whether mean *p*H or H^+) would thus appear peripheral to the primary concern. Their most useful purpose has probably been to stimulate this correspondence.

In conclusion, I would put in a plea to editors and writers. We cannot eliminate statistics, but let us at least use them sparingly and in the certain knowledge that they at least represent something.

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Averaging Acidity Values

To the Editor:—Reading the correspondence concerning acidity values between Giesecke[1] and Pace *et al.*[2] generated a certain sense of *déja vu*. This matter was fully ventilated in *Anaesthesia* in April 1978[3] and in the *British Medical Journal* in 1977.[4] Despite the claim to the contrary, neither *p*H nor hydrogen ion concentration is an independent variable. For the conventional *p*H electrode and meter assembly the true independent variable is the millivolt difference across the glass membrane between the silver–silver chloride reference electrode in the glass and the calomel electrode. From this millivolt scale both *p*H and hydrogen ion concentration can be derived. Any random errors of measurement for any sample will be normally distributed around the mean millivolt measurement of that sample, as multiple measurements of that sample will confirm. In hydrogen ion concentration terms this is a log-normal distribution.

Presumably the purpose of the debated measurements was to determine the effects of different premedicants upon the acid output of the stomach. Therefore, pooling the measurement data obtained from a number of samples to derive this biologic response, that is, the mean acid output or average quantity of hydrogen ions secreted by the gastric mucosa, requires the use of the arithmetic mean of these values; the quantity of hydrogen ions is the molar concentration of hydrogen ions per liter multiplied by the volume secreted.

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Central Venous Catheter Placement for Cardiopulmonary Bypass

To the Editor:—I was interested in the solution offered by Rasmussen and Husum for the problem encountered with central venous pressure (CVP) monitoring by pulmonary-artery catheter during total cardiopulmonary bypass.[1] In my experience, the design of the pulmonary-artery catheter places the