professionals continually reminds our patients of their inescapable, unsightly and unseemly bodily functions and dysfunctions. Our role is to help them accept and see beyond these limiting necessities to the possibilities that illness has temporarily obscured. Gentle humor can help bridge this gap. A physician using humor wisely shows both an awareness of the dual nature of humankind and a willingness to communicate in the nonscientific language of inference, symbolism and spirit. Frequently, a physician and patient can use this alternate path in addition to the hard, cold scientific facts and get a better sense of each other and of the problems and possibilities at hand. And, they can enjoy a lot of therapeutic laughter.

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## The Effect of Heparin Dilution on Arterial Blood Gas Analysis

TO THE EDITOR: Twenty years after Anderson[1] presented his findings on sampling and storing blood, confusion remains regarding the effects of heparin dilution on the accuracy of blood gas determinations. A commonly used text on blood gas analysis states that "if too much sodium heparin is used, it will affect the results to the acidotic side."[2] This concept is quite pervasive within medical teaching. Some studies state that excessive heparin affects the pH only minimally while arterial oxygen pressure ($Pa_{O_2}$) and carbon dioxide pressure ($Pa_{CO_2}$) may be altered more significantly[1,3]; however, the mechanism of these changes is not addressed. Clearly, there is not a wide clinical understanding of the effects of heparin overdilution on arterial blood gas measurements.

To determine what changes occur when blood is diluted with heparin or 0.9% sodium chloride solution (NS) and analyzed in the usual clinical manner we performed the following study.

### *Materials and Methods*

Heparin sodium (1,000 United States Pharmacopeia units per ml) was added to glass syringes in volumes of 0.125, 0.25, 0.5 and 1.0 ml. Arterial blood was drawn from a patient into the syringes to a total volume of 2.0 ml. The syringes were gently rolled and tilted manually to assure mixing, and analysis of pH, $Pa_{O_2}$ and $Pa_{CO_2}$ was done immediately on a calibrated Radiometer ABL-2 automated blood gas analyzer. The study was repeated with arterial blood from a second patient using syringes containing only enough heparin sodium to fill the needle and hub and using NS in the same concentrations as previously used with heparin.

### *Results and Discussion*

The results are summarized in Table 1. Upon dilution with both heparin and NS, the pH remains unchanged even in dilutions of 50%. The $Pa_{CO_2}$, however, falls dramatically while the $Pa_{O_2}$ rises.

In our practice, we often observe confusion on the part of staff physicians, house officers, nurses and respiratory therapists regarding the effects of heparin dilution on arterial blood gas analysis. Most believe excessive heparin will cause the pH to fall significantly but are puzzled by the possibility of an effect on $Pa_{O_2}$ or $Pa_{CO_2}$.

TABLE 1.—*The pH, Arterial Oxygen Pressure ($Pa_{O_2}$) and Arterial Carbon Dioxide Pressure ($Pa_{CO_2}$) of Arterial Blood Diluted With Heparin Sodium or 0.9% Sodium Chloride Solution (NS)*

| Solution | pH | $Pa_{O_2}$ mmHg | $Pa_{CO_2}$ mmHg |
|---|---|---|---|
| Heparin Sodium | 6.393 | 134.6 | 7.6 |
| 0.9% Sodium Chloride | 5.900 | 161.4 | 9.4 |
| | | *Percent of Initial Value* | |
| Heparin Dilution | | | |
| 6% Heparin | 100 | 99 | 95 |
| 12% Heparin | 100 | 105 | 92 |
| 25% Heparin | 100 | 111 | 65* |
| 50% Heparin | 100 | 130* | 38* |
| NS Dilution | | | |
| 6% NS | 100 | 100 | 98 |
| 12% NS | 100 | 100 | 91 |
| 25% NS | 100 | 105 | 70* |
| 50% NS | 100 | 112 | 49* |

*$P < .05$.

The changes we demonstrate follow well-established principles of physics and physiology.[4] The pH, for practical purposes, remains unchanged because of the vast buffering potential of oxyhemoglobin and plasma proteins; for example, for a hemoglobin concentration of 15 grams per dl, $1.62 \times 10^7$ nm of hydrogen are required to lower the pH from 7.40 to 7.15. The hydrogen ion concentration of the heparin used in this study was $4.00 \times 10^2$ nm per liter. Hence, it would take an enormous disproportion of heparin to significantly affect the pH, much more than even a gross clinical error.

Of more practical significance, however, are the dilutional effects on $Pa_{O_2}$ and $Pa_{CO_2}$. The effect on $Pa_{CO_2}$ may be of clinical significance at dilutions of 25% or greater. The effect on $Pa_{O_2}$ is less striking. These dilutional changes are understandable if one considers that the partial pressure of a gas in solution is proportional to the solubility coefficient of the gas and the partial pressure of the gas overlying the liquid. The $Pa_{O_2}$ and $Pa_{CO_2}$ of heparin or NS reflect the air/fluid boundary in the storage bottle. Hence, in obtaining an arterial blood sample one is mixing heparin with a relatively high $Pa_{O_2}$ and low $Pa_{CO_2}$ with arterial blood. As would be expected, the $Pa_{O_2}$ rises while the $Pa_{CO_2}$ drops sharply, both changes in proportion to the relative differences in partial pressure of these gases between blood and heparin.

In summary, when excessive quantities of heparin are added to blood, the primary effect on blood gas analysis is dilutional, and $Pa_{CO_2}$ is the measurement most profoundly affected. This change in $Pa_{CO_2}$ could lead to a misinterpretation of a patient's acid-base status. Additionally, it is conceivable that the rise in $Pa_{O_2}$ might lead to an erroneous conclusion regarding a patient's need for supplemental oxygen. Finally, for

clinical purposes, the pH remains unaffected, even when blood and heparin are added in equal amounts. This finding may also relate to pH determinations of empyema fluid. These observations should be helpful in interpreting arterial blood gas determinations, especially in situations where only small aliquots of blood are available for analysis or carelessness occurs in obtaining a sample.

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## Diagonal Earlobe Creases

TO THE EDITOR: The excellent work reported by Jorde, William and Hunt[1] found a lack of association between diagonal earlobe creases and other cardiovascular risk factors (blood pressure, smoking, weight, height, skinfold thickness, cholesterol, high-density lipoprotein, intracellular sodium, sodium-lithium countertransport and renin).

I would like to take this opportunity to comment on my own incidental observations made while conducting studies on weight gain secondary to amitriptyline maintenance in a hemodialysis population.[2] In two distinctive regards, observed earlobe creases were prominent: (1) the age of the patients was relatively young, the incidence of creases absolutely and relatively high and (2) the degree to which creases existed was great. Frequently the earlobes were divided in two with each half of the bilobe appearing spherical. Unfortunately, no simple quantitative method was devised for measuring the full range of changes observed; so a contemplated study was not carried out. This area remains open for other investigators.

The question of physiologic factors (for example, tissue fluid tides) and external mechanical factors (such as compression by a pillow while sleeping) comes to mind. Obviously anephric patients accumulate water, salt and waste products between dialyses. These are stored in the first, second and even third fluid spaces. With potassium levels high some measure of *intra*cellular as well as *extra*cellular edema could be expected.

A linear observation of my own earlobes made over a 12-year period showed them to be *un*creased at first, then later creased. The depths of my own diagonal creases were generally *not* stable, and the surrounding earlobe tissue composition likewise seemed to vary. When the substance of the lobes was engorged, the lines were deep and suggested cleavage. When dietary restriction and physical activity, on the other extreme, had led to "dehydration," the skin of the earlobes appeared loose, the diagonal lines little more prominent than many other lines that appeared locally; a deflated rubber balloon was suggested.

An additional observation made was that of easy *bruisability;* manipulation of earlobes, even though not severe, occasionally led to bruises. This was a variable finding sometimes suggested by the presence of preexisting discoloration. In my own case, bruisability came and went. The causes were not studied.

The suggestion to be found in all of these observations is that genetic, hormonal, dietary and mechanical factors all can play a role in the development of diagonal earlobe creases. The question is, do these same factors simultaneously influence the development of coronary artery disease?

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## Flexible Sigmoidoscopy—A Provocative Test?

TO THE EDITOR: We are all aware of the "chandelier sign" wherein a patient with pelvic inflammatory disease leaps toward the ceiling during manipulation of the cervix on pelvic examination. We have observed a less dramatic but still impressive response during flexible sigmoidoscopy of patients sent to us with unexplained abdominal discomfort or with a tentative diagnosis of irritable bowel syndrome. Upon manipulation of the instrument or air insufflation, these patients also writhe and contort with pain. When specifically asked if this is the identical discomfort that brought them to us, or whether it is an entirely different sensation, 29 of 41 patients (70.73%) said it was identical.

We suggest that when this phenomenon is observed in the presence of a negative workup, this sign be taken as evidence for irritable bowel syndrome. We feel that flexible sigmoidoscopy should be considered not only a useful tool to exclude other diseases of the distal colon but also a provocative test in this setting because irritable bowel syndrome is primarily a diagnosis of exclusion. Additionally, a positive response is reassuring to both patient and physician.

On the other hand, lack of this sign could be produced by a spastic component proximal to the splenic flexure. Colonic spasm is frequently seen in sigmoidoscopy and appears to have no correlation with production of pain.

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