LETTERS AND COMMENTS

DANGEROUS ARRHYTHMIAS

To the Editor: The case report of Drs. Harrington and Desanctis (Ann. Intern. Med. 70: 105, 1969) describing "Hiccup-Induced Atrioventricular Block" was indeed interesting but not surprising for those of us who have established the routine use of various inspiratory maneuvers in our heart stations as part of the regular electrocardiogram (1).

We continue to be impressed by the great variety of arrhythmias that are brought out by the simple act of taking a deep breath and holding it for a short period. The use of inspiratory leads in routine electrocardiography has had its greatest practical use in bringing to our attention potentially hazardous arrhythmias. If simple singultus and simple deep breathing can be associated with potentially fatal arrhythmias, imagine what is happening at home with coughing, sneezing, micturition, and straining at stool! It therefore seems all the more imperative that the "bushes be shaken" for possibly dangerous arrhythmias in the safe confines of the hospital.

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REFERENCE

ANTI-CANDIDAL AGGLUTINATING ANTIBODY

To the Editor: Drs. Preisler, Hasenclever, Levitan, and Henderson (Ann. Intern. Med. 70: 19, 1969) report a significant rise in agglutinating antibody titer in 14 of 23 patients with acute leukemia who had visceral candidiasis at autopsy. They felt that there was no statistically significant difference in their patients with regard to age, sex, variety, or duration of leukemia, year of diagnosis, or length of time on study. They also felt that the incidence of leukopenia, granulocytopenia, and lymphopenia was similar.

Since this is a population of patients who require frequent transfusions of blood or blood products, the possible role of passive transfer of antibody becomes important. I note that the authors fail to present transfusion data in their study and wonder if an analysis of such might truly implicate passive transfer as a factor.

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[Dr. Slater's letter was referred to the authors, who reply:]

To the Editor: Dr. Lewis Slater raises an interesting question concerning passive transfer of antibodies by blood transfusion. Such a mechanism has been shown to result in elevated titers of antitoxoplasm antibodies in the same group of acute leukemic patients (Vogel and Lunde: Cancer, in press, April 1969). However, the incidence of significant levels of anti-candidal antibodies as defined in the article is quite small in the adult population.

In a study currently in progress anti-candidal agglutinating antibody levels have been repetitively measured in 154 patients with acute leukemia. The average patient on the National Cancer Institute's Leukemia Service receives 20 to 25 units of whole blood or blood fractions per year, so that the study group had a significant chance of "passive immunization." To date no patient has developed a significant rise (a two dilution rise to 1:80 or greater) in anti-candidal agglutinins, who did not have either culturably or histologically demonstrated, or both, visceral candidiasis or a prolonged febrile illness unresponsive to antibacterial antibiotics.

Patients with disseminated bacterial infections of comparable severity and of similar status in terms of bone marrow hypofunction, peripheral blood cytopenia, chemotherapy, and transfusion requirements did not exhibit rising agglutinating antibody titers.

Since donor blood was not screened in all cases one cannot exclude passive anti-candida antibody transfer. We believe that our ongoing experience makes this possibility quite unlikely.

To date, for whatever reason, the anti-candida agglutination test has provided the most reliable and sensitive laboratory diagnostic tool.
for the antemortem diagnosis of visceral candidiasis.

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ISOPOTENTIAL SURFACE MAPS

To the Editor: Several questions may be raised regarding the data and conclusions of Spach, Barr, Blumenschein, and Boineau in "Clinical Implications of Isopotential Surface Maps" (Ann. Intern. Med. 69: 919, 1968).

They purport to show the advantage of isopotential mapping over electrocardiography and vectorcardiography, but they fail to show any electrocardiograms for comparison and only one vectorcardiogram.

Although the two single QRS complexes in Figure 1 were similar, they were not identical; the complex in the case of the right ventricular hypertrophy (RVH) had an R:S ratio of nearly 1, one of the electrocardiographic criteria for RVH (1). Additional leads might have also suggested the presence of RVH.

In the second case of RVH (Figure 2), it is difficult to see how a maximal spatial vector that is directed to the right and anteriorly can inscribe a negative deflection at that time in Vi. Here again, the R:S ratio in Vi is suggestive of RVH.

Although there was some resemblance between the vectorcardiogram in the case of atrial septal defect (Figure 4) and a normal vectorcardiogram (Figure 3), the former showed increased anterior forces together with a large terminal deflection to the right and posteriorly, one of the vectorcardiogram patterns of RVH (2). Reconstruction of the electrocardiogram from the vectorcardiogram should show an rSr' pattern in Vi, which would suggest an atrial septal defect.

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REFERENCES


[To Dr. Weinstock's letter, Dr. Spach and his associates reply.]

To the Editor: Dr. Weinstock's comments concerning the specific data presented are quite appropriate. With respect to his comments, the criteria for right ventricular hypertrophy (RVH) when there is an R:S ratio of nearly 1 does not hold in children, where our experience has been focused. Thus, the work of Sokolow and Lyon does not apply directly to children under 10 years. We have seen patients where selected precordial leads, as presented in Figure 1, did not discriminate mild RVH from normal. The point of Figure 1, however, was merely to illustrate how an individual lead may inscribe somewhat similar curves in the presence of different sequences of ventricular excitation.

With respect to Dr. Weinstock's comment regarding Figure 2, the arrow showing heart excitation pointing to the right and anterior while a negative deflection occurs simultaneously in lead V1 is related to the right lateral chest being enveloped by positive potentials while the area of lead V1 is occupied by a minimum.

Dr. Weinstock's comment that the anterior initial forces are increased for the vector in Figure 4 may be appropriate for adults. On the other hand, the specific patient's data that were illustrated have been included in a series of Frank vectorcardiograms that have been analyzed with an approach similar to Dr. Pipberger's with quantitative computer measurements. The spatial vectors during the first four tenths of QRS were within 1 standard deviation of a group of normal children. Also, the rSr' configuration was not present in lead V1 for this patient, although there was a secundum atrial defect proved at surgery.

We agree with Dr. Weinstock that distinguishing RVH may usually be done with regular lead systems and particularly by the use of additional leads (such as those available in the surface map). However, if nothing else, the surface maps improve our understanding of the electrocardiogram from a more physiologic rather than empiric basis and provide evidence that by using the maps it is possible to distinguish not only the presence of RVH but discriminate as to the type of hypertrophy (Circulation 38: 917, 1968). Such information should be helpful both in better understanding clinical electrocardiograms and in developing clinically applicable and improved lead systems and methods of data presentation.

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FLUOROSIS, FLUID FLUORIDATION, AND FOOD FLUORIDE

To the Editor: In preparing for an educational program on the safety of fluoridation of public water systems as a method of preventing dental caries, I came across a report appearing in the ANNALS (1) which is being used as the basis for an argument against fluoridation of drinking water (2). The authors describe a man with fluorotic radiculomyelopathy, which is usually due to the daily ingestion of 20 to 80 mg of fluoride. The patient had experienced 30 years of polydipsia (4 to 10 liters per day) and had been exposed to water containing between 2.2 and 3.5 ppm fluoride. His diet was said to be unremarkable. His clinical, X-ray, and autopsy findings, without doubt, confirmed severe fluorosis. The authors concluded, "The development of advanced fluorosis in this patient exposed to drinking water with less than 4 ppm of fluoride was unusual and was probably a consequence of his excessive water intake. Prolonged polydipsia may be hazardous to persons who live in areas where the levels of fluoride in drinking water are not those usually associated with significant fluorosis." Although the patient's daily fluoride consumption could have been in the range of 20 mg from water alone, his diet was, in fact, not unremarkable. A sister who had lived with him for 35 years confirmed the polydipsia but added that he "also drank a considerable amount of tea."

Depending on the brand and quality, tea can contain nearly 400 ppm fluoride; an average value is 97 ppm (3). Instant tea may contain more; Standard Brands Instant has 170 ppm (4). Directions for making instant tea prescribe 2 tbsp/qt with more or less added according to taste. Two tablespoons of instant tea weigh 13 g and would contain 2.2 mg fluoride in this case. The amount of additional fluoride this patient received from the "considerable amount of tea" is conjectural. Thus, the authors' conclusion that drinking water alone was responsible for fluorosis seems unjustified under the circumstances.

There are several other major sources of fluoride in the diet which should be mentioned:

<table>
<thead>
<tr>
<th>Food</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Some baking powders</td>
<td>2.2 mg/10 g (3)</td>
</tr>
<tr>
<td>Some wines</td>
<td>6.3 mg/liter (3)</td>
</tr>
<tr>
<td>Fresh mackerel</td>
<td>2.7 mg/100 g (3)</td>
</tr>
<tr>
<td>Dried salmon</td>
<td>1.9 mg/100 g (3)</td>
</tr>
<tr>
<td>Unwashed apples (sprayed with fluoride-containing insecticides)</td>
<td>2.1 mg to 5.7 mg/100 g (5)</td>
</tr>
</tbody>
</table>

At least one author may have fallen into the food fluoride trap. Young (6) failed to take this into account in comparing the incidence of dental caries between American and Italian children. The Italians had fewer cavities and ate more fruit. The author concluded: "A possible protective action of fruit, which may be in part related to the Italian practice of finishing the meal with this food, was observed." He failed to mention if the fruit the Italians were eating had been sprayed with fluoride-containing insecticides.

There is little question that water containing 1 ppm fluoride, which is used to make dialysate, is a real hazard for chronic hemodialysis patients. Plasma fluoride levels rise above normal in these persons (7), and considerable bone storage may take place since kidneys are the major route of excretion of the ion.

The major point of this brief discussion is that some common foods do contain surprisingly large quantities of fluoride and that patients with polydipsia (for whatever reason) and patients undergoing chronic dialysis should be warned about the dangers of fluorosis from these sources. A second point is that such articles, as the one cited initially (1), cannot be used legitimately as an argument against fluoridation of public water systems. Patients with severe polydipsia and those undergoing hemodialysis are usually under the direct care of physicians whose business it is to protect their patients from common substances in the environment which might be unusually hazardous to them because of special medical circumstances.

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REFERENCES
2. Zanfagna, P. E.: Medical aspects of fluoridation; three deaths in fluoridation, in First National Symposium of Fluoridation, sponsored by The National Health Federation, Foothill Printers, Monrovia, Calif., 1966, p. 56.


**Racial Discrimination in the Annals?**

*To the Editor:* I enjoy reading the *Annals of Internal Medicine* for medical information but not for racial identification of patients chiefly of Afro-American heritage.

The article “Rheumatoid Arthritis Terminating in Heavy-Chain Disease” (*Ann. Intern. Med.* 70: 335, 1969) indicates in the summary “a 51-year-old negro man.” Obviously, the proofreader should be made aware that racial tags are offensive and should be corrected as readily as other terms of bad taste.

How about the racial tag in Question 100 in Book A of the Self-Assessment Program?

The use of the racial identification as “negro” in the case study by Dr. Robert B. Scott in the July 1968 issue of the *Annals* had no medical significance and was useless information for the reader. Spelling Negro with a small “n” reflects an endemic practice of the south and again poor proofreading by the editorial staff.

We would appreciate your help in abolishing this discriminatory practice.

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*[For editorial comment on this letter see p. 1041—Ed.]*