Letters to the Editor
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More About Nurse Clinicians

To the editor:
Our article (Vol. 18, No. 3, Fall, 1970) describing the Health Nurse Clinician was not intended to offer a solution to the problem of rising health care costs. The increasing use of para-medical personnel certainly has an impact on cost, and nurses have led the way in delegating duties to others with less professional training.

In Vol. 19, No. 1, Letter to the Editor, Mrs. Zonca points to the law of supply and demand as fundamental to rising costs. The extension of the physician's capability in patient care, in a collegial relationship with the Health Nurse Clinician, strongly suggests a possibility of lowering costs.

I do not agree that "No matter what we do, hospital cost will continue to increase." The fact is what we do is the only thing that can influence the cost.

C. E. Rupe, M.D.

Comments on Australia Antigen

To the editor:
In reference to a recent article by Hayashi and LoGrippo (Vol. 19, No. 1, 1971), the authors found Australia antigen (Au) in 34% of institutionalized patients during a hepatitis epidemic which on all clinical grounds appeared to be "infectious" hepatitis. They concluded: (1) that Au is associated with infectious as well as serum hepatitis and (2) that the test for Au was relatively insensitive since it was negative in 66% of the hepatitis cases.

There would appear to be a third and more reasonable interpretation of these data, namely: (a) that, as in all other large institutions, there was a high frequency of endemic anicteric hepatitis much of which was Au positive (The frequency of Au in Down's and non-Down's patients cited is wholly consistent with that found in other institutions where no such infectious hepatitis outbreak has occurred); (b) that the hepatitis outbreak was indeed Au negative infectious hepatitis and that the authors merely measured pre-existing Au antigen levels.

The data are now fairly convincing that Au is associated with only one
form of the hepatitis virus. This is based primarily on the distinctions between MS-1 and MS-2 viruses isolated at the Willowbrook State School and on the repeated failure to demonstrate the appearance of Au in relation to any point source hepatitis epidemic. One can not contradict this accumulated evidence with data derived from institutions for the mentally retarded where the incidence of Au has consistently been shown to be high and particularly from an institution where no pre-epidemic samples were available for Au testing. Furthermore the authors present no control data on the incidence of Au, CRP, IgM and SGPT in their institutionalized patients who did not have clinical hepatitis. These data are crucial if one is to attempt to causally relate the presence of Au with the epidemic described.

In summary, without pre-epidemic sera for Au determination and without testing “non-hepatitis” controls, the authors are unjustified in drawing any conclusions regarding the relationship of Au to the hepatitis epidemic reported.

Harvey J. Alter, M.D.,
Senior Investigator
Paul V. Holland, M.D.,
Assistant Chief
Blood Bank Department-Clinical Center, National Institutes of Health, Bethesda, Md.

Authors’ Reply

To the editor:

We accept Drs. Alter and Holland’s third interpretation of our data (par. 2b of their letter). Their elaboration of our discussion is welcomed because it must not have been clear in our interpretation where we stated, “. . . we questioned whether our outbreak was IH or SH in nature. If IH and SH varieties were both present among patients, it would be difficult to explain in a relatively new institution the mixed hepatitis varieties present with abrupt onset and sudden termination in four to six weeks” (par. 1, page 32 of our publication).

Although we do not have data on the incidence of Au for 1962 among the institutional personnel, nor of the children before they developed hepatitis, the fact remains that testing for Au in clinically ill patients does not differentiate SH from IH. In addition, Au-antigen positive patients do not convincingly develop Au-antibodies following convalescence. More sensitive radioactive methods are being investigated for this purpose. However, until a more sensitive serologic method for demonstrating serum conversion from negative to positive antibody response to Au from active infection, and not from parental administration of homologous blood products, the association of Au to hepatitis virus protein warrants clarification from a virologic and immunologic standpoint.

Drs. Alter and Holland state that we have no crucial control study on the incidence of Au, CRP, IgM and SGPT values. Normal serum levels for these factors are given under “Materials and Methods” in our publica-
tion, with suitable references to our previous publications. We are not claiming that the presence of Au in 34% of the IH patients in institutionalized children and 23% (57 of 244 patients) admitted to our hospital with active virus hepatitis should be considered a “causal relation to the presence” (as they put it) of Au in our studies. We suggested that “These tests appear to reflect the host’s responses to nonspecific inflammatory conditions” (par. 1, page 31).

Since there is no serologic test for IH, as there is for Au which is hypothetically assumed to be associated with SH, how can one differentiate IH from SH in acute hepatitis cases by testing for Au only? This is particularly so if we assume, as they suggest, that Au in our study is present as pre-existing Au antigen levels in the presence of clinically active IH. Clinicians should not conclude that an active case of virus hepatitis is SH simply because the serum is Au-positive. This is an ever-present situation in clinical hepatitis and the purpose for our conclusion. We stand firm on our conclusion that more virologic and immunologic evidence is necessary before this moot question can be resolved.

G. A. LoGrippo, M.D.
H. Hayashi, Ph.D.