CROSS-REACTION OF SCHWARZ/MANN DIGOXIN ANTIBODY WITH A NORMAL CONTROL SERUM

We would like to report on the underestimation of digoxin serum levels that can result from the use of the ^{125}I-Digoxin radioimmunoassay kit (Schwarz/Mann) in combination with Moni-trol I (Lot # LTD 120B) manufactured by Dade (Division of the American Hospital Corporation). Moni-trol I is often used as the digoxin-free serum required in the preparation of the standard curve and control tubes, i.e., tubes containing no antibody and/or no "cold" digoxin standard.

We have found that the S/M digoxin antibody (Lot # YN 6208) cross-reacts with something in this particular lot of Monti-trol I, perhaps another serum steroid. This results in a displacement of ^{125}I-digoxin from the antibody and will produce a digoxin serum level in the therapeutic range. Other digoxin-free serum samples assayed at the same time did not show this displacement.

If Moni-trol I is used in the preparation of the standard curve, this will result in an *underestimation* of digoxin serum levels. Both the 100% tubes (antibody + ^{125}I-digoxin, no cold digoxin) and the standard curve tubes contain spuriously lower binding of ^{125}I-digoxin to antibody due to antibody cross-reaction with some substance in the Moni-trol I serum. Thus, patient serum sample compared with lower percent binding of standards leads to an underestimation of the digoxin serum level.

Moni-trol I was checked in two other assay systems (^{3}H-digoxin kit from New England Nuclear and ^{125}I-digoxin system using antibody developed at WHMC), and no displacement of tracer was noted in either procedure. Therefore, it is concluded that it is the S/M antibody cross-reacting with some substance in the Moni-trol I that is producing the false digoxin level.

Moni-trol I (Lot # LTD 120B) was assayed in triplicate in three different S/M digoxin assays by two different technicians and a "digoxin" serum value of 0.51 ± 0.04 (s.e.m.) was obtained. This interference was detected due to digoxin-free serum samples producing large negative digoxin levels, i.e., percent of antibody bound to digoxin in the serum sample was much greater than 100%.

This points out the need for assaying and checking for cross-reactivity of normal control serums before their use in radioimmunoassay procedures even though it is thought to be hormone- or drug-free and/or the antibody is thought not to cross-react with other serum steroids.

MARY L. BROWN
THERIAN HANNAH
GLADYS STEPHENS
Wilford Hall USAF Medical Center
Lackland AFB, Texas

RADIONUCLIDE CISTERNOGRAPHY—PREDICTION OF CLINICAL RESULTS OF NEUROSURGICAL SHUNTING IN PATIENTS WITH COMMUNICATING NORMAL PRESSURE HYDROCEPHALUS—FACT OR FANTASY

The concept of a correctable form of communicating hydrocephalus associated with normal cerebrospinal fluid pressure and certain neurological findings has been considered as a clinical entity since the work of Foltz and Ward, 1956 (*1*) and Raymond Adams and his associates in 1965 (*2*). Since that time there has been more than an abiding interest in this syndrome among clinicians after evidence was presented that improvement in the clinical status of these patients could be achieved by neurosurgical shunting. In the past several years radionuclide cisternography has been offered as one of the modalities that could be used in helping to evaluate this condition and assist in the selection of those patients most likely to benefit from shunting.

The classical criteria for considering a radionuclide cisternogram compatible with communicating hydrocephalus has been penetration of the radiopharmaceutical into the ventricular system with delayed or absent migration of the tracer around the cerebral hemispheres (*3*). Delayed absorption of the radiopharmaceutical from the ventricles or from the cerebral subarachnoid spaces has been reported as being characteristic in the more "positive" cases. Much controversy has existed concerning the significance of "early penetration" of the tracer into the ventricles and its subsequent rapid disappearance. Similarly, a "slow rate" of migration of the tracer around the cerebral hemispheres has been considered to be compatible with cortical atrophy but there appears to be a difference in the rate of migration with various tracers, possibly depending upon their molecular characteristics, leading to the difficulty in determining what the "normal rate" is.

Correlative studies between the findings on cisternography and the results of shunting have not