grams, which had shown progress of aortic stenosis and coronary occlusive lesions in spite of diet and drugs during 1968-1972, showed marked regression of the xanthomas in the aortic cusps and coronaries. This confirms the reversal of occlusive arterial disease in the legs (Zelis et al.: J Clin Invest 49: 1007, 1970), and innumerable observations of reversal in animals. suppl. III of Circulation (50: 1974) had four such reports: 361, 362, 364 and 1018.

All these reports, and my own unpublished studies of coronary arterial disease in young soldiers, confirm the view that the myosin-containing lipophages in the intima and media are not causing the lesions but are part of the normal defense against deposition of lipid percolating through the intima, in sucklings, in older animals, and in people with episodes of hyperlipidemia. These cells bring about reversal of xanthomas in skin, tendons, and arteries when hyperlipidemia is controlled. The work of Starzl et al. shows that in Type II hyperlipoproteinemia reversal occurs at cholesterol levels well above these at which deposition can increase in people with hypertriglyceridemia and chylomicronemia. Thus the importance of these macrophages and droplets, low in cholesterol, is clearly demonstrated.

It is to be hoped that the myosin-containing intimal cells, so meticulously studied by the pathologists at the University of Washington, will be further examined as to their sources and lipolytic enzymes. Presumably our grossly visible lesions result from overwhelming this defense mechanism, much as the tubular lesions of nephrosis develop when the cells are overwhelmed by the amount of lipoprotein in the glomerular filtrate. As of late 1974 there is overwhelming evidence that fatty streaks and fatal occlusive disease in our arteries are not "superficially similar" but identical with xanthomas of the arteries evoked experimentally in mammals and birds. All of these are reversible when plasma lipids are reduced, just as xanthomas in the skin or tendons. While there are innumerable aggravating factors — permeability alterations due to many causes, elevations of arterial pressure, the response of our minds to normal environments — none of these, alone or in combination, cause intimal disease in animals or men with plasma lipids normal for each species.

The large coronary arteries have far more smooth muscle cells in their intima than any other arteries in the body. This explains why lipids accumulate most rapidly in these arteries when lipids in the plasma are too high, and why reversal can occur so rapidly when the hyperlipidemia is controlled, by diet, drugs, or, in Type II hypercholesterolemia, by portocaval shunt.

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Electrophysiological Studies in Corrected Transposition

To the Editor:

In an otherwise informative paper concerning the conducting tissue in ventricular inversion, Anderson et al. 1 made these statements with which this correspondent must disagree: 1) They speculate that "... the bundle in those cases makes it ... inaccessible for the recording of His bundle potentials." 2) They further state that "... the recording catheter ... would be in contact only with the anterior node and anterior perforating bundle ... and recording the His potential would require a catheter position more laterally placed than is usually attainable." 3) They also warn that the "susceptibility of the conducting tissue to damage in congenital corrected transposition makes such procedures hazardous."

In response to each of these statements: 1) Two published studies2,3 demonstrated the ease of His bundle recording in seven patients with this anomaly. Since our paper was published an additional eight recordings of His bundle potential have been obtained in patients with ventricular inversion. 2) Positioning the electrode catheter via the right atrium into the right-sided (morphologic left) ventricle permits contact with the proximal anterior bundle, a desirable site for His bundle recording. 3) Intracardiac electrophysiology in patients with ventricular inversion proves helpful in investigation of the arrhythmias which are common in this malformation. Longer follow-up of the conduction abnormalities is necessary before it can be determined if they are of prognostic significance.

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References


The authors reply:

We thank Dr. Gillette for his comments on our paper on classically corrected transposition. We wonder whether we may not be disagreeing over definitions. When we stated that the anterior atrioventricular bundle was inaccessible for catheter recordings, we were referring to that portion which extended from the anulus fibrosus to the bifurcation. As we demonstrated, this part of the bundle is related to the antero-lateral quadrants of the pulmonary outflow tract, and we continue to contend that this part of the bundle would not be accessible unless a catheter was maneuvered into the pulmonary outflow tract. Dr. Gillette has clearly demonstrated that potentials can be recorded from the proximal portion of the connecting atrioventricular system in these patients. However, the precise morphological site of these recordings must be unknown, and the potentials could originate anywhere within the anterior node or the penetrating portion of the bundle. Solely on morphological grounds, we consider it most unlikely that they could be recorded from the nonbranching portion of the bundle, which in the hearts we studied was of an extensive structure.

Our statements regarding hazards of recording were based upon the assumption that it would be desirable to record from this nonbranching portion of the bundle, and to perform this procedure it would be necessary to pass the catheter into the pulmonary outflow tract, introducing the possibility of producing direct trauma to the susceptible connecting bundle. It may also be pertinent that the eldest patient in our series was a symptomless man who died following catheter studies to investigate conduction distur-
Letter: Electrophysiological studies in corrected transposition.
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