Total prosthetic replacement of atroioventricular valves in the dog

Part III. Local tissue reactions in the heart caused by tricuspid valve prostheses

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The metal parts of cardiac valve prostheses cause reactions in the surrounding tissues of the heart. In some dogs rather abnormal reactions were found, and were attributed to corrosion of the stainless steel in the cage of the valve with subsequent local metallosis of the heart and microscopic symptoms of corrosion disease.

Cardiac valve prostheses in current use consist of two distinct parts; one is a stabilizing structure and the other is mobile and performs the actual function of the valve. Either may become defective. This may be due to mechanical problems or to biological reactions in the surrounding tissues. Sometimes these reactions cause clinical failure and this is particularly true of the damage to blood cells and proteins caused by the mobile part, resulting in the formation of thrombi and/or fibrin deposits within the cage.

The reactions in the heart itself are mainly caused by the stabilizing part of the prosthesis, that is, by the cage. This article concerns these reactions as seen and studied in dogs after a survival time of at least two years after implantation of the free-floating cone valve described in a previous report (den Otter, 1968). The device has a cage with three rings, the upper or atrial ones forming a double ring, which holds the sutures (Fig. 1). To facilitate suturing, a Teflon rim may be inserted between these atrial rings, but we did not do this in the cases discussed in this paper; the metal was in direct contact with the heart tissue. The metal used for these prostheses was stainless steel AISI 316. Besides iron it contains 17% chromium, 9% nickel, and 2.5% molybdenum. Inside the cage is a lucite or silicon rubber cone with a central air-chamber.

In a series of 45 consecutive tricuspid prostheses, in which all the animals were on continuous anticoagulant therapy, there were 11 early failures due to clots or fibrin deposits, nine early and late failures through mechanical trouble, and 25 successful cases with a survival time of more than two years.

Of the survivors, 19 showed normal healing. This means that the double ring on the atrial side became covered by thick fibrous tissue within one month, although the covering might have been incomplete and remained so. On the ventri-

Fig. 1. Free-floating cone valve used in this study.

1American Iron and Steel Institute Specification
cular side there was no reaction at all around the cage provided it stayed free from the septal wall. Gradually the covering on the atrial side thinned out and left a narrow and incomplete fibrotic layer on top of the upper ring but a closely fitting fibrous mass in between the two atrial rings. Sometimes scar tissue was formed in the insertion area with some fibrous strands in the ventricle. This was the situation after two years or more.

Microscopy of these normally reacting cases showed the impressions of the upper and lower atrial rings and the tissue reaction in between: the endocardium was thick, with several layers of cells deep to which there was a large area of fibrosis and formation of cartilaginous tissue, sometimes even bone (Fig. 2). All these reactions are normal and serve a purpose in keeping the valve in place and maintaining its normal function for years. The dog’s heart has a natural tendency to the formation of cartilage and bone, especially in scar tissue.

In six long-term surviving animals the local reactions were abnormal and similar in all cases. They caused the death of two animals. After three years there was still a thick granulomatous cover over the atrial rings which bled easily and caused stenosis of the valve. The wall of the atrium was thickened and partially fibrotic. In one case a marked fibrous reaction was seen on the septum intruding inside the cage and blocking the cone, making the valve both stenotic and incompetent. In a single case, polypoid growths were found on the ventricular wall (Fig. 3).
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FIG. 4. Nickel deposits in insertion area (× 25).

FIG. 5. Metallosis by nickel derivative (× 130).
FIG. 6. *Surface of normal prosthesis (× 20).*

FIG. 7. *Overall corrosion in prosthesis, causing local symptoms of corrosion disease of the heart (× 20).*
The reason for the abnormal local tissue reactions in these six cases may be found in the microscopic findings. There was a marked fibrous reaction around the insertion ring and deposition of a yellow pigment (Figs 4 and 5). The Department of Metallurgy of the University of Technology at Delft analysed the material and found it to be a nickel derivative. In these cases there were also many iron deposits. Both were surrounded by heavy subendothelial fibrous tissue containing some areas of necrosis. These iron deposits can easily be differentiated from the iron-containing blood pigments often seen in early postoperative cases where the pigment is more brownish, has a more granular structure, is much smaller in amount, and shows no abnormal reaction in the surrounding tissues. It is submitted that these metallic deposits should be considered as metallosis of the heart.

In view of this the metal of our implants was carefully scrutinized after removal of the prostheses. None of those with normal healing showed pitting corrosion, but in the six cases with abnormal reactions there was a definite overall corrosion (Figs 6 and 7).

It is well known that, even when corrosion occurs and although metal deposits are found, this does not necessarily mean that pathological tissue reactions, in the sense of a corrosion disease, will follow. Tissue reaction may be of several types—formation of granulation tissue, fibrous proliferation, necrosis of cells, chemical non-bacterial inflammation or a combination of any of these. With the exception of active chemical inflammation, these reactions were all present in the six cases just mentioned. Figure 8 shows granulation tissue around a nickel deposit, while Fig. 9 shows necrosis of the muscles in this area. These six cases therefore present the signs of corrosion disease and should be considered as such.
DISCUSSION

The local tissue reaction in the dog's heart after prosthetic replacement of the tricuspid valve is typical in most cases and leads to normal healing. It is very similar to that described by Berger, Sauvage, Wood, and Wesolowski (1967) and Sauvage et al. (1968). In a minority of cases an abnormal reaction is found, clearly caused by the implanted metal.

Any metallic implant in the body may elicit reactions between the metal and the electrolyte solution of the extracellular space that surrounds it. These reactions are well known from long experience with them in bone surgery; they are corrosion of the material, metallosis of the tissues, and, in some cases, corrosion disease (Mulders and den Otter, 1963). It seems remarkable that, as far as the author is aware, no such reactions have been described after implantation of metal-containing heart-valve prostheses in man, although the late postoperative pathological findings after cardiac valve replacement have been studied intensively (Roberts and Morrow, 1967, 1969). It is also well accepted that metallic compounds have different effects on the frequency of local vessel thrombosis (Afshar, Dennis, Fries, and Sawyer, 1966; Chopra, Srinivasan, Lucas, and Sawyer, 1967; Sawyer et al., 1969). This lack of information on metallosis of the heart in man may well be due to the fact that most modern prostheses are manufactured from vitallium or titanium. These materials are very resistant to corrosion, as demonstrated by the high values of their so-called anodic back electromotive force (ABE); this is expressed in millivolts. It is characteristic of the metal and closely proportional to its observed inertness in tissue (Clarke and Hickman, 1951, 1953). Metals or alloys with an ABE above the critical value of about +400 mV are corrosion resistant. Those with an ABE below it corrode rapidly in the extracellular fluid. Vitallium, for instance, has an ABE of +650 mV and titanium of +3500 mV. Easily corroding metals such as copper and iron have an ABE of −30 and −500 mV, respectively.

The AISI 316 steel used in our prosthesis has an ABE of +480 mV, which is just on the safe side but may prove to be insufficient in some prostheses in view of the many additional factors that aggravate corrosion, such as the method of construction, handling, exposure to movement, and the large differences in pH and oxygen tension in the surrounding tissues. This might explain the metallosis and corrosion disease we found in six of our experimental cases. As to human pathology, the author submits that the possibility of corrosion disease should be considered, especially when an unexplained myocarditis or endocarditis is found at necropsy.

REFERENCES


Clarke, E. G. C., and Hickman, J. (1951). Electrical potential and inertness of metal in biological fluids. J. Physiol. (Lond.), 113, 1P.


