CHRONIC VALVULAR FIBROSIS

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Over 60 years ago, when he proposed surgery for mitral stenosis, Sir Lauder Brunton stressed the importance of first testing its practicability and perfecting the technique on animals (1). Other investigators around that time also recognized the value of studying spontaneous valvular fibrosis in animals, and what was probably the earliest attempt at clinical mitral valve surgery was made in 1907 on a dog with spontaneous chronic valve fibrosis mistakenly thought to have mitral stenosis (2). It is certain that routine successful surgery for mitral stenosis would not have taken almost 50 years to come about if spontaneous mitral stenosis did occur commonly in animals and if Sir Lauder Brunton's suggestions could have been put into practice.

Since 1950, experimental studies of valvular disease have centered primarily on the etiology and pathogenesis of rheumatic valvulitis and bacterial endocarditis. Little information is available on the transition stages between inflammatory valvulitis and chronic valvular fibrosis, even though the latter is recognized as an important aspect of rheumatic fever in man. In animals, as in man, there is often no clear demarcation between valve fibrosis due to aging and that degree which any observer would call pathological.
Although an animal counterpart of human rheumatic fever has not as yet been satisfactorily demonstrated, chronic valvular fibrosis has been recognized in animals for many years. In swine, chronic changes (especially of the mitral valve) were found in 10.6% of 1,650 animals at a slaughter house. These changes were thought to represent healed bacterial endocarditis due to previous erysipelas or streptococcal infection. The resemblance of these valvular and certain myocardial changes to so-called rheumatic stigmata in the human heart (lacking only Aschoff bodies) caused the authors to question the accuracy of the commonly used designation "rheumatic stigmata" in man (3).

In dogs, chronic valvular disease of unknown etiology is the most common type of heart disease recognized (4). It may be defined as a diffuse and/or nodular thickening of the heart valves which is characterized by chronic fibrosis with an increase in both collagen and elastic fibers, edema, and mucinous degeneration. The fibrosis in dogs is located mainly in the subendothelial zone of the atrium at the line of closure of the atrioventricular valves (5). In this respect, the changes seen in dogs closely correspond to aging changes in human heart valves not regarded as rheumatic on the basis of gross examination (6). It has been suggested that valve sclerosis and arteriosclerosis are related fundamental collagen alterations (5). Dogs with chronic valvular fibrosis frequently also have intramural coronary arteriosclerosis and foci of myocardial fibrosis; however, a causal relationship for this triad has not been established (4).

The prevalence of chronic valve disease in dogs increases markedly with age (9/1,000 in dogs less than 5 years old, and 388/1,000 in dogs over 12 years of age). The mitral valve is almost always involved
either alone or in combination, usually with the tricuspid valve. The aortic valve is seldom involved in this process (4), which is in contrast to its frequent involvement in human rheumatic fever. When chronic valvular fibrosis is clinically significant in dogs, it is manifested by atrioventricular valve insufficiency rather than stenosis. No evidence of mitral, tricuspid or aortic valvular stenosis has been observed in 6,500 dogs examined for the presence of cardiovascular disease clinically and/or at necropsy, although such lesions have been observed on rare occasions. The cause of chronic valvular fibrosis in dogs is not known; however, most of the general categories of etiologic factors have been suggested.

Certain other features of canine heart valves are also of interest from a comparative standpoint. Lambl's excrescences, which are commonly found on aged human heart valves, have not been observed in dogs. Fibrosis, fusion, and shortening of chordae tendineae are also infrequent findings in dogs with chronic valvular fibrosis, although broken first order chordae tendineae are occasionally observed. Blood vessels are normally present in the atrioventricular valves of dogs and have been suggested as the route by which serous inflammatory changes take place which later are replaced by chronic fibrosis (7). Dilatation of certain blood vessels appears to account for valve telangiectases which were found in 1.7 percent of 1,293 necropsied dogs (8). The telangiectases, however, could not be statistically related to chronic fibrosis. Lymph vessels are normally present in canine mitral valves, and chronic obstruction to cardiac lymph flow causes these vessels to become dilated. One dog with chronic lymphatic obstruction had a
verrucous endocarditis several months later at necropsy, and several
studies in other dogs with lymphatic obstruction showed them to be more
susceptible to endocarditis following staphylococcal injection (9).
A direct relationship of lymphatic obstruction and chronic valvular
fibrosis, however, has not yet been established.

Chronic valve fibrosis is not as common in other domestic animals,
and was not observed in necropsy examination of over 200 zoo canids
(wolves, coyotes, foxes) of different ages (10).

Some of the questions which merit continuing study in chronic
valve fibrosis are: 1) Why is fusion of the commissures resulting in
mitral stenosis so rare in animals? 2) What are the long term effects
of valve changes which can be experimentally produced with methods
already available? 3) What role do blood and lymph vessels play in
the development of chronic valve fibrosis? 4) What accounts for the
apparent difference between species in the prevalence of chronic valve
fibrosis?

The study of valvular disease as well as other forms of cardio-
vascular disease in certain animals and species is of value particularly
with respect to chronic changes, since a shorter life span reduces the
period required to follow the role of aging in apparently age-related
diseases.
REFERENCES


