The Surgical Utility of a Detailed Knowledge of the Basic Sciences Pertaining to the Mitral Valve

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The confusions which occupy us arise when language is like an engine idling, not when it is doing its work.

Philosophy is a battle against the bewitchment of our intelligence by means of our language

—Ludwig Wittgenstein [1]

Thus wrote Ludwig Wittgenstein in his "Philosophical Investigations" (1973). The point that is to be made is that the language that is chosen to describe lesions etc., can rapidly become ingrained and reproduced without thought. It then gets passed on from generation to generation of cardiac surgeons and accepted as truth unquestioned, leading to unchallenged paradigms, which become translated into therapies that may not be wholly appropriate. One of the principle reasons behind this book on the basic sciences is to present the disease of the mitral valve in the light of what is known and where the gaps in knowledge remain, and to question dogma where it is not matched with evidence.

It is common practice to teach and inform aspiring practitioners of Mitral valve surgery along didactic lines. This is understandable when it is appreciated that we are dealing with human lives and that the practice of cardiac surgery outside of well-defined parameters, can lead to death or severe morbid complications. The practice of mitral valve surgery is at once on at least two levels, first, the safe orchestration of the procedure to obviate the potential complications of operating inside the heart of a fellow human being and second to carry out a procedure which requires a creative eye and a degree of insight less essential in many other procedures. It is to the eternal credit of Professor Alain Carpentier that he described a structured approach to the valve allowing a logical format that for the majority of cases produced a functionally competent valve [2, 3].

Others had been developing techniques for Mitral valve reconstruction at the same time, largely driven by the absence of satisfactory replacement devices in the

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mid-1950s onwards into the late 1960s. Pioneers such as McGoon at the Mayo clinic, Wooler, Kay and Paneth all developed techniques that could be applied to selective lesions of the valve. The McGoon valvuloplasty was directed at isolated mural leaflet prolapse and dealt with it by a triangular imbrication or excision of the billowing excess leaflet with significant success [4].

What Carpentier brought to the table was a functional classification of the leaking mitral valve that is now the mantra of all interested in this subject. The trilogy of dilated orifice and/or leaflet perforation, excessive leaflet motion and restricted leaflet motion continues to practically sub-divide the lesions which in turn leads towards surgical solutions laid out and defined by Professor Carpentier. All of that holds true to this day, almost 40 years since its presentation to cardiac surgeons at large, through the 1984 American association of thoracic surgeon's guest lecture, known now as "the French Correction" [4]. His presentation on that occasion and the paper that followed was prescient in many ways not least the prophesising of robotic surgery for the conduct of the operation.

However, the techniques described therein result largely in a fixed mural leaflet and hence a mono-leaflet valve. The addition of a complete rigid annuloplasty ring immobilises the base of the heart just as does a replacement prosthesis. In fact, this can lead one to ask the difference between a well-seated tissue prosthetic valve with complete sub-valve preservation and this kind of repair. That contention has never been tested in a randomised prospective trial. One wonders at the potential outcome.

Others, led by Carlos Duran have championed the use of a flexible band/ring to allow flexing of the atrioventricular junctional complex [5].

A further development was the lateral thought of Dr. Robert Frater (of the Montefiore hospital in the Bronx New York, New York), to reconstruct the leaflet cords using Goretex© [6] sutures [7]. The Gortex becomes covered in fibrous and endothelial tissue. The gaps in the structure of ePTFE sutures encourage the attachment of host tissue, This was verified in an Ovine model [8], and confirmed in human studies [9]. This move towards artificial cord use has transformed the world of mitral valve repair. It allows the retention of the natural length of the mural and aortic leaflets and the retention of the normal orifice area. It is now the preferred method for many surgeons and has had a significant impact on the development of minimal access to mitral valve surgery.

Surgical reconstruction of the valve has now reached a degree of refinement such that all and any combination of lesions can be addressed through a combination of techniques. There is still a place for leaflet resection, whether triangular, quadrangular or variations of sliding techniques. The free-thinking approach to the use of leaflet support with neo-cords, resection of excessively billowing portions of leaflet, and orifice dimension adjustment with the use of annuloplasty rings (preferably flexible bands in my opinion) gives rise to a toolbox of techniques that can be used in virtually any situation confronting the true Mitral valve surgeon. A full working knowledge and familiarity with all of the above and additional techniques such as the reduction of leaflet height with local base of leaflet resections, leaflet advancement using pericardium and various types of commissuroplasty separate the mitral journeyman from the specialist mitral surgeon.

All of this knowledge is enhanced when combined with a deep working knowledge of the basic sciences of the atrioventricular valve and its role in normal heart function. This is the rationale for this book.

When setting up a case for surgery it is invaluable to spend time inspecting the valve in detail, a "golden 5 minutes" in which to learn and to understand the lesions of each individual valve. Inspection at this level reveals many things that even the best ultrasound and MRI examinations cannot reveal; the presence of secondary and tertiary lesions often lost through echo drop-out and the anatomy of the papillary muscles which is very difficult to identify on echocardiographic examination. Here a sound working knowledge of the embryology and anatomy of the adult valve is key to understanding the lesions and the best way to deal with them.

Knowledge at this level also encourages the re-examination of taught dogma. The classification of valve pathology into fibro-elastic deficiency and myxomatous disease with the Barlow's valve at the extreme of the spectrum, whilst edifying the type of lesion, it does little to inform the mechanism of their development. It is of interest that in the so-called fibro-elastic deficient group the part of the leaflet that is prolapsed (most often P2) is the only thickened part of the valve leaflets. The rest being homogeneous in appearance. In the paper describing the comparative histopathological analysis of mitral valves in Barlow disease and fibro-elastic deficiency (FED) the glaring error is that the only parts of the valve examined histologically in the FED group are those resected at surgery [10]. It is the author's experience that the remainder of the valve is usually functionally normal. This is also borne out by the fact that in Carpentier's own data the repaired valves in this group have at least an 80% 20-year functional survival [11]. If it was a disease of the valve as a whole, other parts would surely degenerate and fail over this time period and that is not the case in practically all reported series.

It is my contention that what we are seeing is the result of an excessively loaded portion of the valve in a congenitally malformed valve. If we think of the valve as a force-field at end systole bearing pressures of up to 200 mm Hg on extreme exercise if the leaflets cannot spread that load around the whole valve then these pressure points will be traumatised over time. If we then look at the histological changes in this light, we see that they correspond to trauma-induced change with collagen disruption and the laying down of myxoid tissue, all changes seen around excessively traumatised joints.

Thus, the presence of deep interruptions in the mural leaflet which in almost all of these valves occurs, exposes these segments to a load that is isolated and traumatic. In addition, we commonly see in these patients highly abnormal papillary muscle arrangements that also prevent the normal load sharing of the normal valve. Hence in summary what we are seeing is a valve that cannot deal with the systolic stresses that a normal valve is able to do.

Pre-operative imaging focusses upon the leaflet lesions and is excellent at recognising the three groups of lesions described by Carpentier. However direct inspection is superior in all but functional aspects. If the surgeon focusses upon what the actual visible lesions are rather than looking for those described in the cardiologist's report, a better understanding of the real situation is forthcoming. The actual

anatomy of the valve around the prolapsing segment usually reveals interesting additional lesions, such as interruptions in leaflet growth rendering what many refer to as deep clefts (which are in fact interruptions in leaflet growth), highly abnormal papillary muscles, and calcification. The complete understanding of the valve will often lead to a modified approach to the repair. This may also prevent early recurrence that can be the case if other lesions are missed, or the height of coaptation is irregular and all of the leaflet(s) are not fully supported.

Hence the broadest understanding of the atrioventricular valve complex and its particular morbid anatomy is essential to optimise the reconstruction of the valve in each individual case.

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