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## “What Made Me Study Aortic Stenosis?”

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**Abstract** I, Professor Nobuaki Tanaka, gave a Farewell Lecture on February 29, 2024. First, I talked about the episode that led me to start researching aortic stenosis, and reflected on my own lectures on this topic to students aiming to become medical laboratory scientist. Lastly, I spoke to students majoring in medical laboratory scientist about the importance of practical clinical training.

*Key words:* aortic stenosis, on-the-job training (OJT)

### Opening Remarks

*To the all of audience – student, colleagues, and professors in attendance.* Thank you everyone for taking time out of your busy schedules to attend my final lecture today. The content of the lecture is mainly aimed at students of Laboratory Sciences.

Now, upon my retirement, I thought a lot about what I should talk about, and I decided to title it “What made me study Aortic Stenosis (AS)?” Please feel free to listen to my talk about AS of my experience. (Fig. 1)

Fig. 2 is from “Researcher Information of the Health Sciences Booklet, Yamaguchi

University Graduate School of Medicine 2023”, which was published for the recruitment of graduate students for the Health Sciences Course. I wrote some comments on AS there. Namely, the ratio of acceleration time (AT) and ejection time (ET) of the aortic ejection flow well correlates with AS severity.



Fig. 1 Final lecture

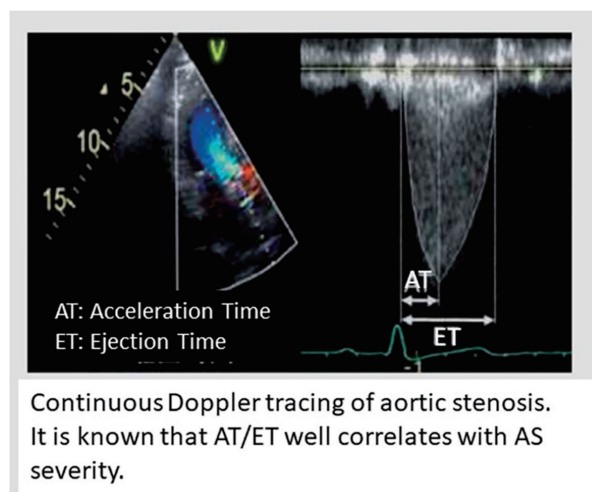


Fig. 2 Continuous Doppler record of AS. (from “Researcher Information of the Health Sciences Booklet, Yamaguchi University Graduate School of Medicine” 2023)

This article was written based on the final lecture held on February 29, 2024 at Yamaguchi University, School of Medicine, S1 room. Nobuaki Tanaka is a Professor of Emeritus, Yamaguchi University.



Fig. 3 Autopsy case of AS with bicuspid aortic valve.

### A case of AS

This photo (Fig. 3) is an autopsy example of AS with a bicuspid aortic valve.

The medical history of this autopsy case is as follows.

**Case:** 67yo, male.

**Chief Complaint:** Shortness of breath on exertion.

**Present History:** March 198X, while on a domestic trip, he had no particular symptoms up until the middle of the trip when climbing stairs or climbing hills, but on the way home he began to feel fatigued all over his body. In the evening after returning home, the patient's appetite decreased and he began to have difficulty breathing even when resting. The next day, his breathing difficulties did not improve, and he developed a condition like "cardiac asthma", and was admitted to our hospital after visiting his nearby clinic. At the time of admission, he was 162 cm tall, weighed 58.5 kg, and had a pulse rate of 82 bpm, regular. His blood pressure was 156/96 mmHg, and respiratory rate was 31/min. According to the record, the first heart sound was increased, the second heart sound was decreased, the third heart sound was not audible, and the fourth sound was audible. A coarse mid-to-late systolic murmur of Levine 3/VI was audible at the 2nd intercostal space of the right sternal border (2RSB). This systolic murmur radiated to the right neck. Piping rale was heard in all lung fields.

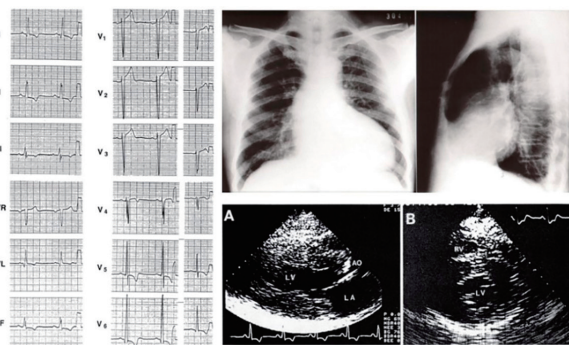


Fig. 4 ECG, Chest X-ray, Echocardiogram of the case (Left) ECG. (Right upper) Chest x-ray. (Right lower) Echocardiogram. A Parasternal long axis view. B Parasternal short axis view.

Crepitation was observed at both lung bases during inspiration. No significant findings were observed on the abdomen.

This electrocardiogram (Fig. 4, left) was taken after the acute symptoms of heart failure had subsided. Heart rate was 72 bpm in regular sinus rhythm, and typical left ventricular hypertrophy with left ventricular strain pattern was observed. Chest X-rays showed cardiomegaly (Fig. 4, right upper).

As of 2024, echocardiography is tremendously advanced compared to 1980's, and very useful even in the acute heart failure stage. Echocardiography can make it possible to reveal the cause of heart failure, and the severity of AS in a short time. However, in 1980's, although continuous wave Doppler was available in Japan, it was not yet widespread, and recording and evaluation of blood flow velocity through the aortic valve was not common. After the acute phase symptoms subside, echocardiographic examination was done in the laboratory room. The examination was mainly performed using 2D scanning by the SSH40A (Toshiba, Japan), which was equipped with pulsed Doppler, but not equipped with a continuous wave Doppler yet. Left ventricular ejection fraction was 77%, ventricular septal wall thickness was 22 mm, and left ventricular posterior wall thickness was 21 mm (Fig. 4, right lower). Calcification of the aortic valve and restriction of its opening was observed, number of sinuses of Valsalva might be 3, but the number of aortic

cusps was unknown. Echocardiographic summary of the best possible diagnosis was 1) the patient had severe aortic stenosis caused by a degenerative deformed calcified aortic valve, and 2) marked concentric left ventricular hypertrophy.

Phonocardiogram after the symptoms of heart failure have subsided is shown in Fig. 5. In 1980's, phonocardiograms were recorded normally. A diamond-shaped ejection systolic murmur was recorded at 2RSB, and the carotid pulse tracing was a slow-rising “pulsus tardus.” These tracings are typical for the severe AS.

Records of cardiac catheterization are also available. There is a pull-back pressure tracing from the left ventricle to aorta, and the pressure gradient between left ventricle and aorta is likely to be about 130 mmHg as peak to peak (Fig. 6).

Aortic valve area (AVA) and its index AVAi were  $0.38 \text{ cm}^2$  and  $0.23 \text{ cm}^2/\text{m}^2$ , respectively, therefore the patient was considered to be indication of aortic valve replacement (AVR). However, he became an autopsy case

unexpectedly. He died suddenly in the hospital ward. This is a photo after formalin fixation, and it shows AS with a bicuspid valve (Fig. 3).

He had a Holter ECG test on May 2nd - 3rd, and since May 3rd was a holiday for Constitution Day in Japan, he was removed the Holter equipment in the ward, but just after that, he was found collapsed in the bathroom while taking a shower. Cardiopulmonary resuscitation was immediately started, but there was no response and the patient was confirmed dead.

At a later conference, my professor at the time consoled me by telling me that even if I performed cardiac massage powerfully, I would not be able to obtain an effective cardiac output with severe AS. Ever since this sudden death happened right in front of my eyes, it has become clear to me that “sudden death” is possible when it comes to severe AS. This thought continues in my head even now.

Atherosclerotic or degenerative AS of normal tricuspid aortic valve is now a very common type seen in people over 80 years of age. However, bicuspid valve AS, as seen in this autopsy case, is congenital and may develop into severe AS relatively young ages, like in their 60's (the autopsy case was 67 years old). This kind of knowledge is now widely known, and I also talk about sudden death in severe AS in my lectures to students. I didn't really want to experience patient's sudden death by myself, but in a sense, it was traumatic for me to experience something like that at the beginning of my third year as a doctor. I think this is the reason why I asked my students data of patients with AS.

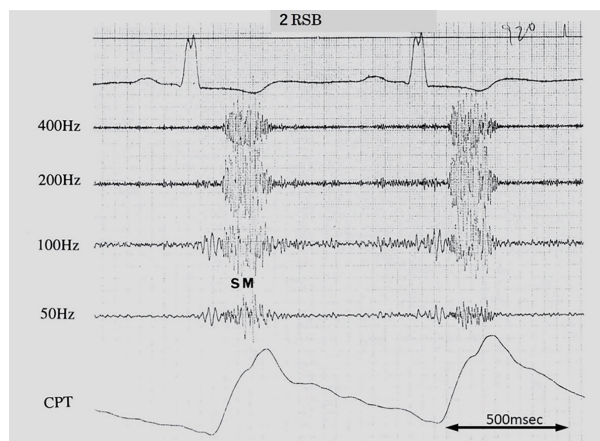


Fig. 5 Phonocardiogram



Fig. 6 Pull-back pressure recording from Left ventricle (LV) to the aorta (AO).

**When severe subjective symptoms appear in AS, AVR is indicated**

The data published in the late 1960's paper is still often cited.<sup>1</sup> The prognosis is poor when severe symptoms appear in AS, so AVR should be considered in such cases. A characteristic harsh, systolic murmur can be heard radiating from the 2RSB to the neck, and paradoxical splitting of the 2<sup>nd</sup> heart sounds is thought to suggest severe AS.

I stayed two years from the fall of 1993 to the fall of 1995 in John Ross Jr.'s laboratory



Fig.7 Seaweed Canyon Lab in 1995

at UCSD (Fig. 7). On that occasion, I have showed Dr Ross this slide of the bicuspid aortic valve (Fig. 3), which was very impressive to me.

When AVR is indicated for severe AS, it is replaced with a mechanical valve or bioprosthetic valve. Recently bioprosthetic valves have been replaced with catheters.<sup>2</sup> It's transcatheter AVR (TAVR). It has already been more than 10 years since TAVR was introduced to Japan. With the advancement of treatment methods, in lectures for laboratory students, whenever I talk about valvular heart disease, I also talk about the measurement of the valve orifice area including the continuity equation for echo diagnosis of AS. I also talk about assessment of AS severity.

By the way, in the national exam for medical technologist of this year, peak Pressure Gradient (PG) of AS based on the simplified Bernoulli formula was questioned! I don't think any of my students did it wrong.

Actually, peak PG by echocardiography and that by catheterization are different. Former is peak instantaneous gradient between left ventricular (LV) pressure and aortic (Ao) pressure, on the other hand, latter is just the pressure difference between peak LV pressure and peak Ao pressure. Generally, PG by echo is greater than PG by catheterization, as well known described in the many echo textbook.

### For Laboratory students

In lectures for laboratory students, I give priority to simple and clear understanding, that is all about diagnosis of AS. However, when a sonographer is actually in charge of testing real patients, he or she will be faced with slightly complicated topics such as low-flow and low-gradient AS. To experience and understand such a wide range of things, high-quality on-the-job training (OJT) is necessary. Therefore, students who come to me for their master degree course are required to undergo training in the echocardiographic laboratory of the affiliated hospital. Without this training, they will be of no use in clinical practice, and what may seem like a waste of time is actually valuable time for training. I think such circumstances in detail of things is probably the same in any specialized field.

Low-flow and low-gradient AS are also important in the actual clinical diagnosis of AS, but they are intentionally omitted from my lectures at laboratory students for simple and clear understanding for beginners. Therefore, even if you are interested in this field and want to become a specialist who can be useful to society, you will need to continue studying in this field and understand slightly complicated situation of the disease.

### Closing: what made me study aortic stenosis?

Around 1985, when I experienced the case I mentioned earlier, most of the diagnostic cases by cardiac catheterization were ischemic heart disease (IHD) and rheumatic valvular heart disease like mitral stenosis (MS) and aortic regurgitation (AR). However, I didn't see many AS cases in those days. It might be that AS was just less common at that time, or it might be the relatively short average lifespan compared to recent days. At the very least, there has never been as many AS cases as there are now, 2-4 TAVR cases a week. Nowadays, AS has become a very common disease reflecting the aging of the population, but the shocking case I showed you at the beginning has made me study AS with my master course students.

Whenever I hear the word "severe AS," I don't feel at peace, even now. Perhaps, it is because of the impressive sudden death case I came across as my first case of AS, and it

made me study AS.

Thank you very much for your attention.

### Acknowledgment

I graduated from Kagoshima University (KU) (Kagoshima, Japan) in 1983 and moved to Yamaguchi University (YU), 2nd Dept of Internal Medicine (Ube, Japan) where I started the career as a medical doctor. After studying at YU, the National Cardiovascular Center Research Institute (Suita, Osaka) and UCSD (La Jolla, USA), I worked at Shimonoseki-city Medical Association Hospital (Shimonoseki, Japan), YU Hospital (Dept of Laboratory, Ube, Japan), Yamaguchi Prefectural Grand Medical Center (Depts of Clinical Laboratory, and Cardiology, Hofu, Japan). For the past 11 years, I have worked as a professor at YU Graduate School of Medicine (Dept of Clinical Laboratory Sciences, Ube, Japan) then retired. For 41 years since graduating from KU School of Medicine, my foothold was always YU. I wish to thank my family, spouse Etsuko, children, parents, and brothers, also my great teachers, seniors, peers, juniors, and everyone who has supported me.

### Conflict of Interest

The author declares no conflict of interest.

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