A simple method was proposed to calculate the work of human ventricle that has been transmitted to arterial system so as to make clinical laboratory examination cheap and quick. Measured arterial pressure was approximated by a serial expansion of time as an input for the equivalent electrical circuit of arterial system. The transmitted ventricular work to arterial system is obtained by calculating the power at the equivalent electrical circuit of arterial system which parameters are different among the patients. This method provides the cheapest cost in the routine laboratory examination for ventricular function.

Key words: Ventricular function, Catheter technique, External work, Power, Resistance, Compliance

1. Introduction

Evaluating ventricular function is important not only for investigating physiological circulatory state but also for patho-physiological state of patient who are suffering from cardiovascular diseases such as hypertension, ischemic heart disease and dissecting aneurysm of aorta. Up to now, the most reliable index of ventricular contractility is the peak value of the instantaneous pressure-volume ratio, $\text{Emax}^{(1)}$. It can be measured at the endo systolic pressure-volume loop, ESPVR$^{(2)}$ of ventricular contraction. To obtain $\text{Emax}$, we have to measure the instantaneous ventricular volume precisely by conductance catheter. This method, however, is expensive and is still not used extensively in routine clinical laboratory examination.

In the present investigation, we show a simple method to calculate the external work of ventricle that has been transported to the arterial system by routine laboratory examination.

2. Physiological back ground

The ventricular function can be expressed by the $\text{Emax}$, the peak of the instantaneous pressure-volume ratio$^{(3)}$. This index expresses pure ventricular contractility which is independent from the loading conditions, venous return, arterial pressure and heart rate$^{(4)}$. Fig. 1 illustrates the endo systolic pressure-volume loop, ESPVR. The peak value of the ESPVR is the $\text{Emax}$. This loops is composed of the external work of ventricle (shaded area) and the potential energy of ventricle (triangle facing to the external work). The amount of the external work depends on the stroke volume and the peak ejection pressure. The potential energy is a function of inherent elastic potential energy in the cardiac muscle fibers and is devoted to increase the elasticity of ventricle itself. When a ventricle ejects out blood, about 2/3 of the potential energy of ventricle is shown to be converted to the external work of ventricle$^{(5)}$. The external work and the potential energy constitute the total work of a ventricular contraction. By the series of experiments by Suga$^{(6)}$, the sum of the
external work and the potential energy was shown to be proportional to the oxygen consumption of a ventricular contraction\(^\text{[5]}\).

The most important item in the circulatory system is to transmit the work of ventricle to arterial system. The transmitted work evokes the arterial pressure wave in aorta. The pressure wave is further transmitted to arterial bed where the energy is expended at arterial resistance by viscous friction. The arterial pressure curve is therefore, determined by the amount of work of ventricle and properties of arterial system. Thus, the work of ventricle that has been transmitted to arterial system can be approximated by the electrical power loss in an equivalent electrical circuit of the arterial system. We calculate the (electrically equivalent) power consumption of arterial bed as the energy transmitted to the arterial bed from the ventricle.

3. Method

3.1 Measuring ventricular and aortic pressure of human

9 patients under the hospitalization for diagnosis and treatment of cardiovascular diseases were examined. Swan gantz catheter was inserted from right femoral artery under a local anesthesia of 5% Lidocaine. The upper part of Fig.2(a) shows a typical example of continuous recording of pressure curves from Hypertensive patient. First pressure curve is left ventricular pressure. The second and third ones are recorded immediately beyond the aortic valve. They showed elevation of diastolic pressure and were identified as aortic pressures. Arrows indicate the aortic notches that manifest at the closure of the aortic valve. The lower part of Fig. 2(a) is the ECG recorded simultaneously from the patient.

Pressure curves in Fig.2(b) are those from the normal subject. The notches in ventricular pressure curves are the artifacts by turbulence. We have certified that there was no pressure drop nor step up within the ventricle nor between ventricle to aorta. The diastolic arterial pressure curves were also recorded to obtain arterial compliance.

Another catheter was inserted from right femoral vein to measure the cardiac output by thermo dilution method. This procedure was iterated for four to five times so as to get a stable value of cardiac output.

3.2 Calculating resistance and compliance

Usually ventricular pressure recorded by Swan gantz catheter method as shown in Fig.2(b) has artificial notches at near the peak pressure due to turbulence.
Fig. 2. Examples of recording of pressure curves of a patient. The top of Fig. 2(a) is a continuous recording of the ventricular pressure and aortic pressure. The lower part of Fig. 2(a) is the ECG (electrocardiography) recorded from a patient with hypertension. Fig. 2(b) is those from the normal subject. The notches in aortic pressure curves are the artifacts by turbulence. The aortic notches caused by the closure of aortic valve are indicated by arrows.

Therefore, we adopted the mid values of these oscillating pressures. The onset of ejection was determined from the R wave of ECG. Arterial resistance, $R_a$ is calculated by dividing the cardiac output by the mean arterial pressure. The diastolic properties of the arterial system is approximated by

$$P_d(0) = \exp(-T_e/(R_aC_a))P_d(t_e) \quad (1)$$

where $t=0$ is the end diastolic instance, $P_d(0)$ is the end diastolic arterial pressure, $t_e$ is the ejection duration, $P_d(t_e)$ is the end ejection aortic pressure and $T_d$ is the diastolic period. All of these parameter values can be obtained by an on line recording system. Operating the logarithm on the both sides of equation (1) and dividing by $R_a/T_d$, aortic compliance, $C_a$ is

$$C_a = -T_d[\log(P_d(0)/P_d(t_e))R_a] \quad (2)$$

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3.3 Calculating the power on arterial bed

For the simplicity, we use an equivalent electrical circuit model (the Wind Kessel model) to describe the arterial system (Fig. 3). It is composed of aortic valvular resistance with characteristic impedance $R_i$, arterial resistance $R_a$ and aortic compliance $C_a$. Putting $V(t)$: the ventricular pressure, $I(t)$: the flow rate across the aortic valve (ml/s), $V(t)$: aortic flow rate and $V(t)$: the arterial pressure as an output. Then, the arterial system is described by

$$\begin{align*}
V(t) &= I(t)R_i + V(t) \\
V(t) &= I(t)R_a \\
I(t) &= \frac{V(t)}{R_a} + C_a \frac{dV(t)}{dt}
\end{align*} \quad (3)$$

By operating the Laplace transformation, the transfer function $G(s)$ of this system is ($s$ is Laplace operator)

$$G(s) = \frac{P(s)}{I(s)} = \frac{R_i}{[R_i + R_a + C_aR_iR_a]}$$

In the present work, we define the equivalent electrical power that are transmitted to arterial system across the aortic valve by

$$PWA = \int_0^\infty I(t)^2R_i dt + \int_0^\infty \frac{V(t)^2}{R_a} dt \quad (7a)$$

The first integration describes the viscous frictional loss when blood flows through aortic valvular resistance $R_i$. We tentatively call this integral value, $PWC_a$. The second integration is the energy consumed at arterial resistance $R_a$ and we name it $PWR_a$. The sum of these two integrals (we name it $PWA$) is purely electrical defined energy loss in the arterial system.

We calculate another power that are hydraulic one by

$$PWB = \int_0^\infty I(t)V(t) dt \quad (7b)$$

Equation (7b) ($PWB$) describes the fluid dynamical power. This power is equivalent to the product of aortic pressure $V(t)$ and aortic flow $I(t)$. $I(t)$ can be...
obtained from equation (5) by measuring aortic pressure $V(t)$.

The temporal change in ventricular pressure $V(t)$ can be expressed by a function of time, $t$ through the serial expansion

$$V(t) = a_0 t^6 + a_1 t^5 + a_2 t^4 + a_3 t^3 + a_4 t^2 + a_5 t + a_6$$

by the chebychev approximation method. Dimension are, $R_a$ (mmHg s/ml), $C_a$ (ml/mmHg), $T_a, T_e$ (sec), stroke volume, $V_a$ (ml), Peak pressure (mmHg), $HR$ (beats/min), $CO$ (Cardiac out put) (L/min), Power (mmHg ml). Table 1 shows the system parameters and powers with patients diseases.

4. Results

Table 1 shows all the clinical data of nine subjects with 2 normal subjects (indicated by $N$ in table 1). The powers, $PWR_a + PWC_a$ in the table 1 equal to $PWA$ and power, $PF$ equals to $PWB$. The amount of calculated powers are different among the subjects which have different patho physiological states.

5. Discussion

The purpose of the present investigation is to show a simple method to calculate the external work produced in ventricle that has been transmitted to arterial system. The maximum value of $E_{\text{max}}$ is the most reliable index. Measuring the $E_{\text{max}}$, however, costs considerable and wastes time. These difficulties are undesirable for the patients under the cardiac catheter examination. The method we describe can be performed under the routine clinical examination. The advantages of the present method comparing with older indices are

1. cheaper price to obtained data, 2. easier and quick and safety., 3. easy repeatability. 4. to evaluate the total function of the circulatory system.

<table>
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<tr>
<th>Patient</th>
<th>HR (beats/min)</th>
<th>$CO$ (Cardiac output)</th>
<th>Vs (Stroke volume)</th>
<th>Pf (product of arterial pressure and aortic flow rate)</th>
<th>s-BP (systolic arterial blood pressure)</th>
<th>d-BP (diastolic arterial blood pressure)</th>
<th>N: Normal subject</th>
<th>A.SD: Atrial septal defect</th>
<th>Ht: Hypertension</th>
<th>AS: Aortic stenosis</th>
<th>MV-pro: Mitral valve prolapse</th>
<th>Post-Op: Post cardiac surgery</th>
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Table 1. Data recorded from the patients in Asahikawa medical college.

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The present method is not developed for the diagnosis of diseases but to evaluate the systolic function of the ventricle.

5.1 Description of arterial system

The models of arterial system are categorized to distributed parameter models and lumped models. Distributed parameter model can describe the transmission of pressure pulse wave. This model has been used to characterize the local arterial circulation through the input impedance, the local complex impedance and the characteristic impedance. This model can describe minute properties in arterial pulse wave transmission. It, however, is inadequate to describe an entire behavior of the arterial tree as a whole system. Because it is almost difficult to measure all the bio-mechanical properties of arterial blood flow even at the local arterial system.

The lumped parameter model is the most frequently used characterization of arterial system. The validity has been verified in terms of the total length of the systemic arterial tree and the length of the pulse wave. Because the pulse wave velocity of the arterial system (even of aorta (4 m/s)) is sufficiently fast to transmit an entire arterial tree (170 cm on an average from the top of the head to the bottom of foot finger). Therefore, the pulse can be approximated to transmit almost simultaneously at a time to all the portion of the arterial system until the tip of the capillary. Thus, lumped model can sufficiently approximate the distributed parameter model. There are many modification of the lumped model. For the clinical application, however, the simplest Wind Kessel model is the most suitable. Because in actual clinical examination, it requires much damage for the patient to measure proximal aortic resistance, peripheral arterial compliance, internal viscous resistance and inertance of aorta. Therefore, we have adopted the simplest three elements model.

5.2 Energy consumption as the transmitted ventricular power produced in the ventricle

Ventricle system can extract only the external work of the total pressure volume area of the ventricular contraction. The external work is equivalent in dimension (product of pressure and flow) to the power in an electrical circuit. The power is consumed as an energy loss in the ventricle by the viscous retardation. For the quantitative evaluation of the amount of the external work, therefore it is conceivable to calculate (pressure)^2/(Resistance) or (flow)^2/(Resistance) by the Ohmic law. Because aortic flow can be approximated by equation (5), only the time courses of pressure curves are needed to measure. Another measure is the product of pressure and flow which is also equivalent to the power. This quantity is comparable in the physical meaning to the stroke work which has the same dimension and the same physical meaning with the present power that is immediately before being transmitted to arterial system. In the following discussing, we show the comparison with reported human data.

5.3 Evaluation of calculated power

There scarcely found of measuring the powers by the present method. We have compared the present calculated data to the stroke work (mmHg ml) of human subjects measured by a conductance catheter method. Stroke work in the normal human subjects ranged 4,025±1,544 mmHg ml (Takano 1993), 3,225 - 8,105 mmHg ml (Asano H. 1989), 3,682-6,528 mmHg ml (Kameyama 1991), 5,850-7,200 mmHg ml (McKay R.G. 1986). The radio nuclei technique for measuring ventricular volume reported stroke work ranges 6,875-7,590 mmHg ml (McKay R.G. 1984). In the present investigation, for the normal subjects, the power loss ranged 3,694-8,740 mmHg ml evaluated by equation (7•a) and 5,074 to 8,321 evaluated by equation (7•b), respectively. Therefore, we consider that about the normal subjects, the present method can approximate the stroke work measured by the conductance catheter technique.

About hypertensive patients, powers evaluated by the integration (7•a) have enhanced (11,811-11,527 mmHg ml) considerably than the normal subjects. These elevation are particularly due to the increase of the arterial resistance 1.51 mmHg s/ml (subject C), 1.22 mmHg s/ml (subject E) and reactive elevation of the ejectional pressure. Decrease in hydraulic power B (equation (7-b)) in ASD (aortic septal defect) is due to the inter ventricular shunt from left atrium to right atrium. As a result, total venous return for the left ventricle has been reduced.

It is, however, impossible to compare the data among individual subjects because, the reported patients are not perfectly the same to the present subjects.

6. Conclusion

A simple method to calculate the power transmitted from the human left ventricle to the arterial bed in terms of the ventricular external work was proposed. The present method is available in the actual clinical medicine.

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