

Spectral Analysis of Radial Pulse in Patients with Acute, Uncomplicated Myocardial Infarction

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SUMMARY

The aim of this study was to examine the frequency spectrum of arterial pulses in 17 patients with acute, uncomplicated myocardial infarction. Recordings of right radial pulses were taken from these patients immediately after their arrival at the emergency room. This information was considered as baseline data and was monitored at the same time each day for up to 7 days. These serial recordings of radial pulses were then analyzed together with recordings from 3 control groups of patients not suffering from myocardial infarction. These included subjects who underwent coronary arteriography ($n=24$), coronary angioplasty ($n=18$), and mitral valvuloplasty ($n=13$). The method of analysis involved a discrete Fourier transformation of radial pulses recorded from an external pulsation transducer to obtain "power spectra" for pulse waves. There was no significant change in the power spectrum for each of the 3 control groups although significant clinical and hemodynamic improvement was observed in the coronary angioplasty and mitral valvuloplasty patients. In sharp contrast, the intensity of the frequency moduli C2 and C3 in the target group fell on arrival at the emergency room and then gradually increased while the average pulse energy (frequency modulus C0) decreased simultaneously with stable recovery from the acute myocardial infarction. In order to investigate the changes in C2 and C3 in the target group, the effective renal plasma flow (ERPF) was first determined for 10 of the 17 patients using intravenous I-131 labelled iodo-ortho-hippurate. A second reading was taken a week later. Eight of the 10 patients were found to have a relatively "higher" ERPF at the onset of acute myocardial infarction with an average reading of 450.1 ± 168.9 ml/min, compared with the data obtained a week later (374.1 ± 130.4 ml/min, $p < 0.02$). An inverse correlation existed between the percentage of "initial drop" in frequency moduli C2 and C3 in the power spectra of the radial pulses and the percentage of "initially higher" ERPF ($r = -0.66$ and -0.70 , respectively, $p < 0.05$). It is concluded that there was a specific change in the power spectrum of the radial pulses which were recorded and

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analyzed noninvasively in patients with acute, uncomplicated myocardial infarction. Changes in the ERPF may exert an influence on the frequency moduli C₂ and C₃ of the power spectra of the radial pulses after heart attack, suggesting that the aorta and the closely organs may cause coupled oscillation. Theoretically this structure is equivalent to a resonance circuit. (Jpn Heart J 34: 131-143, 1993.)

Key Words:

Discrete Fourier transformation Power spectrum Radial pulse
Acute myocardial infarction

IT is well documented that the volume and contour of arterial pulses reflect a combination of cardiocirculatory functions.¹⁾ It follows that an analysis of the pulse wave can be a useful method with which to assess cardiocirculatory function.²⁾

One of the most powerful tools in signal processing is the frequency analysis technique involving Fourier transformation. Bennett and Fischer³⁾ applied a spectrum analyzer to assess the diagnostic utility of plethysmographic waveforms in peripheral vascular disease. Ting et al⁴⁾ resolved the pressure and flow signals into their Fourier harmonics to calculate the input impedance modulus and phase angle for each harmonic. Marble and his colleagues⁵⁾ used similar techniques to demonstrate the correlation between the amplitudes of the 7-14th harmonics and the occurrence of coronary artery disease. Furthermore, in one animal study, Young and his colleagues consistently demonstrated different harmonic patterns of pulse waveform in the tail artery of rats when the left renal or superior mesenteric arteries were very briefly clamped.⁶⁾

We thought it appropriate to undertake a study to examine the frequency spectrum, also called the power spectrum, of arterial pulses in patients with acute, uncomplicated myocardial infarction as well as the possible mechanism responsible for such a special spectrum, if one occurred. It was our hope that such a noninvasive technique would provide some diagnostic clues which would be helpful not only in the identification of the disease but also in providing further insight into our understanding of arterial pulses.

MATERIALS AND METHODS**Patients**

Four groups of patients were included in this study. All these patients were selected from the Veterans General Hospital-Taipei.

Group 1. Patients with acute, uncomplicated myocardial infarction

This group included 17 patients, 15 males and 2 females, with an age range of 44 to 84 years (mean 65 years). The diagnosis of acute myocardial infarction

was documented by typical chest pain, typical electrocardiographic (ECG) S-T segment and Q wave changes and cardiac enzyme/isoenzyme elevation. Ten patients were found with anterior and 7 with inferior myocardial infarction (with combined right ventricular infarction in one, posterior infarction in one and anterolateral infarction in another, according to the ECG changes). Patients who had experienced shock or congestive heart failure even with very mild basal, moist rales, or who were admitted to hospital 6 hours after the onset of acute severe chest pain, were excluded. Medication was prohibited except for low dose propranolol (5 or 10 mg twice a day) or diltiazem (30 mg twice a day) and an intramuscular injection of meperidine or morphine where necessary. No diuretic or inotropic agent was allowed. Immediately after admission, daily recording of the radial pulse was taken for 7 days. However, only 4 sets of data are shown in this report: the data obtained immediately after hospitalization (D0, before intravenous infusion of streptokinase SK 1.5 million units, if given) and at 24 (D1) and 48 (D2) hours after the first recording, respectively, as well as the data obtained when the cardiac enzyme, CK, returned to normal (Dn, about 4.7 ± 1.2 days after hospitalization). All patients in this group were treated to ensure an uneventful course during this period.

Group 2. Patients admitted for coronary arteriography

Twenty-four patients (18 males and 6 females) with ages ranging from 45 to 72 years (mean 58 years) were admitted for coronary arteriography to evaluate their chest pain which was typical of ischemic heart disease. Each patient had a radial pulse recording one day before and after coronary angiography using the standard Judkins method. After angiography, they took no additional medication prior to the subsequent radial pulse recording.

Group 3. Patients admitted for percutaneous transluminal coronary angioplasty (PTCA)

To evaluate whether or not successful coronary angioplasty would have any effect on the power spectrum of the radial pulse, 18 patients (14 males and 4 females) were studied. Their ages ranged from 48 to 72 years (mean 62 years). After angioplasty, each patient's general condition and treatment remained the same as that before the procedure. PTCA was defined as successful when either the residual stenosis was less than 30% or the transstenotic pressure gradient was less than 20 mmHg, while the cardiac enzyme, CK, was maintained at less than 1.5 times the initial value or not greater than 200 IU/L. The radial pulse was recorded before, as well as 3 and 10 days after the PTCA.

Group 4. Patients admitted for percutaneous transluminal mitral valvuloplasty (PTMV)

In our experience, a successful PTMV can significantly increase cardiac output and reduce both the pulmonary artery pressure and the pressure gradient

across the mitral valve in patients with isolated rheumatic mitral stenosis.^{7),8)} Thirteen such patients, all females with ages ranging between 24 and 54 years (mean 46 years), were included in this study. Patients who had greater than grade 2 plus mitral regurgitation on the left ventricular angiogram or who were in atrial fibrillation were excluded. A successful PTMV should be able to reduce the transmitral pressure gradient by 50% or more, increase the mitral valve area by 50% or more and significantly increase cardiac output by 50% or more. Blood loss should be minimal during the procedure (which was undertaken in our case using the Inoue balloon catheter technique). No additional medication was allowed after successful PTMV. The radial pulse was recorded one day before, as well as 3 and 10 days after the procedure.

Radial pulse recording and analysis

The prophylactic infusion of 2% xylocaine or heparin was temporarily

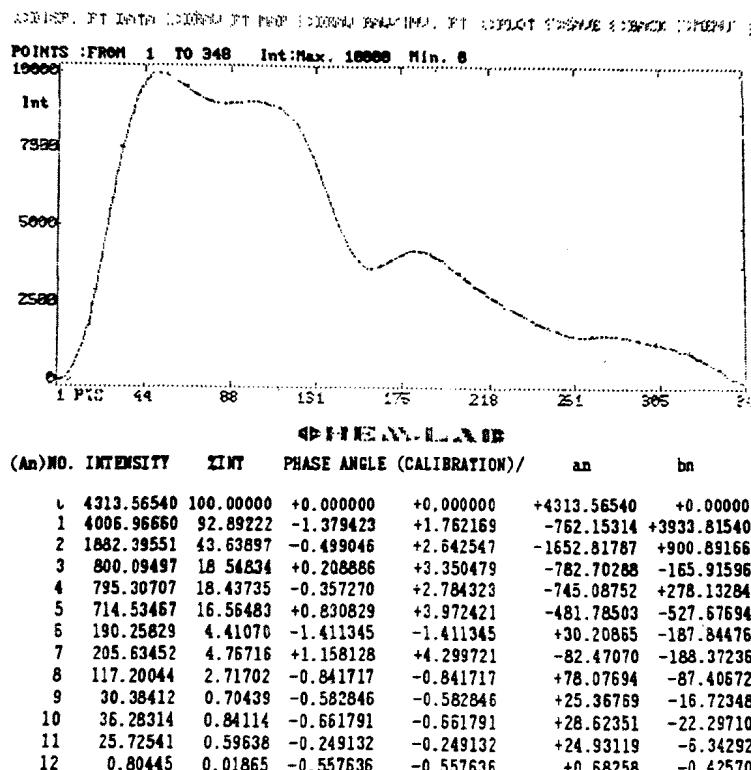


Fig. 1. Upper panel shows one radial arterial pulse (after digitization) recorded from patient #4. Lower panel shows the average intensity and percentage of the average intensity as well as the phase angle of an arterial pulse of frequency moduli C0 up to C12 after discrete Fourier transformation. Phase angle is actually derived from inverse tangent of "bn/an". The data under calibration indicated the phase angle corrected by "an" and "bn" between $-\pi$ and π . "an" and "bn" indicated the real and image part of discrete Fourier transformation, respectively.

discontinued at least 30 min before radial pulse recording, if such medications were already being given to the acute myocardial infarction patients. Each patient was asked to lie on the bed quietly for more than 15 min prior to right radial pulse recording with a pressure transducer (PSL-200 GL, Kyowa Electronic Instruments Co. Ltd., Japan, frequency response flat to 100 Hz) which was initially attached with adhesive tape but later fastened with an adjustable belt which had a small knob on it to give a suitable pressure to the transducer. A good radial pulse recording was one of large amplitude clearly demonstrating the anacrotic or dicrotic notches of the pulse wave (Fig. 1). Variations in heart rate, blood pressure and pulse pressure were also recorded. These were required to vary less than 10% among two or more radial pulse recordings.

The transducer was connected to an analog-to-digital (A/D) converter and then to an IBM PC-AT. The pulse wave was analyzed using discrete Fourier transformation software with T (period) equal to one pulse (sampling frequency 500 Hz). The details of the recording technique and analysis have been previously reported.^{6),9)} After discrete Fourier transformation, the Fourier components were termed the first harmonic (C1), and its multiples (higher harmonics, C2 through C10), whereas C0 indicated the average height or intensity of the original radial pulse ($C_0 = 1/T \int_0^T pdt$). The frequency moduli C1 up to C10 were presented as a percentage of the average height or intensity of the pulse wave (C0) (Fig.1). Beyond C6, the variations between recordings were too small to be considered. In addition, the phase angle of each harmonic was also presented for comparison between the recordings.

Determination of effective renal plasma flow (ERPF)

At the same time as the first recording of the radial pulse, which was obtained just before intravenous infusion of SK if indicated, ERPF was also determined in the last 10 Group 1 patients by use of I^{131} -iodo-ortho-hippurate (IOH). This examination was repeated 1 week later on these patients. I^{131} -IOH 0.3 mCi, which was prepared by the Nuclear Medicine Department of this hospital, was injected intravenously into one arm and the venous blood was collected exactly 44 min later from the other arm for radioactivity counting using a fixed formula.^{10),11)} In addition, all 10 patients had their urine output, urine electrolytes, renal function and serum electrolytes monitored regularly.

Twelve other patients, who had stable coronary artery disease treated with standard antianginal therapy (e.g. nitrate, beta-blocker and calcium blocker, etc.), served as a control group to study the reproducibility of ERPF determination. At the time of the second determination, which was 1 week after the first recording, each patient was taking the same medications as at the time of the

first ERPF determination.

Statistics

All data are presented as mean value \pm one standard deviation. The Wilcoxon signed rank test was used for paired variables. The Friedman ANOVA test was initially used to assess the serial results and if there was significance, a multiple comparison was then carried out. P<0.05 was considered to be statistically significant.

RESULTS

Power spectrum analysis of radial pulse

No change could be found in the power spectrum of the radial pulse of the 24 coronary arteriography patients (Table I, group 2). Although all 18 patients admitted for coronary angioplasty manifested an improvement in exercise tolerance (from 234 \pm 28 to 346 \pm 26 sec, p<0.05) and left ventricular ejection fraction (from 43 \pm 6 to 56 \pm 8%, p<0.05) 10 days after PTCA, there was also no change in the power spectrum of the radial pulse either 3 or 10 days after intervention (Table I, group 3).

One week after PTMV, all 13 patients manifested hemodynamic improvement and increased exercise tolerance. Mitral valve area increased from 0.82 \pm 0.26 to 1.67 \pm 0.66 cm² and cardiac output increased from 3.8 \pm 1.1 to

Table I. Power Spectrum of Radial Pulses in Different Groups of Patients

Group		C0	C1	C2	C3	C4	C5	C6
1 (n = 17)	D0	3827 \pm 408	94.8 \pm 79	47.0 \pm 5	23.7 \pm 7	17.4 \pm 4	17.1 \pm 6	9.7 \pm 6
	D1	3507 \pm 329	91.9 \pm 7	54.7 \pm 8	35.7 \pm 9	18.3 \pm 4	17.4 \pm 6	11.9 \pm 4
	D2	3327 \pm 257	89.4 \pm 7	62.7 \pm 11	37.2 \pm 7	19.2 \pm 7	14.7 \pm 5	10.8 \pm 5
	Dn	3111 \pm 199*	92.9 \pm 11	69.5 \pm 15*	39.5 \pm 8*	19.7 \pm 5	15.5 \pm 5	11.4 \pm 5
2 (n = 24)	b.	3681 \pm 386	96.2 \pm 10	54.4 \pm 8	31.7 \pm 8	14.8 \pm 4	11.0 \pm 4	10.3 \pm 3
	a.	3467 \pm 489	91.7 \pm 9	54.7 \pm 7	36.1 \pm 9	16.9 \pm 8	12.7 \pm 3	9.8 \pm 3
3 (n = 18)	b.	3913 \pm 470	95.3 \pm 7	49.6 \pm 7	27.8 \pm 8	13.1 \pm 4	15.2 \pm 5	13.8 \pm 3
	a3.	3915 \pm 334	92.0 \pm 11	52.1 \pm 6	27.4 \pm 8	12.2 \pm 3	14.7 \pm 3	11.9 \pm 2
	a10.	3927 \pm 412	94.8 \pm 9	50.6 \pm 4	26.9 \pm 5	12.0 \pm 3	16.3 \pm 4	13.1 \pm 4
4 (n = 13)	b.	3693 \pm 550	94.7 \pm 13	59.7 \pm 14	33.4 \pm 12	13.5 \pm 6	7.8 \pm 4	9.0 \pm 5
	a3.	3674 \pm 532	90.5 \pm 11	56.9 \pm 9	36.4 \pm 14	17.5 \pm 12	6.9 \pm 5	7.0 \pm 2
	a10.	3684 \pm 541	93.8 \pm 10	57.7 \pm 11	37.4 \pm 10	16.8 \pm 9	7.1 \pm 3	7.8 \pm 4

Group 1: D0 immediate, D1 1 day, D2 2 days, and Dn CK back to normal after acute myocardial infarction; Group 2: b before and a after routine coronary angiography; Groups 3 and 4: b before, a3 3 days and a10 10 days after PTCA and PTMV, respectively.

* Indicates p < 0.05 as compared with the data on D0.

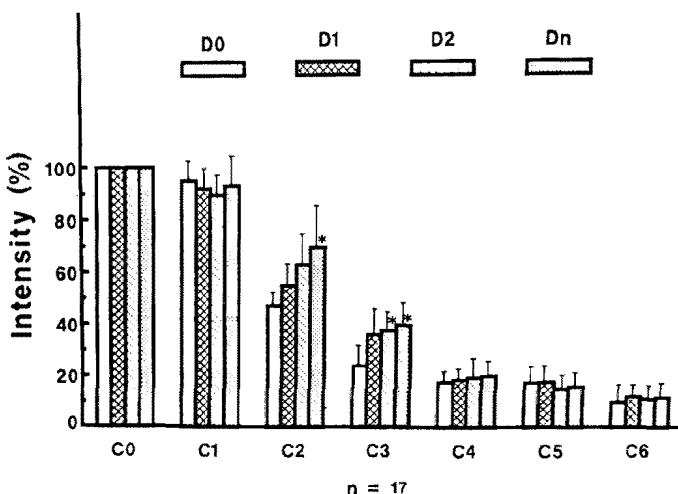


Fig. 2. Frequency moduli on various days from 17 patients with acute myocardial infarction. The average intensity of an arterial pulse [C0] is taken as 100%. The percentage intensity of frequency moduli C2 and C3 shown here is significantly lower ($*p<0.05$) than that after gradual recovery from acute myocardial infarction.

4.5 ± 1.2 L/min ($p<0.05$). However, spectral analysis of the radial pulses did not demonstrate any change 3 or 10 days after intervention (Table I, group 4).

On the contrary, the spectrum of the radial pulses in patients who had suffered from acute, uncomplicated myocardial infarction was different from the 3 above-mentioned groups. On the first recording, 14 (82%) patients registered a lower percentage intensity of the frequency moduli C2 and C3, while the C0 (the average intensity of the pulse) and remaining frequencies were not significantly different from the control groups (Table I). As the patients gradually recovered from the acute event, the percentage intensity of the frequency moduli C2 and C3 gradually increased to the same level as seen in the noninfarct patients (Table I). These readings remained stable for up to 7 days after admission (hence, only one set of data is presented here) as the serum CK cardiac enzyme returned to normal. These changes were statistically significant ($p<0.05$, Fig. 2). The average intensity of the power spectrum (C0) at the time when CK returned to normal was significantly lower than that at the first recording ($p<0.05$, Table I). However, in the serial recordings of the radial arterial pulse, the phase angle of the corresponding harmonic modulus showed no marked change with recovery.

ERPF determination

The reproducibility of ERPF determination in 12 control individuals was acceptable. The variation between the two sets of data for each patient obtained 1 week apart was 8.6% at most (range 2.0 to 8.6%) with the absolute differences ranging between 8 and 43 ml/min. The mean values of the two determinations

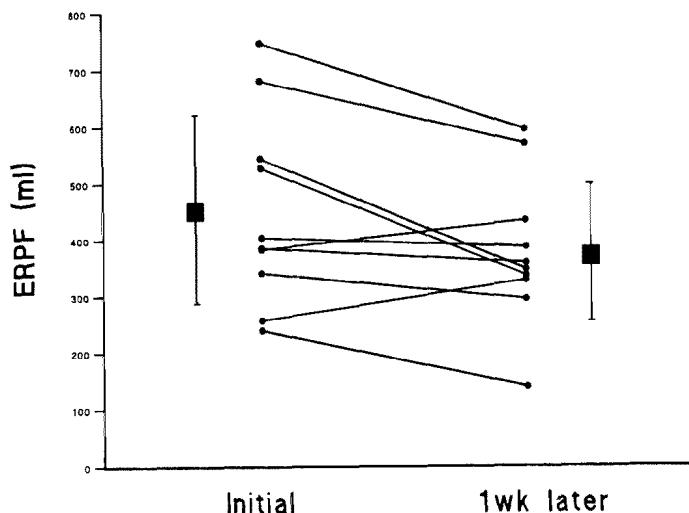


Fig. 3. Effective renal plasma flow at the time of admission, and 1 week later, in 10 patients with acute myocardial infarction.

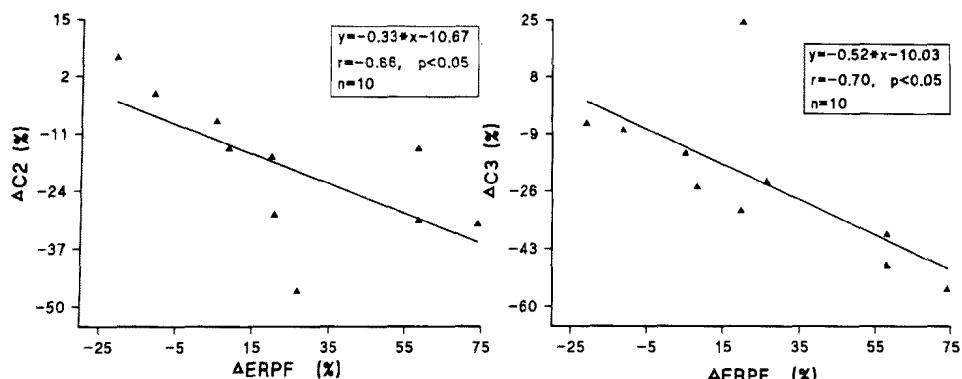


Fig. 4. This figure shows a significant inverse correlation between the change of effective renal plasma flow and the change of percentage intensity of moduli C2 and C3 in 10 patients with acute myocardial infarction.

were 457 ± 113.2 vs. 460 ± 107.2 ml/min, respectively, the difference being statistically insignificant.

Eight of the 10 patients were observed with relatively "higher" ERPF at the onset of acute myocardial infarction, with an average of 450.1 ± 168.9 ml/min, compared with the data obtained a week later (374.1 ± 130.4 ml/min) ($p < 0.02$, Fig. 3). Even though ERPF fluctuated significantly during the first week following acute myocardial infarction, renal function, including serum urea nitrogen, creatinine and uric acid, as well as serum electrolytes, was well maintained in all

10 patients. Because we selected patients with uncomplicated, acute myocardial infarction, no patient received diuretic therapy.

Correlation between ERPF and frequency moduli C2 and C3

In the 10 acute uncomplicated myocardial infarction patients, we found a significant correlation between the percentage of "initial drop" in frequency moduli C2 and C3 { $\% \text{ of difference} = [(C2 \text{ or } C3 \text{ on D0-C2 or C3 on Dn})/C2 \text{ or } C3 \text{ on Dn}] \times 100\%$ } in the power spectrum of the radial pulses and the percentage "initially higher" of the ERPF { $\% \text{ of difference} = [(initial \text{ ERPF} - second \text{ ERPF})/second \text{ ERPF}] \times 100\%$ } ($r = -0.66$ and -0.70 , respectively, $p < 0.05$, Fig. 4).

DISCUSSION

Frequency analysis of cardiovascular signals was first carried out by Aperia in 1940.¹²⁾ Since then, the calculation of the Fourier series of varied hemodynamic waveforms, e.g. femoral artery pressure gradient, arterial input impedance and flow waveforms, has been reported by several pioneer researchers.¹³⁾⁻¹⁶⁾ These offline studies emphasized the potential usefulness of frequency analysis in providing a better understanding of the dynamics of cardiac function and arterial wave transmission. Additionally, the results of the fast Fourier transform (FFT) of pressure and flow waveforms also allow the calculation of the pulsatile and steady components of fluid power and, consequently, the efficiency with which the arterial system transmits the output pressure and flow from the heart to the peripheral vascular bed.^{17),18)}

Estimation of the power spectrum of the arterial pressure has been obtained for humans¹⁹⁾ and animals.²⁰⁾⁻²²⁾ For instance, using a low-compliance, passive pressure transducer and a microprocessor-based data acquisition system, Daniels et al have demonstrated that most of the energy in the spectra obtained from the femoral arteries of anesthetized rats is concentrated in the narrowed bands corresponding to the harmonics of the cardiac and respiratory cycles.²³⁾ However, the use of a noninvasive technique to record an arterial pulse, followed by Fourier analysis, has seldom been applied in clinical practice. Nevertheless, the successful application of fast Fourier transformation (FFT) to analysis of certain hemodynamic data in patients with coronary artery disease, hypertension or peripheral vascular disease³⁾⁻⁵⁾ has provided us with the idea that the technique may also have some clinical value in monitoring patients with acute myocardial infarction.

The group of 17 myocardial infarction patients studied demonstrated no significant difference in the power spectrum analysis of the radial pulse com-

pared to non-myocardial infarction patients (in groups 2, 3 and 4) except in frequency moduli C2 and C3 (Table I). In the first recording, which was made immediately after the patient arrived at the emergency room, the percentage intensity of C2 was lower (though not statistically significant) in 14 of 17 patients with acute, uncomplicated myocardial infarction than that in non-infarct patients, whereas the frequency modulus of C3 was significantly lower ($p<0.05$). Along with a stable recovery from acute myocardial infarction, the average energy or intensity of the radial pulse (frequency modulus C0) gradually decreased, but C2 and C3 gradually increased as a percentage of intensity of C0. All changes were statistically significant ($p<0.05$, Table I and Fig. 2).

What mechanism could be responsible for the changes in the spectrum of the radial pulse? We suspect that it is unlikely to be associated with the heart. The reason is that in the group 3 and 4 patients, the power spectrum of radial pulses registered no difference after successful intervention, even though the patients manifested both clinical and hemodynamic improvement.

Examining the data from the first 7 cases of acute myocardial infarction, we noticed a consistent change in frequency modulus C2 and C3 of the spectral analysis of the radial pulse, while the average energy (C0) remained virtually constant. Two studies in the literature led us to believe that changes in renal plasma flow after acute myocardial infarction may have certain effects on specific parts of the power spectrum of radial pulses. First, Young and his colleagues conducted an animal experiment in which they consistently found different patterns of waveform in the tail artery of Sprague-Dawley rats when the left renal or superior mesenteric arteries were very briefly clamped.⁶⁾ When only the left renal artery was clamped, a significant variation above the second harmonic modulus was found, the values varying from -7.16% to $-51.0\%.$ ⁶⁾ In addition, Falicov et al demonstrated different responses to experimental myocardial infarction in dogs between renal and femoral vascular beds, viz. vasoconstriction in the femoral bed while early and persistent vasodilatation is the net renal response (evident when renal vascular resistance fell by $28\pm4\%.$)²⁴⁾ The mechanism of this renal vasodilatation is still undefined.

Furthermore, Razzak and colleagues used radioiodinated human serum albumin which was rapidly injected as a bolus into the vein to determine the renal blood flow in patients being followed up after cardiac surgery as well as those suffering from myocardial infarction.²⁵⁾ In their study, renal blood flow was found to be $1,144\pm100$ ml/min in 15 normal individuals and 2,600 ml/min in one patient with myocardial infarction, with the ratio of renal blood flow to cardiac output increasing from 18.8 to 31.2%. In 3 other nonselective myocardial infarction patients, with unknown functional class, the serial determinations of cardiac output and renal blood flow showed no consistent change during the

course of the condition.²⁵⁾

Using this information, we decided to determine the ERPF by intravenous injection of I^{131} -iodo-ortho-hippurate (I^{131} -IOH) in 10 patients with uncomplicated, acute myocardial infarction. According to Tauxe, ERPF can be readily measured by the 44-min, single-sample method which uses I^{131} -IOH. It is reported to be fast, accurate, noninvasive and readily adaptable to unusual clinical situations.^{10),11)} The method was also used to assess the renal hemodynamics before and after taking beta-blocking agents.²⁶⁾

The reproducibility of ERPF determination in 12 control individuals was acceptable. Of the last 10 patients who had ERPF determinations, 8 were observed to have relatively "higher" ERPF at the onset of acute infarction, compared with the data obtained a week later.

In these 10 acute, uncomplicated myocardial infarction patients, we found a significant correlation between the percentage of "initial drop" in frequency moduli C2 and C3 in the power spectrum of the radial pulses and the percentage "initially higher" of the ERPF ($r = -0.66$ and -0.70 , respectively, $p < 0.05$, Fig. 4). The result seemed consistent with that in Young et al's animal experiment.⁶⁾ We therefore believe that renal vasodilatation in acute but uncomplicated myocardial infarction may probably exert some influence on the arterial pulse waveform and result in a characteristic power spectrum of the arterial pulse.

What does the C0 of the power spectrum of the arterial pulse indicate? We thought that the C0 of the power spectrum indicated the average height of the power spectrum ($C_0 = 1/T \int_0^T pdt$), which we believe is directly proportional to the mean arterial pressure. According to the data obtained from the group 3 and 4 patients, C0 cannot be used to evaluate or to represent left ventricular function.

It is concluded that (1) there is a specific change in the power spectrum of the radial pulses, recorded and analyzed noninvasively, in patients with acute, uncomplicated myocardial infarction; (2) this is probably the first time that ERPF has been observed to influence the frequency moduli C2 and C3 of the power spectrum after acute, uncomplicated myocardial infarction. This observation may provide further related evidence in support of Wang et al's theory²⁷⁾ that the aorta and closely organs may cause coupled oscillation. Theoretically, this structure is equivalent to a resonance circuit. Further investigation using a much larger sample size is necessary to elucidate whether discrete Fourier transformation of radial pulses may be of additional benefit in determining the condition of the other internal organs such as the kidneys and liver in addition to the heart.

From the clinical observations of the present study, we believe that record-

ing the radial arterial pulse and performing Fourier transformation of the pulse wave may be of clinical significance in monitoring the recovery of a patient from acute myocardial infarction. Furthermore, our observations suggest that variation of the blood flow to the kidney or other internal organs (not demonstrated in this study) may affect the power spectrum of arterial pulse waves.

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