原 著

Waveform Analysis of Blood Pressure by Arterial Tonometry during Controlled Hypotensive Anesthesia with Nitroglycerin or Nicardipine

Nobuya Katoh*, Ryo Ogawa*

Abstract

Hemodynamic changes during controlled hypotension after administration of nitroglycerin (TNG) differ from the changes after nicardipine (NIC), but no previous investigations have analyzed the differences in waveforms. This study examined hemodynamic differences from radial artery wave contours obtained noninvasively by using tonometry.

Twenty patients scheduled for elective surgery, 10 who received TNG and 10 who received NIC, were studied during general anesthesia under controlled hypotension with mean arterial pressure (MAP) maintained at approximately 60 mmHg. We monitored the waveforms and recorded systolic arterial pressure (SAP), MAP and diastolic arterial pressure (DAP) and simultaneously measured central venous pressure (CVP) invasively.

The LSW ratio (peak pressure of late systolic wave-DAP/SAP-DAP), EDW ratio (peak pressure of early diastolic wave-DAP/SAP-DAP), %MAP (MAP-DAP/SAP-DAP×100) and SVI (stroke volume index determined by tonometry using the modified Warner method)[1] were calculated during five phases : phase 1 (stable point after incision)as a control, phase 2 (10 min after the start of hypotension), phase 3 (30 min after the start of hypotension), phase 4 (60 min after the start of hypotension), and phase 5 (30 min after the hypotension period), the recovery period.

Hemodynamic changes showed that DAP was higher in phase 2, 3 and 4, CVP was lower in phase 2, 3, 4 and 5 and SVI was lower in phase 3 and 4 in the TNG group than in the NIC group. With regard to the waveforms, the LSW ratio was lower in phase 2, 3 and 4 and %MAP was lower in phase 2, 3, 4 and 5 in the TNG group, but there were no differences in the EDW ratio.

It was concluded that the hemodynamic difference could be explained by the fact that the wave contour difference after administering TNG was sharper due to reduction in the late systolic wave, possibly consequent to lower SVI, decreased velocity of the reflection wave, and the difference in the dilating action sites.

Key words : Arterial tonometry, Blood pressure, Controlled hypotension, Nitroglycerin, Nicardipine, Waveform

Introduction

Controlled hypotension is used to decrease the amount of bleeding during surgery. Recently, TNG and NIC have become to be commonly used as hypotensive agents. As reported previously, hemodynamic changes during controlled hypotension differ with these two drugs[2,3], but there have been no investigations analyzing the differences from the standpoint of waveforms.

Arterial blood pressure is displayed on monitors as systolic pressure, mean blood pressure, and diastolic

*Department of Anesthesiology, Nippon Medical School, Tokyo, Japan

pressure, however, it sometimes seems impossible to understand the status of patients' blood pressure from just the blood pressure figures obtained as a result of observing blood pressure waveforms. For example, mean blood pressure values are often lower in arteriosclerosis patients than in healthy subjects, even when their systolic pressure and diastolic pressure are the same, a finding that can be explained by the fact that the waveforms have become sharper.

We, therefore, tried to clarify hemodynamic differences during controlled hypotension by investigating waveforms obtained by using tonometry, which enables measurement of the entire blood pressure waveform and determination of systolic, mean, and diastolic blood pressure noninvasively and continously[4,5], even during controlled hypotension[6]. Father, we also compared blood pressure waveforms simultaneosly obtained from the radial artery invasively and by tonometry, and investigated the similarities between them.

Materials and methods

After informed consent was obtained from each patient, we studied 20 adults scheduled for elective surgery under controlled hypotension. Their American Society of Anesthesiologists (ASA) physical status was 1 or 2, age was 43±11 yr, and body weight was 54±8 kg. Two groups were recruited at random : a group of 10 patients scheduled to undergo controlled hypotensive anesthesia with TNG, and a group of 10 patients scheduled for treatment with NIC.

All subjects were premedicated with atropine (0.01 mg/kg) and hydroxyzine (1mg/kg) intramusculary 60 min prior to surgery. Anesthesia was induced with intravenous thiamylal (5mg/kg) and the trahcea was intubated following intravenous succinylcholine (1 mg/kg). Anesthesia was maintained with nitrous oxide (67%) and isoflurane (0.5%-1.5% inspired). Controlled hypotension was induced while monitoring the patients by tonometry and maintaining MAP at approximately 60 mmHg. All patients were ventilated with the tidal volume and respiratory rate adjusted to maintain Sa_{O_2} and Pa_{CO_2} within their normal ranges.

The oscillometric blood pressure of each patient was measured bilaterally before induction of anesthesia to exclude patients with blood pressure differences between the right and left side. After induction, a 20-G Teflon cannula (Baxter, Quick-Cathe) was inserted into the left radial artery to measure invasive blood pressure (IBP) and CVP was monitored from via the internal jugular or femoral vein. Tonometry blood pressure (TBP) was monitored using JENTOW7700 (Colin) on applied over the right radial artery at the wrist. TBP, IBP, CVP, and electrocardiogram (ECC) were recorded simultaneously on a magnetic tape recorder Life Scope II (NIHON KODEN).

The LSW ratio (peak pressure of late systolic wave-DAP/SAP-DAP), EDW ratio(peak pressure of early diastolic wave-DAP/SAP-DAP) (Fig.1), %MAP (MAP-DAP/SAP-DAP×100) (Fig.2)[7], and SVI (stroke volume index using the modified Warner's method as previously reported)[1], five phases : phase 1 (stable point after incision)as a control, phase 2 (10 min after the start of hypotension), phase 3 (30 min after the start of hypotension), phase 4 (60 min after the start of hypotension), and phase 5 (30 min after the hypotension period)as the recovery period. There were calculated from the values determined during the end-expiratory pause.

Supplementary fluid, Ringer's lactate solution, was infused by intravenous drip at 10 ml/kg/hr. Hypotensive agents were administered intravenously using an infusion pump continuously. TNG was administered at a rate of 1～5 μg/kg/min and NIC at a rate of 3～5 μg/kg/min after 10 μg/kg in a bolus.

Data are shown as mean values±SD with $P<0.05$ considered statistically significant. Analysis of variance followed by Fisher's test was used for comparison of data within groups and the unpaired Student's t-test was used for comparison between groups. Linear regression analysis using Pearson's correlation coefficient was performed to assess the relationship between the values obtained by tonometry and the invasive method.

Results

Patient characteristics and surgical data are listed in Tables 1 and 2. There were no differences between the

two groups with regard to age, height, weight, consumption of isoflurane, infusion volume, blood loss, urine volume or duration of surgery (Tables 1 and 2).

Hypotension was induced smoothly within 10 min in both groups. The doses required for reduction and maintenance of MAP at 60 mmHg were TNG 4.4±2.3 μg/kg/min and NIC 3.4±1.1 μg/kg/min.

Table 1. Patient characteristics.

	Sex (Male/Female)	Age (years)	Height (cm)	Weight (kg)
NIC	2／8	39±10	160± 6	54± 7
TNG	2／8	46±14	158± 8	54±10

NIC = nicardipine, TNG = nitroglycerin. Values are expressed as means ± SD. No difference was found between the two groups in any parameter.

Hemodynamic changes are shown in Table 3. Compared to the phase 1 values, SAP were significantly lower at phases 2, 3 and 4 in both groups (by tonometry and invasively), MAP was lower in phases 2, 3 and 4 in both groups(by tonometry and invasively)and in phase 5 in the TNG group (by tonometry), DAP was lower in phases 2, 3 and 4 in both groups(by tonometry and invasively)and in phase 5 in the NIC group (by tonometry), and CVP was decreased in phases 2, 3 and 4 in the TNG group, with no difference in the NIC group. Compared to the NIC group, DAP was higher in phases 2, 3 and 4 (by tonometry and invasively), and CVP was lower in phases 2, 3, 4 and 5 in the TNG group. There were no significant differences in HR within or between

Table 2. Inhaled isoflurane, infusion volume, blood loss, urine volume, duration of surgery in the two groups.

	Inhaled isoflurane (ml)	Infusion (ml)	Blood loss (ml)	Urine volume (ml)	Duration of surgery (min)
NIC	42.3±17.4	1348±199	69±103	459±234	112±47
TNG	48.5±11.0	1401±234	29± 34	276±152	104±33

NIC = nicardipine, TNG = nitroglycerin. Values are expressed as means ± SD. No difference was found between the two groups in any parameter.

Table 3. Hemodynamic data in the control phase, during the controlled hypotension phase, and the recovery phase.

			Controlled hypotension			
		Control (Phase 1)	10 min (Phase 2)	30 min (Phase 3)	60 min (Phase 4)	Recovery (Phase 5)
SAP(mmHg)	NIC (T)	103± 6	81± 5##	79± 3##	79± 3##	101± 8
	(I)	108± 8	82± 9##	80± 3##	79± 5##	104± 7
	TNG(T)	108±12	80± 1##	82± 3##	82± 3##	104± 8
	(I)	107±11	83± 4##	83± 3##	82± 6##	110±14
MAP (mmHg)	NIC (T)	78± 6	57± 3##	55± 2##	56± 2##	72± 8
	(I)	77± 6	58± 5##	57± 3##	58± 3##	76± 5
	TNG(T)	80±10	58± 3##	58± 5##	56± 4##	75± 9#
	(I)	81±10	61± 3##	60± 5##	60± 5##	80±11
DAP (mmHg)	NIC (T)	61± 6	42± 4##	40± 3##	41± 3##	53± 7#
	(I)	62± 6	44± 6##	44± 4##	43± 3##	58± 5
	TNG(T)	60± 7	48± 5## *	47± 6## *	45± 4## *	60±11
	(I)	63± 8	51± 3## *	52± 5## **	51± 4## *	66± 9
CVP (mmHg)	NIC	5± 0	4± 1	5± 1	4± 1	5± 1
	TNG	4± 1	2± 1## *	2± 0## **	2± 0## **	3± 1*
HR (beats/min)	NIC	75± 9	79±14	78±17	79±18	80±11
	TNG	73±11	77± 9	83±16	75±14	67± 8

Values are expressed as means ± SD. SAP = systolic arterial pressure, MAP = mean arterial pressure, DAP = diastolic arterial pressure, CVP = central venous pressure, HR = heart rate, NIC = nicardipine, TNG = nitroglycerin, (T) = tonometry aid, (I) = invasive method

: $P<0.05$, ## : $P<0.01$ significant difference from the control group.

* : $P<0.05$, ** : $P<0.01$ significant difference from the NIC group.

groups.

The values for the calculated data are shown in Table 4. Compared to the control values, %MAP was significantly lower in phases 2 and 3 in the NIC group (by tonometry) and phases 2, 3, 4 and 5 in the TNG group (by tonometry and invasively), and SVI was lower in phases 2, 3 and 4 in both groups(by tonometry). Compared to the NIC group, % MAP was lower in phases 2, 3, 4 and 5 (by tonometry and invasively) and SVI was lower in phases 3 and 4 in the TNG group.

Waveform changes are shown in Fig. 1 and Table 4. TNG markedly reduced the late systolic and early diastolic wave during hypotension. Compared to the controls, the LSW ratio was lower in phase 3 in the NIC group(invasively), in phases 2, 3 and 4 in the TNG group (by tonometry and invasively) and in phase 5 in the TNG group(by tonometry). The EDW ratio was lower in phases 2, 4 and 5 in the NIC group (by tonometry), and in phases 2, 3, 4 and 5 (by tonometry) and in phase 3 (invasively) in the TNG group. Compared to the NIC group, the LSW ratio was lower in phases 2, 3 and 4 (by tonometry and invasively) and the EDW ratio was higher in phase 5 (by tonometry and invasively) in the TNG group.

Significant correlations were observed between % MAP(by tonometry) and%MAP (invasively)($r=0.60$, $P<0.0001$), the LSW ratio (by tonometry) and the LSW ratio (invasively)($r=0.86$, $P<0.0001$), and the EDW ratio (by tonometry) and the EDW ratio (invasively)($r=0.85$, $P<0.0001$)(Figs. 3, 4 and 5).

Discussion

Blood pressure must be monitored continuously during controlled hypotension with TNG or NIC. However, monitoring blood pressure invasively can cause complications, such as vascular compromise, disconnection, accidental injection, infection, and damage to nearby nerves. The use of tonometry avoids hazards related to intra-arterial cannulation, and the differences between TBP and IBP values were found to be small over a wide range of systolic, mean, and diastolic blood pressures during clinical anesthesia using vasodilator agents[6].

Therefore, in the present study we determined blood pressure values by using tonometry, but also obtained

Table 4. Calculated data in the control phase, during the controlled hypotension phase, and the recovery phase.

			Controlled hypotension			
		Control (Phase 1)	10 min (Phase 2)	30 min (Phase 3)	60 min (Phase 4)	Recovery (Phase 5)
LSW ratio	NIC (T)	0.72±0.05	0.71±0.04	0.70±0.06	0.70±0.06	0.66±0.11
	(I)	0.73±0.06	0.70±0.07	0.68±0.07#	0.70±0.07	0.65±0.11
	TNG(T)	0.76±0.05	0.54±0.07## **	0.54±0.07## **	0.50±0.07## **	0.61±0.11#
	(I)	0.74±0.08	0.54±0.10## **	0.54±0.08## **	0.49±0.08## **	0.64±0.16
EDW ratio	NIC (T)	0.30±0.05	0.23±0.06#	0.23±0.08	0.23±0.06#	0.25±0.05#
	(I)	0.31±0.08	0.23±0.08	0.23±0.08	0.23±0.07	0.26±0.06
	TNG(T)	0.30±0.04	0.23±0.07##	0.20±0.07##	0.24±0.06##	0.38±0.06## **
	(I)	0.31±0.04	0.24±0.08	0.21±0.07##	0.26±0.07	0.41±0.12**
%MAP	NIC (T)	41.0 ±3.2	39.9 ±5.6#	37.5 ±2.6#	38.9 ±4.2	38.6 ±3.4
	(I)	38.7 ±2.7	37.2 ±5.5	38.3 ±3.4	41.6 ±5.5	38.2 ±3.3
	TNG(T)	40.9 ±4.9	30.8 ±2.8## **	32.2 ±3.3## **	30.6 ±3.3## **	33.6 ±2.6## *
	(I)	39.7 ±5.4	30.6 ±4.5## *	31.7 ±3.9## *	28.7 ±4.3## **	31.5 ±4.2## *
SVI	NIC	32.4 ±3.1	29.5 ±2.6##	29.3 ±2.7##	29.2 ±2.1##	31.7 ±2.4
	TNG	32.2 ±3.1	26.7 ±3.1##	26.6 ±2.5## *	26.0 ±2.8## *	30.9 ±3.4

Values are expressed as means ± SD. LSW ratio = peak pressure of the late systolic wave - DAP / SAP - DAP, EDW ratio = peak pressure of the early diastolic wave - DAP / SAP - DAP, % MAP = MAP - DAP / SAP - DAP × 100, SVI = stroke volume index by the modified Warner method with tonometry, (T) = tonometry aid, (I) = invasive method.

: $P<0.05$, ## : $P<0.01$ significant difference from the control group.

* : $P<0.05$, ** : $P<0.01$ significant difference from the NIC group.

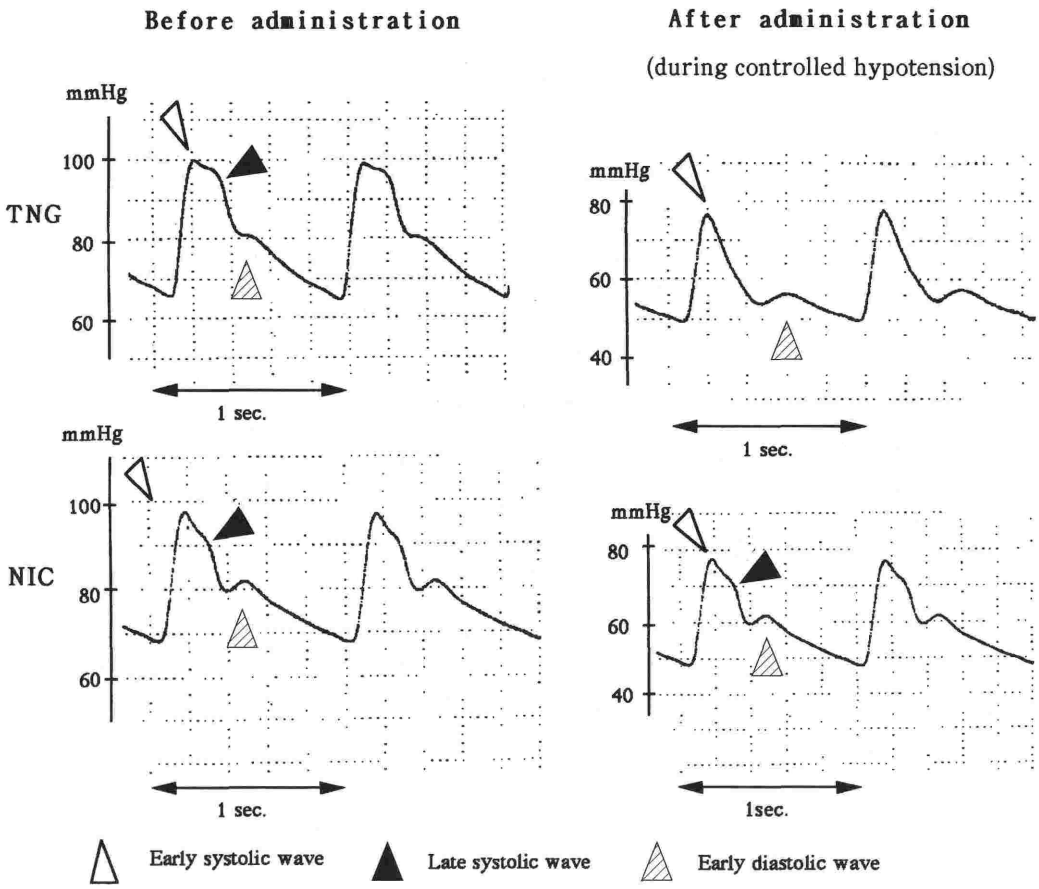

Fig. 1 Explanation of radial pressure wave contour obtained by tonometory before and after hypotensive agents were administered. The early systolic waves were generated by the heart, and the late systolic and the early diastolic waves were caused by peripheral wave reflections. TNG reduced the amplitude of the late systolic waves. NIC=nicardipine, TNG=nitroglycerin

them invasively, and assessed similarities in relation to the shapes of the two waveforms.

Arterial tonometry, a noninvasive technique, was invented in 1963[4]. Tonometry has been improved and eventually leading to an instrument called JENTOW. The tonometric method is applied to a superficial artery lying over a bone structure and thus it is often applied to the radial artery at the wrist. The transducer does not measure absolute pressure but a voltage signal that varies with the level of pressure, and therefore, the transducer must be caliblated by using an oscillometric cuff placed on the same side of the arm 5 minutes intervals for 30 seconds. The wrist, where the tonometric sensor is attached, must be fixed because it is very sensitive to movement artifacts. The tonometry sounded an alarm because of movement artifacts during recording, measurments were performed again after recalibration.

MAP was smoothly reduced to approximately 60 mmHg within 10 min after the start of infusion of TNG or NIC in all patients. Both resistance and capacitance vessels were dilated, TNG has a major effect on the latter[8]. Because the capacitance of the venous circulation increases markedly with TNG, stroke volume may decrease if preload is compromised (Table 3)[9]. The correlation coefficient between the stroke volumes calculated by the Warner method from the arterial waveform obtained noninvasively by to-

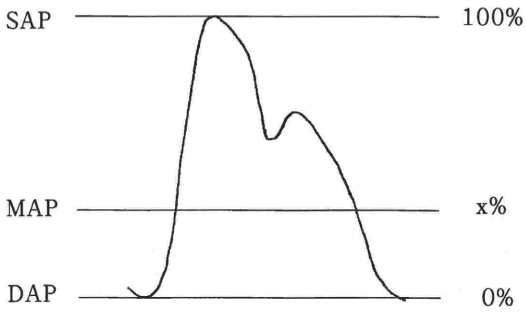

Fig. 2 %MAP=MAP-DAP/SAP-DAP×100
SAP= systolic arterial pressure, MAP = mean arterial pressure, DAP=diastolic arterial pressure

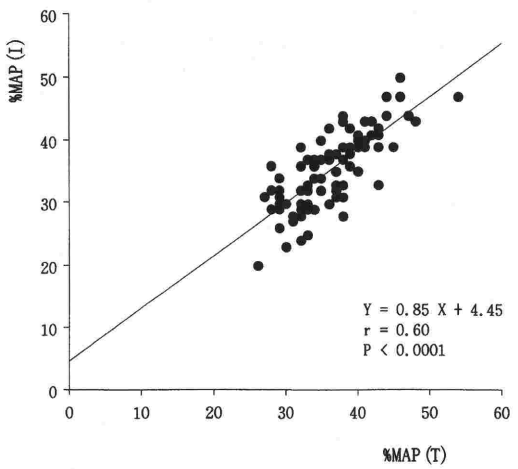

Fig. 3 Relationship between the %MAP by tonometry (T) and the invasive method (I). A linear correlation was observed.($y=0.85\chi-4.45$, $r=0.60$, $P<0.0001$). %MAP =MAP-DAP/SAP-DAP×100, SAP = systolic arterial pressure, DAP=diastolic arterial pressure, MAP = mean arterial pressure.

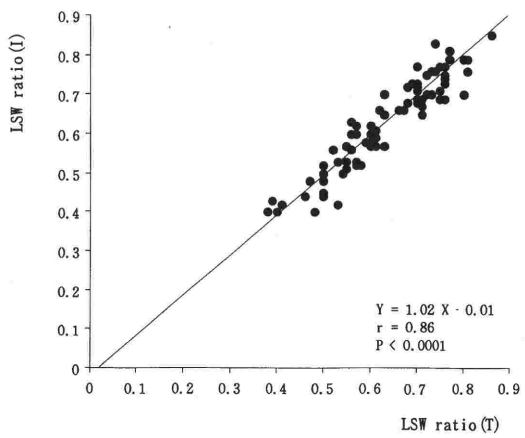

Fig. 4 Relationship between the LSW ratio by tonometry (T) and the invasive method (I). A linear correlation was observed.($y=1.02\chi-0.01$, $r=0.86$, $P<0.0001$). LSW ratio=peak pressure of the late systolic wave-DAP/SAP-DAP, SAP = systolic arterial pressure, DAP = diastolic arterial pressure.

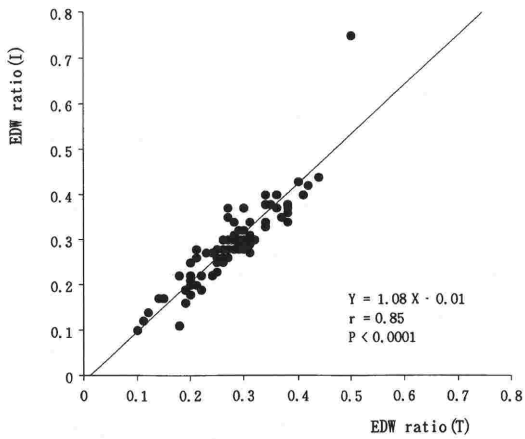

Fig. 5 Relationship between the EDW ratio by tonometry (T) and the invasive method (I). A linear correlation was observed.($y=1.08\chi-0.01$, $r=0.85$, $P<0.0001$). EDW ratio =peak pressure of the early diastolic wave-DAP/SAP-DAP, SAP=systolic arterial pressure, DAP= diastolic arterial pressure.

nometry and the stroke volumes obtained by the thermodilution method was good ($r=0.878$), and lower in the TNG group than in the NIC group (Table 4). On the other hand, NIC is a calcium channel blocking drug that dilates resistance vessels, causing lower DAP than TNG while maintaining stroke volume without tachycardia[11]. The effect of reduced stroke volume may be offset by an increase in sympathetic nervous activity elicited though barorecepter activity and resulting in increased heart rate. However, in both groups the adrenergic response might be partially blocked by isoflurane[12]. None of the patients complained of headache, abdominal pain, diarrhea, arrhythmias, or other known side effects of TNG or NIC infusion in the recovery room after general anesthesia.

When the changes in hemodynamics induced by the two drugs during controlled hypotension were compared, the decrease in SAP in the TNG group was similar and DAP was significantly higher (Table 3). Thus it was assumed that MAP in the TNG group would also be significantly higher. However, because the difference was found not to be significant, we decided to investigate the difference in hemodynamics in the two groups by comparing the waveforms.

To explain the degree of sharpness of the waveforms, we used %MAP calculated as follows: %MAP =MAP-DAP/SAP-DAP× 100 (Fig. 2). MAP was shown on the tonometry display with SAP and DAP as average pressure after integrating through three cardiac cycles. SAP, DAP, and MAP obtained by the invasive method were calculated the same way and displayed simultaneously on a magnetic tape recorder (NIHON KODEN, Life Scope II). This parameter, %MAP, is lower when the waveform is sharper (Fig. 6). Lower% MAP during hypotension and recovery in the TNG group indicated that the waveforms during administeration of TNG were sharper, and have a more slender shape (Fig. 1).

Arterial waves are made up of a forward travelling and backward travelling waves. The former are generated by the heart, and the latter are caused by peripheral wave reflection. TNG markedly reduced the amplitude of the backwared travelling (reflected) waves, but had little effect on the foward travelling waves[13]. Thus, the change in contour of the TBP waves during and after administration of TNG could be explained on the basis of decreased peripheral wave reflections. The effect of decreased peripheral wave reflection was revealed as reduction in the amplitude of the peak of the late systolic wave (Fig.1), causing lower %MAP and a sharper wave contour.

The problem, however, was to explain the reduction in the late systolic waves in the TNG group. There seemed to be three reason. First, low stroke volume produced a low-amplitude reflection, so that lower SVI after administering TNG (Table 4) made lower late systolic waves and sharper waveforms. Second, the amplitude of the reflection waves depend on the travelling velocity from peripheral to central. If the reflection waves return early, they would produce late systolic waves, and if late, early diastolic waves (Fig. 1). In the presence of high blood pressure or arteriosclerosis, which accelerate the velocity of the

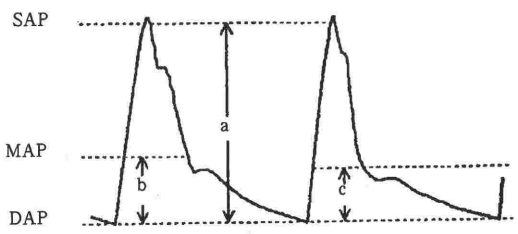

Fig. 6 %MAP(MAP-DAP/SAP-DAP×100)is an indicator of sharpness on waveform. This parameter decreases when the waveform is sharper. %MAP=b/a>%MAP=c/a, SAP= systolic arterial pressure, MAP = mean arterial pressure, DAP=diastolic arterial pressure.

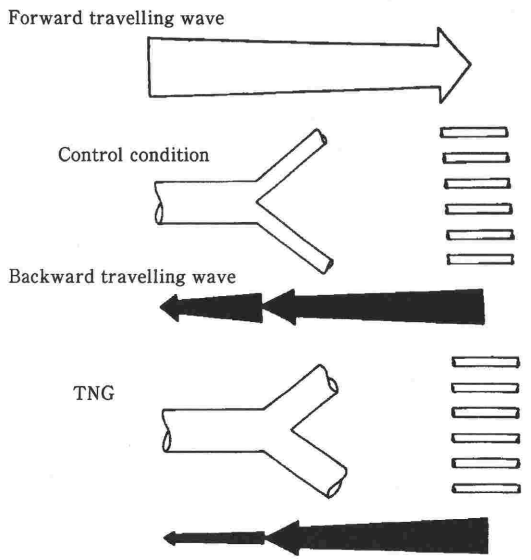

Fig. 7 The proposed mechanism for reduction in peripheral reflection coefficient with nitroglycerin. The forward travelling wave is shown above, reflected at the peripheral arteriols (right). The backward travelling reflected waves (dark arrows) are shown in the top panel under control conditions, and in the bottom panel after nitroglycerin. Wave reflection in the peripheral arterioles is unchanged, but the amplitude of the reflected wave observed centrally is decreased by increased re-reflection at the junction of the parent artery with disproportionally dilated daughter branches[13].

reflected waves, the late systolic waves are higher and the early diastolic waves are lower[14,15]. The lower LSW ratio and absence of a statistical difference in EDW ratio after administering TNG (Table 4)may mean that TNG decreased the velocity of the reflection waves. Third, we can explain by the difference in dilating action sites between NIC and TNG. The former acts on arterioles, while the latter acts on daughter segments (small arteries, not arterioles) at each branching[16]. The changes in the caliber of arterial daughter segments reduced wave reflections at the normally well-matched junction points and created a type of negative reflection, so that less of the reflected wave returned from the peripheral arterioles[13] (Fig. 7). The smaller DAP (mostly defined as peripheral resistance)change in the NIC group compared to the TNG group during controlled hypotension is also attributable to the difference in sites of action.

It would be desirable to determine the relationship of systolic blood pressure between central and peripheral arteries. Wave reflections have different effects on the contour and amplitude between at the central aortic and carotid pulse, and at the brachial and radial pulse. In the proximal aorta and carotid artery, the late systolic pressure wave is responsible for the pressure peak, whereas in the brachial and radial arteries, wave reflection from the lower part of the body causes a fluctuation during the descending limb of the pulse, not the peak. Thus, the reduction in wave reflection reduces the late systolic wave and alters wave contour, but usually dose not affect peak systolic pressure in peripheral arteries[17,18]. Consequently, controlled hypotension with TNG reduces wave reflections, and thus may reduce central peak systolic pressure to a far greater degree than is apparent from measurements of peripheral pressure. It is possible that SAP in the central arteries is lower during administeration of TNG than NIC because of less reduction of reflection waves in the NIC group. We ordinarily measure blood pressure in the brachial artery with a cuff, or invasively by inserting canula in the radial artery, however, if possible, it seems that it would also be easy to estimate changes in blood pressurre waveforms at more central sites, e. g., the aorta, carotid arteries, and coronary arteries, by observing peripheral artery waveforms.

All these studies appear to indicate more beneficial effects with TNG than NIC in regard to left ventricular afterload, myocardial oxygen demands, and left ventricular function. Since the pressure of the late systolic wave reaches the left ventricle[19], the decrease in the late systolic wave is considered useful in avoiding excessive left ventricular afterload. Furthermore, TNG maintains the early diastolic wave with a slighter decrease than NIC, and these hemodynamic changes with TNG may be useful in maintaining coronary perfusion pressure.

Tonometric systolic, diastolic and mean values were very similar to the invasive systolic, diastolic, and mean values (Table 2). Furthermore, as shown in Fig. 3, 4, and 5, transcutaneous tonometric waveforms were similar to those obtained by the intra-artrial method.

In conclusion, in clinical practice, it is important not only to measure SAP, MAP, and DAP but to monitor the wave contour as well.

References

1) Katoh N, Wajima Z, Ogawa R, et al : Quantitation of cardiac output from noninvasive arterial pulse contour (Tonometry) in man. Circ Cont 16 : 53-58, 1995
2) Terashima, M : Haemodynamic effects of vasodilators in dogs. -a comparison of prostagrandin E_1, nicardipine and nitroglycerin. Masui (Jpn J Anesthesiol) 42 : 173-184, 1993
3) Fahmy NR : Nitroglycerin as a hypotensive drug during general anesthesia. Anesthesiology 49 : 17-22, 1978
4) Pressman G, Newgard P : A transducer for continuous external measurement of arterial blood pressure. IEEE Trans Biomed Electro 10 : 73-81, 1963
5) Kemmotsu O, Yokota S, Yamamura T, et al : J. Eckerle. A noninvasive blood pressure monitor based on arterial tonometry (abstract). Anesth Analg 68 : S145, 1989
6) Kemmotsu O, Ueda M, Otsuka H, et al : Blood pressure measurement by arterial tonometry in controlled hypotension. Anesth Analg 73 : 54-58, 1991
7) Watanabe H, Nanba H, Murahara Y, et al : Continuous non-invasive blood pressure wave analysis by dP/dt and % MAP with tonometric manometer. Japanese journal of clinical monitoring 5 : 169-173, 1994
8) Alad B, Mellanger S : Comparative effects of hydralazin, sodium nitrite and acetylcholine on resistance and capacitance blood vessels and capillary filtration in skeletal muscle

in the cat. Acta Physiol Scand 58 : 319-329, 1963
9) Mason DT : Afterload reduction and cardiac performance phisiologic basis of systemic vasodilators as a new approach in treatment of congestive heart failure. Am J Med 65 : 106-125, 1978
10) Campbell BC, Kelman AW, Hillis WS : Noninvasive assessment of the haemodynamic effects of nicardipine in normotensive subjects. Br J Clin Pharmacol 20 : 55S, 1985
11) Kishi Y, Okumura F, Furuya H : Haemodynamic effects of nicardipine hydrochoride. Br J Anaesth 56 : 1003-1006, 1984
12) Hysing E, Chelly J, Doursout M, et al : Cardiovascular effects of and interaction between calcium blocking drugs and anesthetics in chronically instrumented dogs. III. Nicardipine and isoflurane. Anesthesiology 65 : 385-391, 1986
13) Yaginuma T, Avolio A, O'Rourke M, et al : Effect of glyceryl trinitrate on peripheral arteries alters left ventricular hydraulic load in man. Cardiovasc Res 20 : 153-160, 1986
14) Nye E : The effect of blood pressure alteration on the pulse wave velocity. Brit Heart J 26 : 261-265, 1964
15) Bjurulf P : Atherosclerosis in different parts of the arterial system. Am Heart J 68 : 41-50, 1964
16) O'Rourke M, Kelly R, Avolio A, et al : Effects of arterial dilator agents on central aortic systolic pressure and on left ventricular hydraulic load. Am J Cardiol 63 : 381-441, 1989
17) Kelly R, Gibbs H, O'Rourke M, et al : Nitroglycerin has more favourable effects on left ventricular afterload than apparent from measurement of pressure in a peripheral artery. Eur Heart J 11 : 138-144, 1990
18) Salans A, Katz L, Graham G : A study of the central and peripheral arterial pressure pulse in man : correlation with simultaneously recorded electrokymograms. Circulation 4 : 510-521, 1951
19) Takazawa K : A clinical study of the second component of left ventricular systoric pressure. J Tokyo Med Coll 45 : 256-270, 1987

(Circ Cont 18 : 226～234, 1997)