COMPARISON AMONG ARTERIAL GRAFTS AND CORONARY ARTERY

An attempt at functional classification

Various arterial conduits have been used for coronary artery bypass grafting. However, arterial grafts are not uniform either in anatomy or in function. Some conduits are more spastic than others and there may be possible differences in long-term patency rates. The diverse biologic characteristics promote a necessity of classification of arterial grafts, which may facilitate the understanding of surgeons of biologic characteristics of various arterial grafts and provide a scientific basis for searching for new grafts. Another important issue is the comparison of reactivity between arterial grafts and coronary arteries. In this study, we aim to compare the pharmacologic reactivity among the human arteries (grafts and coronary arteries) and to classify arterial grafts. Segments of three arterial grafts (gastroepiploic, internal mammary, and inferior epigastric) taken from patients undergoing coronary artery bypass grafting and coronary arteries taken from explanted hearts were studied in organ baths for the contraction to four vasoconstrictors (endothelin-1, thromboxane A₂ mimetic U46619, full adrenoceptor agonist norepinephrine, and depolarizing agent potassium) under physiologic pressure. The diameter of the four arteries at a pressure of 100 mm Hg was similar ($p > 0.05$). However, the gastroepiploic artery contracted to higher forces (9.41 ± 2.0 gm for endothelin, 11.79 ± 1.85 gm for U46619, 13.54 ± 2.7 gm for norepinephrine, and 11.11 ± 1.97 gm for potassium) than did the coronary artery and internal mammary artery ($p < 0.05$) for all the tested vasoconstrictors and higher than the inferior epigastric artery for potassium and norepinephrine ($p < 0.05$). There was no significant difference among the other three arteries (internal mammary artery, inferior epigastric artery, and coronary artery) regarding the maximal contraction force to any vasoconstrictor. No difference was detected in regard to the sensitivity (effective concentration causing 50% of the maximal response) to the vasoconstrictors among the four arteries. This study reveals that among the arterial grafts and the coronary artery, the gastroepiploic artery has the highest contractility to various vasoconstrictors. On the basis of our findings and physiologic and embryologic knowledge we propose a classification for arterial grafts: type I (somatic arteries), type II (splanchnic arteries), and type III (limb arteries). Types II and III are prone to spasm because of higher contractility whereas type I arteries are usually less spastic. This classification may have important clinical implications for the understanding of arterial graft spasm or patency and may be useful in the search for new grafts. (J THORAC CARDIOVASC SURG 1995;109:707-15)

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Because of superior long-term results of internal mammary artery (IMA) grafting, various arterial conduits have been used for coronary artery bypass grafting (CABG).¹⁻⁴ Such conduits include the radial artery,⁵ gastroepiploic artery (GEA),⁶ inferior epigastric artery (IEA),^{7,8} splenic artery,^{9,10} and subscapular artery.¹¹ However, although long-term patency rates for the IMA are well established, there are only a few reports on other arterial conduits with a relatively small number of patients involved.¹²⁻¹⁴ It

is expected that other arterial conduits will have good long-term results as well. This kind of expectation is based on a hypothesis that all arterial conduits have similar biologic characteristics such as contractility, relaxing characteristics, endothelial function, and anatomic structure. However, studies have shown that there are differences among these arterial conduits regarding contractility.^{15, 16}

Histologic studies have also revealed that there are major differences among various grafts in terms of structure of smooth muscle and elastic lamellae.¹⁷ The differences observed from these studies suggest that arterial grafts, although all are arteries, are not uniform either in anatomy or in function. These differences are the anatomic and physiologic basis of the divergent clinical manifestations of the grafts and may also account for possible differences in long-term patency rates. One of the diverse manifestations clinically observed is a different tendency among these arterial grafts to develop spasm during surgical dissection and during the perioperative period. It is the experience of many surgeons that the GEA has a higher tendency to spasm than the IMA.¹⁸ Similarly, spasm of the radial artery is a serious problem that, together with a low patency rate that may be also related to this characteristic of the artery, led to the abandonment of this arterial graft at an early stage in the 1970s.¹⁹ Only after the development of a method to overcome spasm of this arterial graft has it recently been readopted for use.²⁰ The diverse biologic characteristics promote a necessity for classification of arterial grafts, which may facilitate the understanding of surgeons of biologic characteristics of various arterial grafts and provide a scientific basis for the search for new grafts.

Another important issue is the comparison between arterial grafts and coronary arteries. There are obvious reasons for this. As conduits for the coronary artery, the grafts and the coronary artery form a new blood-carrying system after operation. Lesions at any part of this system would affect coronary artery flow early after operation and perhaps also long-term patency. Systemic administration of drugs, in particular vasoactive agents, would affect both arterial grafts and coronary arteries. The net effect of these agents will depend on the effect of the agent on each part of the new system.

In this study, we aim to classify arterial grafts by comparing pharmacologic reactivity of the grafts and to compare the function of the grafts to that of the human coronary artery.

Material and methods

Human GEA, IEA, and IMA segments were collected from patients undergoing CABG with use of these grafts. Approval to use discarded GEA, IEA, and IMA tissue was given by the Hospital Human Ethics Committee. Human coronary arteries were taken from explanted hearts of patients undergoing heart transplant. After sternotomy, a full-length left IMA pedicle was carefully dissected from the chest wall. Either the left or the right IEA or the right GEA was harvested as a free graft. The required length for IMA, IEA, or GEA was carefully measured. Any discarded segments were collected and put into a container with oxygenated physiologic (Krebs) solution maintained at 4° C and then transferred to a laboratory immediately. The IEA, GEA, and IMA were transferred into a glass dish and dissected out from their surrounding connective tissue. The arteries were cut into 3 mm long rings, and the rings were then suspended on wires in organ baths.²¹⁻²³ The coronary artery rings used for the pharmacologic study were only taken from coronary arteries (large epicardial arteries) that did not show any gross atherosclerosis. The Krebs solution had the following composition (in millimoles per liter): Na⁺ 144, K⁺ 5.9, Ca²⁺ 2.5, Mg²⁺ 1.2, Cl⁻ 128.7, HCO₃⁻ 25, SO₄²⁻ 1.2, H₂PO₄⁻ 1.2, and glucose 11. The solution was aerated with a gas mixture of 95% O₂ and 5% CO₂ at 37° ± 0.1° C.

Organ-bath technique. An organ-bath technique that allowed normalization of vascular rings to a physiologic condition *in vitro* was used to set the vascular rings at a pressure comparable to that of the *in vivo* situation. The details of the technique were published before.^{21, 22} Briefly, each arterial ring was stretched in progressive steps to determine the individual length-tension curve. A computer iterative fitting program (VESTAND by Yang-Hai He, Princeton, University) was used to determine the exponential line, the pressure and the internal diameter. When the transmural pressure on the rings reached 100 mm Hg, determined from their own length-tension curves, the stretch procedure was stopped and the rings were then released to 90% of their internal circumference at 100 mm Hg. This degree of passive tension was then maintained throughout the experiment.

We intentionally preserved the endothelium by cautiously dissecting and mounting the rings. This technique allowed the experiments to be done with an intact endothelium, as determined by the functional relaxation response to acetylcholine.^{21, 24}

Protocol. After the normalization procedure the vascular rings were equilibrated for 45 minutes. The following protocols were designed for the experiments.

Diameters of the IEA, GEA, IMA, and coronary artery at a pressure of 100 mm Hg (D100) and resting tensions at 0.9 D100 (optimal point of the length-tension curves) were recorded from the normalization procedure. The cumulative concentration-contraction curve was randomly established for the following vasoconstrictor substances: endothelin-1; U46619, a stable thromboxane A₂ (TxA₂) mimetic; membrane depolarizing agent potassium chloride (K⁺); and norepinephrine, a full adrenoceptor agonist. Because norepinephrine induced a weak contraction in the coronary artery, in some coronary artery rings β -adrenoceptor antagonist propranolol (1 μ mol/L) was

Table I. Comparison of the diameter at 100 mm Hg and the resting force in various human arteries

	Coronary artery (n = 24)	IMA (n = 57)	IEA (n = 24)	GEA (n = 28)	p Value
D100 (mm)	2.40 ± 0.12	2.04 ± 0.06	2.20 ± 0.12	2.07 ± 0.12	NS
Resting force (gm)	3.65 ± 0.26	3.20 ± 0.19	2.32 ± 0.1	1.71 ± 0.17*	<0.0001

Data given as mean plus or minus standard error of mean. D100, Diameter at 100 mm Hg; NS, not significant.

*p < 0.01 compared with the coronary artery, IMA, or IEA (Scheffe's F test).

Table II. Comparison of the maximal contraction and the EC₅₀ of arterial grafts and the coronary artery to various vasoconstrictors

	Maximal contraction (gm)				p Value	EC ₅₀ (-log M)*				p Value
	CA	IMA	IEA	GEA		CA	IMA	IEA	GEA	
ET	3.73 ± 0.51 (n = 7)	4.38 ± 0.54 (n = 14)	4.88 ± 0.69 (n = 6)	9.41 ± 2.0†‡	0.002	8.15 ± 0.13	8.22 ± 0.09	8.22 ± 0.04	8.06 ± 0.01	NS
U46619	3.78 ± 0.58 (n = 6)	6.84 ± 0.74 (n = 15)	8.04 ± 1.23 (n = 6)	11.79 ± 1.85†‡	0.001	7.74 ± 0.08	8.56 ± 0.2	8.25 ± 0.09	8.44 ± 0.24	NS
K ⁺	2.62 ± 0.58 (n = 5)	5.59 ± 0.72 (n = 15)	5.30 ± 0.87 (n = 6)	11.11 ± 1.97†‡§	0.0006	10.21 ± 2.87	20.19 ± 1.72	14.24 ± 1.9	15.17 ± 3.5	NS
NE	0.4 ± 0.31 (n = 6)	3.75 ± 0.51 (n = 13)	5.13 ± 0.67 (n = 6)	13.54 ± 2.70†‡§	<0.0001	6.35 ± 1.32	5.90 ± 0.1	6.59 ± 0.18	6.36 ± 0.11	NS

CA, Coronary artery; ET, endothelin; NE, norepinephrine; NS, not significant. p > 0.05 among coronary artery, IMA, and IEA in regard to endothelin, U46619, and norepinephrine (Scheffe's F test).

*Concentration for K⁺ is in millimoles per liter.

†p < 0.05 compared with CA.

‡p < 0.05 compared with IMA.

§p < 0.05 compared with IEA, Scheffe's F test.

added 30 minutes before the concentration-contraction curve to norepinephrine was established.

Analysis of variance (and Scheffe's F test as post-hoc test) was used to compare the contraction force and effective concentration causing 50% of the maximal response (EC₅₀)^{21, 26} for each vasoconstrictor among the four arteries. A value of p < 0.05 was considered significant. The standard error of the mean was used throughout.

Drugs. Drugs used in this study and their sources were as follows: (-)-norepinephrine bitartrate (Sigma, St. Louis, Mo.); U46619 (Cayman Chemical, Ann Arbor, Mich.); endothelin-1 (Peptides International, Louisville, Ky.). Stock solution of norepinephrine was freshly made each day. Stock solution of endothelin-1 and U46619 was held frozen until required.

Results

The number of vascular rings studied is listed in Table I, which also lists the diameter and the passive resting force for the arterial conduits and for the coronary artery. The diameter of the four arteries at a pressure of 100 mm Hg was similar (p > 0.05). However, the resting force for the GEA was significantly lower than that for other vessels and this

reflects the different compliance of this artery compared with that of others.

Table II gives the maximal contraction force and the EC₅₀ values for endothelin, U46619, K⁺, and norepinephrine in the four arteries. There was a significant difference among the four arteries for all these four vasoconstrictors regarding the maximal contraction. In particular, the GEA contracted with all the vasoconstrictors tested to higher forces than those measured in the coronary artery and the IMA (p < 0.05, Scheffe's F test) and also higher than those in the IEA for potassium and norepinephrine (p < 0.05, Scheffe's F test). There was no significant difference among the other three arteries (IMA, IEA, and coronary artery) regarding the maximal contraction force to either vasoconstrictor. In regard to EC₅₀, there was no difference detected (p > 0.05) among the four arteries. The average concentration-contraction curves for these vasoconstrictors are shown in Figs. 1 through 4. Figure 4 also demonstrates that when β-adrenoceptor function is blocked by propranolol (1 μmol/L), the coronary

Fig. 1. Mean concentration ($-\log M$)-contraction (force) curves for endothelin-1 (endothelin), in GEA, IEA, IMA, and coronary artery (CA). Symbols represent data averaged from at least six rings for each vessel (see Table II for number of rings). Vertical bars are 1 standard error of mean.

artery contracts with norepinephrine to a significantly higher level (Fig. 4, b).

Discussion

Although comparisons between the IEA and IMA and between the GEA and IMA have been made, the results are contradictory. Dignan and associates¹⁵ have found that the GEA is more reactive to K^+ , norepinephrine, and 5-hydroxytryptamine, whereas others have suggested that the GEA and IMA have similar responses to norepinephrine,²⁷ phenylephrine, and 5-hydroxytryptamine²⁸ and that the IMA is more reactive to TxA_2 mimetic U46619.²⁷ The diverse results from different groups may reflect the variation of technology used in the studies. In fact, results of in vitro pharmacologic studies of blood vessels at large depend on the set-up condition of the vessels. For example, at various resting forces, the vessel may have different reactions to vasoactive substances. Therefore to set the vessels at an optimal point of its own length-tension exponential curve may mimic the in vivo pressure and make a comparative basis for different vessels.^{21, 22} From this point of view, to compare the pharmacologic reactivity of various arterial conduits in one laboratory has a particular advantage and accuracy. With such a basis, the functional compar-

Fig. 2. Mean concentration ($-\log M$)-contraction (force) curves for TxA_2 (U46619) in GEA, IEA, IMA, and coronary artery (CA). Symbols represent data averaged from at least six rings for each vessel (see Table II for number of rings). Vertical bars are 1 standard error of mean.

ison and classification of grafts are attempted in our present study.

The present study, because of the technology used, also provides information on vascular diameter at a comparable basis. Judgment of the diameter of arterial grafts could be difficult because this parameter at large depends on intramural pressure. The diameter measured at zero pressure, as in autopsy, is different from that in the in vivo situation. Similarly, even at in vivo status the diameter is changeable with the pressure change in a certain range. Therefore without pressure measurement the measurement of the diameter does not provide valid information. With use of a special technology²¹⁻²⁵ that allows measurement of the individual length-tension (diameter-pressure) curve, we are able to compare the diameter of the arterial grafts and coronary arteries at a comparable basis, that is, at a pressure of 100 mm Hg. From the present study, the average diameter of these vessels at 100 mm Hg was 2.04 mm for IMA, 2.2 mm for IEA, 2.07 mm for GEA, and 2.4 mm for coronary artery ($p > 0.05$, Table I). These measurements demonstrate suitability of various arterial grafts as conduits for the coronary artery as far as the size is concerned.

In previous studies, although numerous vasoconstrictors have been used to investigate the reactivity of the grafts, the reactivity of various arterial grafts to endothelin has not been compared. Endothelin

Fig. 3. Mean concentration ($-\log M$)-contraction (force) curves for potassium chloride (K) in GEA, IEA, IMA, and coronary artery (CA). Symbols represent data averaged from at least five rings for each vessel (see Table II for number of rings). Vertical bars are 1 standard error of mean.

has been proposed as the most potent vasoconstrictor known.²⁹ An elevated plasma level of endothelin has been measured during cardiopulmonary bypass.³⁰ Therefore this vasoconstrictor may have a pathogenic significance in vasospasm related to cardiac operation. Other vasoconstrictors tested in the present study were U46619, norepinephrine, and K^+ . Norepinephrine was selected for the present study because it is a full adrenoceptor agonist and an important neurotransmitter. Its effect on the human IMA has been extensively studied.^{22-25, 31-34} The TxA_2 mimetic U46619 was used in this study because this vasoconstrictor, derived from platelets, is a potent agonist in the IMA and IEA. An elevated plasma level of TxA_2 during cardiopulmonary bypass, similar to endothelin, has been found.^{35, 36} In addition, the cellular membrane depolarizing agent potassium ion (K^+) was also used to study contractility.

The reactivity of various blood vessels is different. In a previous study,²¹ we have demonstrated a divergence of pharmacologic reactivity among the canine IMA, the saphenous vein, and the coronary artery. In general, saphenous veins and coronary arteries are more reactive compared with the IMA. However, such a comparison regarding reactivity has not yet been made in human vessels. One of the major aims of the present study, therefore, was to compare and classify arterial grafts according to their reactivity. Our results demonstrate that among the four arteries (three arterial grafts and the coro-

nary artery) the GEA is the most reactive artery. This is shown by the fact that this artery is the most reactive one in response to all the four vasoconstrictor substances tested. The maximal contraction force to these vasoconstrictors was 9.41 gm for endothelin, 11.79 gm for U46619, 11.11 gm for K^+ , and 13.54 gm for norepinephrine. Because the length of the vascular segment was 3 mm for each artery, these contraction forces reflect tension developed by each unit of vascular segment and are comparable among the arteries. Our results regarding contractility to TxA_2 are different from those of others. O'Neil and associates²⁷ has found that the IMA is more reactive to TxA_2 than the GEA is. Conversely, in the present study, we have found that although TxA_2 is a potent vasoconstrictor in all the four arteries, it produced the highest force in the GEA, as did the other three vasoconstrictors. This uniform characteristic of the GEA clearly demonstrates that this artery is the most reactive artery among the arteries tested. This is in accordance with our clinical observations and those of others¹⁸ that the GEA is prone to vasospasm during surgical dissection.

Other arteries, that is, the IEA, the IMA, and the coronary artery, are similar regarding the maximal contraction to endothelin, U46619, or K^+ ($p > 0.05$). In regard to norepinephrine, the contraction force of coronary arteries is slightly ($p > 0.05$, Scheffe's F test; Table II) less than that of the IMA or IEA. However, this does not simply mean that

Fig. 4. **a**, Mean concentration ($-\log M$)–contraction (force) curves for norepinephrine in GEA, IEA, IMA, and coronary artery (CA). **b**, Comparison of contraction curves for coronary artery with ($n = 6$) or without ($n = 4$) β -adrenoceptor antagonist propranolol ($1 \mu\text{mol/L}$). Coronary artery incubated with propranolol for 30 minutes had significantly higher contraction ($p < 0.05$, unpaired t test on maximum contraction). Symbols represent data averaged from group of vessel (see Table II for number of rings in each group). Vertical bars are 1 standard error of mean.

coronary arteries are less reactive to norepinephrine. Norepinephrine is a full agonist for adrenoceptors, which have α and β types. It is well known that the smooth muscle of coronary arteries contains both receptors and that α -adrenoceptors mediate contraction whereas β -adrenoceptors mediate relaxation. Norepinephrine stimulates both α - and β -adrenoceptors in coronary arteries and therefore the net effect is little contraction. When the β -adrenoceptor is blocked the α -adrenoceptor-mediated

contraction will be unmasked. This has been demonstrated by previous studies³⁷ and also by the present study. Fig. 4, *b* demonstrates that when the human coronary artery is incubated with the β -adrenoceptor antagonist propranolol ($1 \mu\text{mol/L}$) the α -adrenoceptor-mediated contraction is unmasked. Therefore contraction force to norepinephrine is markedly enhanced. In contrast, the human IMA is an artery with predominant α -adrenoceptors.²⁴ From the similar reaction of the IEA to norepineph-

rine, and even more enhanced contraction in the GEA, we may suspect that these two arteries are also α -adrenoceptor predominant and may also have weak function of β -adrenoceptors. However, this is to be studied.

As mentioned previously, coronary arteries are highly reactive vessels as demonstrated in the dog.²¹ In the present study, we could not demonstrate that the human coronary artery was more reactive than other arteries such as the IMA or IEA. In fact, in our experiments there was a tendency that the reactivity of the human coronary artery was lower than that of other arteries. This may be partially because the explanted hearts, from which the coronary arteries were taken, were from patients undergoing heart transplant. The cause of the heart disease was disregarded, and some of those patients had coronary artery disease. Although the specimens were taken from the coronary arteries without obvious atherosclerosis, these arteries could have had pathologic changes that might reduce the reactivity.

The results from the present study may be used to classify arterial grafts. From anatomic considerations, other splanchnic (visceral) arteries, such as the splenic artery, may be similar to the GEA although no data are available yet. Somatic arteries located in the body wall such as the subscapular artery or the intercostal artery may be similar to the IMA. As already demonstrated, the IEA is similar to the IMA as far as contractility is concerned,³⁸ and both are somatic arteries. As for the radial artery, this artery is located in the distal part of the arm and therefore is more spastic, as arteries at the extremities are usually prone to spasm (as seen in Raynaud's disease). On the basis of these anatomic considerations and the results of our present study, we propose a classification for arterial grafts (Table III). This classification suggests that there are three types of arterial grafts. Type I is composed of somatic arteries, whereas type II are splanchnic arteries and type III are limb arteries. Type II arteries are prone to spasm because of the higher contractility of splanchnic arteries. This characteristic of splanchnic arteries has a physiologic meaning that blood flow through splanchnic arteries is subject to tremendous changes under various circumstances in accord with the function of the alimentary tract. The flow increases after meals and decreases under critical situations. In contrast, type I arterial grafts (somatic arteries) are less reactive than type II grafts because they are mainly conduit arteries except at the end of the artery, which is a muscular

Table III. Functional classification of arterial grafts on the basis of contractility; the contractility of the coronary artery is also compared

	Type I (somatic arteries)*		Type II (splanchnic arteries)†	Type III (limb arteries)	Coronary artery‡
	IMA	IEA	GEA	Radial artery	
ET	+++	+++	++++	§	+++
U46619	+++	+++	++++	+++	+++
NE	+++	+++	++++	+++	+++
KCl	+++	+++	++++	++++	+++

Grade from + to ++++ (+ is the least). Both the maximal contraction (+ to +++) and EC_{50} (+) are taken into account. The grade is based on the present study except that for radial artery (from reference 16). In general, the contractility is on the order of splanchnic arteries > limb arteries $\geq$ coronary arteries $\geq$ somatic arteries. ET, Endothelin; NE, norepinephrine.

*Intercostal arteries and subscapular arteries may also belong to the type I group because of anatomic and embryologic considerations, although there are no pharmacologic data available.

†Because of anatomic and embryologic considerations, splenic arteries may belong to the type II group. However, no data on the contractility of these arteries are available, and this is still speculative.

‡Coronary artery contracts with norepinephrine to a low force because this artery has strong β -adrenergic function (see Fig. 4, b).

§No data available.

||The reactivity of coronary artery in the present study may be underestimated because the arteries taken from explanted hearts may have coronary disease that could reduce the reactivity. In our canine experiments, coronary artery is highly reactive to K^+ and U46619.²¹

regulator for blood flow, as recently demonstrated in the human IMA.³²⁻³⁴ Type III arteries are located in limbs, represented by the radial artery, and have higher tendency for spasm compared with somatic arteries (type I).

Although our classification is based on pharmacologic studies and anatomic considerations, there is evidence that it is in accordance with embryologic factors.³⁹ From embryologic considerations, somatic arteries are intersegmental branches to the body wall. These arteries persist in the thoracic and lumbar regions as the posterior intercostal, subcostal, and lumbar arteries. Near the anterior median line intersegmental arteries are linked by a ventral somatic anastomosis. These vessels persist bilaterally as the internal mammary artery and the superior and inferior epigastric arteries. In our classification, these are the type I arteries. In contrast, type II arteries (splanchnic arteries) are from segmental branches of primitive dorsal aorta to supply the digestive tube. The dorsal splanchnic anastomosis persists in the GEA. The arteries of the upper limbs (type III in our classification), such as the radial artery, are also developed from intersegmental arteries as somatic arteries, although the arteries of

the lower limb arise from the dorsal root of the umbilical artery.

This classification may have an implication in regard to the search for new arterial grafts. As far as vasospasm is concerned, type I arteries may be less spastic than type II and type III arteries. The IMA is a typical example of a type I artery. Although the IMA is also a reactive artery, vasospasm is encountered less in this artery than in type II or type III arteries. This is true in particular when its most reactive portion, the distal section, is trimmed off.^{32, 40} The less spastic characteristics are obviously advantageous perioperatively. In fact, in common clinical practice today, the IMA is used as the first choice of arterial grafts. In general, the correlation between contractility and long-term patency is still unclear. Other factors, particularly the structure of the endothelium, may also contribute to long-term patency. A recent report on the long-term patency rates of the GEA suggests an equal patency rate for this arterial graft to that for the IMA,¹⁴ although the follow-up was not as long as that for the IMA. At the least it is clear that higher contractility of arterial grafts, which means a tendency to reduce the diameter of the artery, is a disadvantage for the maintenance of coronary artery flow perioperatively and may necessitate pharmacologic treatment.

The reason to classify arterial grafts on the basis of contractility rather than on the basis of relaxation is that although vascular reactivity is composed of contractility and relaxation, the latter depends on the contractility. Relaxation occurs only in an artery that is already contracted and therefore is secondary to existing contraction. Although various vasodilators may have different relaxing effects on arterial grafts and one particular vasodilator may also have different effects on different arteries, these differences are often vasoconstrictor dependent, that is, contraction dependent.^{23, 25, 31} Therefore we have attempted to classify the arterial grafts on the basis of reactivity to vasoconstrictors (contractility) rather than on the basis of relaxation.

In the present study, no attempt was made to compare arterial grafts or coronary arteries with venous grafts because it is well known that there are marked differences between arterial and venous grafts regarding contractility⁴¹ and long-term patency.¹⁻⁴

In summary, the present study reveals that among the arterial grafts and the coronary artery, the GEA has the highest contractility to various vasoconstrictors. On the basis of our findings and the anatomic,

embryologic, and physiologic knowledge we have proposed a classification for arterial grafts: type I (somatic arteries), type II (splanchnic arteries), and type III (limb arteries). Types II and III are prone to spasm because of higher contractility whereas type I arteries are usually less spastic. This classification may have important clinical implications for the understanding of arterial graft spasm or patency and may be useful in the search for new grafts.

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