Heart Vessels (1995) 10: 106–110

Heart and Vessels


# Rheological significance of tandem lesions of the coronary artery

Yasumi Uchida, Fumitaka Nakamura, Takanobu Tomaru, Atsuko Miwa, and Toshihiro Morita

Second Department of Internal Medicine, University of Tokyo, 3-1, Hongo 7-chome, Bunkyo-ku, Tokyo 131, Japan

**Summary.** It is not known whether the individual lesions that constitute tandem lesions of the coronary artery are developmentally or rheologically related. Luminal changes and their rheological significance were examined by percutaneous angioscopy in 44 tandem lesions of 21 patients with ischemic heart disease. Angioscopically, individual narrowing of angiographically documented tandem lesions appeared only as a tangentially expressed prominent portion of an atherosclerotic spiral fold. The directions of the fold were counterclockwise in the proximal to middle segments and clockwise in the distal segment of the right coronary artery, clockwise in the proximal to middle segments and counterclockwise in the distal segment of the left anterior descending artery, and counterclockwise in the proximal to middle segments of the left circumflex artery. The bloodstream always ran along the spiral folds in the tandem lesions. The results suggest that angiographically documented tandem coronary lesions are merely a tangential expression of atherosclerotic spiral folds and that they may act to prevent blood turbulence by generating a spiral laminal flow.

**Key words:** Percutaneous coronary angioscopy – Tandem lesions – Spiral folds – Bloodstream – Rheological significance

## Introduction

When examined angiographically the stenotic coronary arteries in patients with ischemic heart disease exhibit various configurations [1, 2]. Of these configurations, stenoses arranged in series, which simulate a tandem, are called "tandem lesions." Similar to solitary segmental lesions, tandem lesions are believed to interfere with the blood flow by multiplying the resistance of the individual lesions and by generating flow turbulence [3]. However, there has been no definitive evidence of these phenomena in humans. The three-dimensional structure of the individual stenoses that constitute the tandem lesions, the structural interrelationship of the individual lesions, and their definitive rheological significance are not known. To our knowledge, no one has ever suggested the existence of any rheologically beneficial structural interrelationship of the individual stenoses that constitute the tandem lesions.

Percutanous angioscopy has enabled ramins investigators to observe luminal changes of the coronary artery directly, and this procedure has provided much new macroscopic pathological information on the coronary luminal changes, which would not otherwise have been obtainable [4–7]. In a previous study, during angioscopy, we observed spiral folds in angiographically smooth and non-stenotic, as well as in stenotic coronary segments, and we suggested their possible rheological significance; however, we had no definitive evidence [7].

In this study, using percutaneous angioscopy, we examined the spacial structure and rheological significance of angiographically documented tandem coronary lesions.

## Patients and methods

### Patients

Between March, 1990 and March, 1993, 189 patients with ischemic heart disease underwent percutaneous coronary angioscopy during routine cardiac catheterization and coronary angiography. Of these 189 patients, 21 had tandem lesions (less than 75% stenosis; two or more lesions in one segment) in at

*Address correspondence to*: Y. Uchida
Received September 1, 1994; accepted October 1, 1994.

least one coronary artery. Lesions in which stenosis exceeded 75% were excluded, since insertion of the fiberscope or the guiding catheter across them may have caused damage that could have resulted in acute occlusion. The 21 patients (16 men and 5 women) ranged in age from 38 to 76 years; 9 patients had angina pectoris and 12 had old myocardial infarction.

### *Angioscopy procedures*

The angioscopy system consisted of a 1.7-French fiberscope (Olympus Co. Ltd., Tokyo, Japan), a 5-French inner guiding balloon catheter devised in our laboratory, a 9-French outer guiding catheter, a CCD camera, an illumination source, a videoconverter, a videorecorder, and a monitor. The guiding balloon catheter had three lumens, one for the fiberscope, one for a guide wire, and one for a saline flush [2]. Because of the experimental nature of the investigation, informed consent for the angioscopy was obtained from all patients. After routine coronary angiography, the 5-French inner guiding balloon catheter was introduced through the 9-French outer guiding catheter into the desired coronary segment over a 0.014 or 0.018 guide wire. The balloon was then inflated with carbon dioxide to stop the blood flow, and 5–10 ml of saline (including heparin 10 IU/ml) at body temperature was then infused manually at 0.5–1 ml/s to displace the blood, to allow for observation of the luminal changes. During the saline infusion, the fiberscope was advanced or pulled back for serial observation. After each observation, the balloon was deflated to observe the laminal stream formed by the blood draining into the saline-filled coronary segment. After the final observation of artery, contrast material (Iopamidol) was injected into the segment through the guiding balloon catheter to confirm the location of the tip of the fiberscope.

The angiographic and angioscopic images were compared to clarify their dimensional interrelationship.

### *Statistical analysis*

Statistical analysis was performed using Fisher's exact probability test; when the $P$ value was below 0.05, the difference was considered significant.

## Results

Figure 1 shows tandem lesions in the proximal segment of the left anterior descending artery in a patient with angina pectoris. Angiographically, there were at least six narrowings in series from the proximal to middle segments. Serial angioscopic observation revealed a clockwise spiral fold, but no other demonstrable changes in these segments. The individual stenoses identified by angiography were correlated with those observed by angioscopy. The individual narrowing was shown to be a tangential expression of the spiral fold.

The direction of the spiral folds that constituted the tandem lesion was counterclockwise in all 9 of the proximal to middle segments, and clockwise in 5 of the 6 distal segments of the right coronary artery; clockwise in 9 of the 12 proximal to middle segments and counterclockwise in 6 of the 7 distal segments of the left anterior descending artery; and counterclockwise in 7 of the 8 proximal to middle segments of the left circumflex artery (Table 1).

In four patients in whom all three coronary arteries exhibited tandem lesions, the direction of the spiral fold was counterclockwise in all four proximal to middle segments in the right coronary artery, but was clockwise in three of four proximal to middle segments of the left anterior descending artery and counterclockwise in three of the four segments in the middle segment of the left circumflex artery. Thus, the directions of the spiral folds differed among the three arteries and between the proximal to middle and the distal segments.

The laminal flow was observed immediately after deflation of the balloon in each case. The flow ran along the spiral fold in the tandem lesions (Fig. 2).

## Discussion

Tandem lesions of the coronary artery have been believed to disturb the coronary circulation, both by multiplying the resistance caused by individual stenoses and by generating flow turbulence [3]. In this study, the majority of tandem lesions with less than

**Table 1.** Direction of spiral folds in angiographically documented tandem lesions

| | Right coronary artery | | Left anterior descending artery | | Left circumflex artery | |
|---|---|---|---|---|---|---|
| | Prox to Mid | Dist | Prox to Mid | Dist | Prox to Mid | Dist |
| Clockwise | 0/9<br>$P < 0.01$ | 1/6<br>$P < 0.05$ | 9/12<br>$P < 0.01$ | 1/7<br>$P < 0.05$ | 1/8<br>$P < 0.01$ | 1/2 |
| Counter-clockwise | 9/9 | 5/6 | 2/12 | 6/7 | 7/8 | 1/2 |

*Prox*, Proximal; *mid*, middle; *Dist*, distal

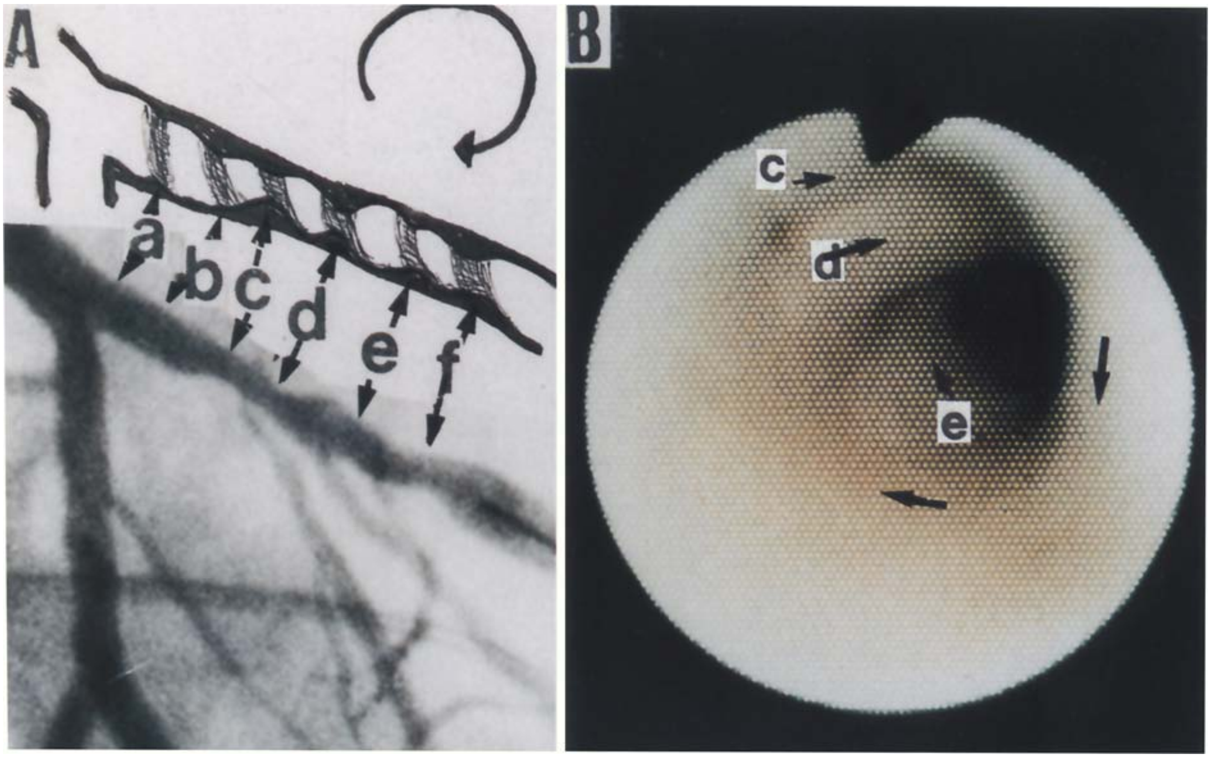


**Fig. 1A,B.** Tandem lesions in the proximal to middle segment of the left anterior descending artery in a patient with angina pectoris. **A** Angiographic features of the artery at the right anterior oblique projection and schematic representation, based on angioscopic features. Each angiographic narrowing corresponds to the fold labeled with the same letter in the schema. The direction of the fold toward downstream is shown by the *circular arrow*. **B** Angioscopic features of the tandem lesions, showing yellow clockwise spiral folds. *Arrows* show direction of the spiral folds. The folds, labeled *c*, *d*, and *e*, correspond to the narrowings labeled with the same letters as in **A**

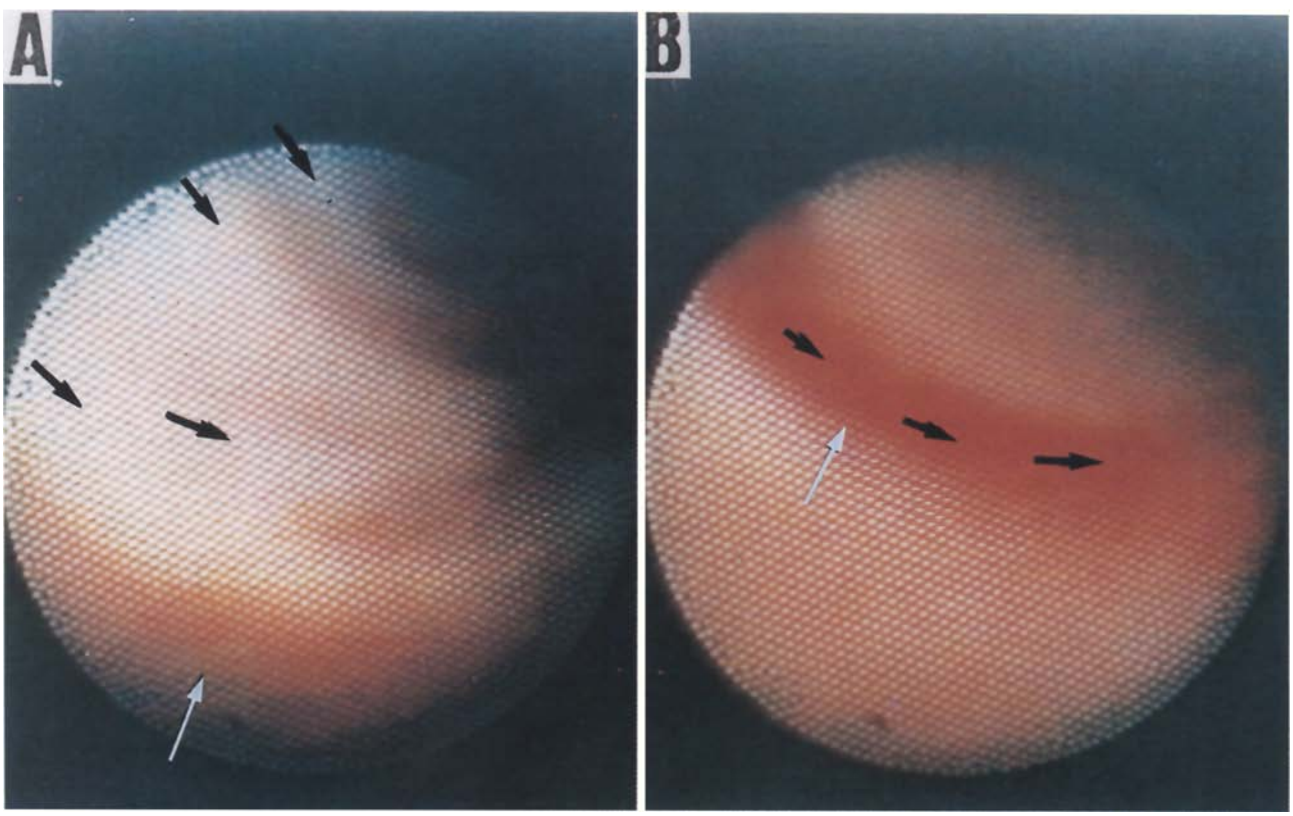


**Fig. 2. A** Counterclockwise spiral folds in the proximal segment of the right coronary artery in a patient with angina pectoris (*larger black arrows*). Laminal flow and its direction are labeled with the *white arrow* and *smaller black arrows*, respectively. **B** The same laminal flow, moved to a distal fold (*white arrow*). *Black arrows* show direction of the laminal flow

75% stenosis were found to be composed of spiral folds and the individual tandem or irregular narrowing was seen to be, merely, a tangentially expressed shadow of a spiral fold. In addition, the laminal blood flow always ran along the spiral fold.

Since it was possible that the guiding balloon catheter located upstream of the lesion could have caused the spiral laminal flow, we carried out an experiment to determine whether a spiral flow was present in the absence of a catheter. We perfused a human coronary artery removed during autopsy with Krebs-Henseleit solution and drained canine arterial blood into the perfusion circuit to produce a laminal flow, using a method reported previously [8]. A spiral blood flow was observed along the spiral fold, irrespective of the presence or absence of the catheter.

The heart is twisted during each beat and generates rotation in the ejected blood [9]. Doppler ultrasound measurement has shown that blood appeared to rotate in the ascending [10] and descending aorta [11]. Angioscopy also revealed a spiral flow in the peripheral arteries [12]. Thus, it seems that, as a result of cardiac motion and the anatomy of the arterial system, a spiral by patterned blood flow occurs from the heart to small vessels, including the coronary artery.

Shear stress on the vascular wall modifies the endothelial function and causes a rearrangement of the endothelial cells [13] and their stress fibers along the shear stress [14], and can even cause damage of the endothelial cells. The shear stress enhances the release of vasoactive substances, such as prostacycline, from the endothelium [15] and enhances the uptake by the endothelium of substances such as low-density lipoprotein [16]. Deposition of lipids occurs in regions of flow separation and in regions with low shear stress, resulting in atherosclerotic stenosis [17]. Therefore, it is likely that spiral laminal flow induces spiral shear stress, which, in turn, accelelates spiral atherosclerosis, resulting in the formation of spiral folds in the coronary artery. This theory is supported by the finding that even angiographically smooth and non-stenotic coronary segments frequently exhibited shallow spiral folds [7]. It is likely that such shallow spiral folds grow into angiographically demonstrable tandem lesions.

The direction of the spiral fold differed among the three major coronary arteries. The changes in secondary flow patterns also probably differed among the three coronary arteries, leading to the different directions of the spiral folds. The direction of the spiral fold in the distal segment was opposite to that seen in the proximal to middle segments. In the coronary artery, the major inflow occurs during diastole, while backflow occurs during systole.

This backflow may have modified the direction of the spiral fold in the distal segment. Except during interventional therapy, such as percutaneous transluminal coronary angioplasty or laser angioplasty [4, 18], it is our policy not to observe lesions with significant stenosis (exceeding 75%), since passing the fiberscope or guiding catheter across such a lesion may damage it, leading to acute occlusion. Therefore, we were unable to determine whether lesions with significant stenosis also exhibited spiral folds.

Angioscopically, coronary lesions are classified as regular and complex plaques and thombus [2]. Complex plaques exhibit an irregular shape angiographically [1], and show cleft, dissection, and rupture, with or without thrombus, angioscopically [2, 5]. In patients with unstable angina pectoris, the culprit lesions are frequently complex [2, 5]. In addition to the complex plaque itself, disruption of the spiral fold by the complex plaque may result in blood flow turbulence, which, in turn, may further accelelate thrombosis [19].

Thus, the results indicate that, angiographically, tandem coronary lesions are merely a tangential expression of atherosclerotic spiral folds whose directions differ among the three major coronary arteries and between the proximal to middle and distal segments; further, the spiral folds may act to prevent, rather than to cause, turbulence of the blood flow by generating a spiral laminal stream.

## References


1. Ambrose JA, Winters SL, Fuster M (1986) Angiographic evolution of coronary artery morphology in unstable angina pectoris. Am Coll Cardiol: 427–432
2. Uchida Y (1992) Percutaneous coronary angioscopy. Jpn Heart J 33:271–294
3. Robbins SL, Bentov I (1967) The kinetics of viscous flow in a model vessel: Effects of stenoses of varing size, shape and length. Lab Invest 16:864–874
4. Uchida Y, Hasegawa K, Kawamura K, Hasegawa K, Furuse A (1989) Angioscopic observation of coronary luminal changes induced by percutaneous transluminal coronary angioplasty. Am Heart J 117:1153–1155
5. Sherman CT, Litvak F, Grundfest W (1989) Coronary angioscopy in patients with unstable angina pectoris. New Engl J Med 315:913–919
6. Spears JR, Spokojny AM, Mariais HJ (1985) Coronary angioscopy during cardiac catheterization. J Am Coll Cardiol 6:93–97
7. Uchida Y, Tomaru T, Nakamura F, Hasegawa K (1987) Percutaneous coronary angioscopy in patients with ischemic heart disease. Am Heart J 114:1216–1222
8. Uchida Y, Tomaru T, Sumino S, Kato S, Sugimoto T (1986) Fiberoptic observation of thrombosis and thrombolysis in isolated human coronary artery. Am Heart J 112:694–696
9. Karini T, Goldsmith HL, Motomiya M (1987) Flow patterns in vessels; simple and complex geometries. In: Leonard EF, Turrito WT, Vroman L (eds) Blood in contact with natural and artificial surfaces. Academic, New York, pp 422–441
10. Segadal L, Matre K (1987) Blood velocity distribution in the human ascending aorta. Circulation 76:90–100

11. Frazinh L, Lanza G, Mehlman D (1990) Rotational blood flow in the thoracic aorta (abstract). Clin Res 38:311A
12. Stonebridge PA, Brophy CM (1991) Spiral laminal flow in arteries? Lancet 338:1360–1361
13. Silkworth JB, Stehbens WE (1975) The shape of endothelial cells in en face preparations of rabbit blood vessels. Angiology 26:474–487
14. Franke RP, Graffe M, Schnittler H (1984) Induction of human vascular endothelial stress fiber by fluid shear stress. Nature 307:648–649
15. Frangos JA (1985) Flow effects on prostacycline production by cultured human endothelial cells. Science 227: 1477–1479
16. Spraque EA (1987) Influence of a steady-state fluid-imposed wall shear stress on the binding, internalization and degradation of low density lipoproteins by cultured arterial endothelium. Circulation 76:648–656
17. Caro CG, Fitz-Gerald JM, Schroter RC (1971) Atheroma and arterial wall shear: Observation, correlation and proposal for a shear-dependent mass transfer mechanism for atherogenesis. Proc R Soc Lond [Biol] 117:109–159
18. Nakamura F, Kvasnicka J, Uchida Y, Geschwind HJ (1992) Percutaneous angioscopic evaluation of luminal changes induced by excimer laser angioplasty. Am Heart J 124:1467–1472
19. Ridolfi R, Hutchins GM (1977) The relationship between coronary artery lesions and myocardial infarcts: Ulceration of atherosclerosis plaques precipitating coronary thrombosis. Am Heart J 93:468–486