**Editorial Comment**

**Same fur, different animals**

Lan Nguyen, 1 Priscilla Wan, 2 Thach Nguyen3

1 BS in Aeronautics, University of Texas in Dallas, Dallas TX, USA
2 University of Texas in Dallas, Dallas TX, USA
3 Director of Cardiology, Community Health System, St Mary Medical Center, Hobart IN, USA

Slow flow is not an uncommon phenomenon for cardiologists. It can happen in many clinical scenarios, requires different tools of investigation and responds to various modalities of treatment.

In the interventional laboratories: During percutaneous coronary interventions (PCI), no-reflow is defined as stagnant contrast agent in the distal vasculature without apparent proximal obstruction. The incidence is 2% with plain balloon angioplasty (PTCA), 7% in patients undergoing rotational atherectomy, 12% for primary angioplasty, and much higher at 42% for PCI of degenerated saphenous vein graft (SVG). The causes are mainly due to embolization of atheromatous material (gruel) and aggravated by microembolization of platelet-rich thrombi that release vasoactive agents (e.g., serotonin), causing intense arteriolar vasospasm in the distal vasculature. The mortality of patients who developed no-reflow was 8%.1

The differential diagnosis of an apparent no-reflow phenomenon is dissection or acute thrombotic formation in the proximal segment, which is not well appreciated by conventional angiography. If in doubt, a transport or infusion catheter can be inserted through the wire and advanced to the distal segment of the no-flow area. Then the wire is removed. Pressure gradient is measured, and contrast injection through the end-hole will help to make the distinction between no-reflow or proximal obstructive lesions. Then injection of 3–5 ml of contrast agent with slow withdrawal of the catheter into the guide is useful to reveal any proximal disease, however hemodynamically insignificant.2

The treatment includes forceful injection of blood through the guide in order to raise driving pressure across the capillary bed. Another approach is to inject small boluses of nitroglycerin (100–200 μg: a very quick try) and/or calcium channel blockers (100–200 μg of verapamil) or adenosine (12–18 μg). Verapamil is effective in 67% of cases in alleviating arteriolar spasm and restoring antegrade flow. Nitroprusside 40 μg bolus up to 100–200 μg can also be given with action to be seen in 2 minutes.3 Epinephrine can be given especially in patients with hypotension. The dosage ranges between 50–200 μg and multiple doses can be given and adjusted according to the presence and severity of hypotension.4 It is important to deliver these agents into the distal artery through a balloon catheter or drug delivery catheter.

In the office: When talking to a clinical cardiologist, slow flow has a different meaning. A patient can have typical angina, has an abnormal stress test, so undergoes a coronary angiogram. The angiographic results could show slow flow without any severe stenosis. This patient is having endothelial dysfunction which slows the coronary flow. The underlying pathology is diffuse plaques from coronary artery disease. The favorite treatment is angiotensin-converting enzyme inhibitor (ACEI) or angiotensin receptor blocker (ARB) to sustain a healthy endothelium. However the definitive treatment is statin to convert the cholesterol-laden soft plaques into fibrotic stable plaques.

In the emergency room: The presentation of slow flow has a different meaning. A patient can have typical angina, with ST segment elevation on the electrocardiogram and troponin level positive. The emergency cardiac catheterization laboratory team is called in and the on call interventional cardiologist is informed to arrive to do primary intervention. The angiographic results could show slow flow without any severe significant stenoses as reported by Lin et al. in this issue of the Journal of Geriatric Cardiology. All the major branches should be accounted to be patent [left anterior descending (LAD), left circumflex (LCX), right coronary (RCA), first and second diagonals, first and second obtuse marginals (OM), posterior descending artery (PDA) and posterior lateral branch (PLB)]. The treatment is short term vasodilation, glycoprotein 2B3A inhibitor to dissolve any possible micro-thrombi in the distal microvasculature. The long term treatment includes statin, ACEI (to sustain a healthy endothelium) and antiplatelet agent (aspirin or clopidogrel) to prevent any future microscopic thrombotic formation.

In the electrophysiologic services: A patient with typical angina is given beta-blocker. Because of an abnormal stress test, the patient is arranged to have a coronaryangiogram. During the procedure, the heart rate is persistent in the low 30s. The angiogram shows slow flow without any
other abnormalities. This phenomenon is seen clearer in the femoral angiogram. The flow almost stops between the ventricular contractions. The explanation is that the blood flow is phasic. It advances with systolic contractions. In a normal rate (above 40) the flow looks continuous. However, as the diastolic pause is too prolonged, then during diastole, the peripheral flow stops. While in the heart, during systole, with the contraction of the ventricle, all of the microvascular system in the myocardium is squeezed and shut so the contrast in the epicardial arteries looks stagnant (or slow flow). It happens more commonly in the elderly patients with diastolic dysfunction. The management would be avoiding severe bradycardia. If the patient is asymptomatic, then there is no need for treatment. The slow flow phenomenon is physiologic.

Goals of treatment: The goal of the treatment of slow flow during PCI is to achieve a Thrombolysis in Myocardial Infarction (TIMI) 3 flow. This flow would guarantee the lowest possibility of subacute arterial thrombosis (SAT) by assuming that there is no significant residual stenosis (or obstructive flow) at the just stented proximal segment.

In the case of the patient with AMI, slow flow without significant lesion as in the reported by Lin et al. in this issue of the Journal of Geriatric Cardiology, the treatment would include short term anticoagulant, anti-platelet drug and long term statin and ACEI or ARB. In the case of patient with stable CAD, the goal is to improve the endothelial function by ACEI or ARB. In the case of an incidental finding of slow flow during bradycardia, then avoidance of bradycardia is suggested or no treatment is needed if the patient is asymptomatic. Intuitively, ACE inhibitor to improve the endothelial health is suggested.

Dilemma in the treatment: Can slow flow happen forever? If a patient has AMI and arrives late to the hospital, the endothelium could be irreversibly damaged forever. Slow flow is refractory to all forms of treatment including venous or arterial vasodilators (NTG, calcium channel blocker, adenosine, nitroprusside) or vasoconstriction (epinephrine). In a case seen by one of the authors, the patient had a third myocardial infarction in 1 month after electing not to take clopidogrel for drug-eluting stent. Slow flow was obvious during the third primary PCI and was still seen persisting in a follow-up coronary angiogram 5 years later.

Same fur, different animals: The slow flow phenomenon is a commonly seen problem. The treatment varies according to the causes. The majority of the problems could be solved for a better result. In rare cases, it is impossible to restore the health of the endothelial layer and the slow flow would persist during the lifetime of the patient. Slow flow is a common denominator of various different conditions causing endothelial dysfunction. Because of its many etiologies, even the slow flow looks the same, the treatment has to be different.

References