Persistent Catheter-induced Coronary Artery Spasm

The induction of coronary artery spasm during coronary artery catheterization is a relatively common occurrence. In most cases, spasm is related to the catheter tip and is promptly relieved following removal of the catheter and administration of sublingual nitroglycerin. We report here a case of catheter-induced spasm which persisted following removal of the catheter and administration of sublingual nitroglycerin, but was promptly relieved by intracoronary nitroglycerin.

CASE REPORT

A 39-year-old builder underwent coronary angiography with a Judkins catheter without premedication as investigation for chest pain. Left coronary angiography was normal and left ventriculogram showed mild global hypokinesis.

At angiography, the first injection into the right coronary artery was normal. A second injection was associated with marked pressure damping and severe focal proximal spasm was demonstrated. The catheter was removed from the coronary orifice.

A fresh tablet of sublingual nitroglycerin (0.6 mg) was administered, and after 4 minutes, the tablet had completely dissolved and the patient reported slight headache. A decrease in systolic blood pressure of 40 mm Hg was recorded. Right coronary artery injection was repeated and was once again associated with pressure damping and demonstrated unchanged catheter tip spasm (Fig 1). At this stage, the patient was asymptomatic and no ECG change was recorded. Right coronary artery injection (0.1 mg) was injected into the right coronary artery. Repeat injection 1 minute later demonstrated that the spasm had resolved.

DISCUSSION

To our knowledge, this is the first case report of catheter tip-induced coronary artery spasm which persisted following sublingual nitroglycerin in a dose which produced systemic vasodilatation, and where the spasm was relieved promptly by administration of intracoronary nitroglycerin. Recent reports in the literature have identified a number of patients who were able to reach the maximum level of the lower scale but are unable to move the indicator on the higher scale. This group of patients is in place. The second scale reads 260 to 520 liters per minute and is used when the end piece is in place. The second scale reads 260 to 520 liters per minute and is utilized with the end piece removed. In our clinical practice we have observed significant clinical difficulty in the use of the HealthScan peak flow meter distributed by Organon. This flow meter has a removable end piece together with two flow scales so that flows ranging from 80 to 520 liters per minute can be determined. One scale reads 80 to 260 liters per minute and is used when the end piece is in place. The second scale reads 260 to 520 liters per minute and is utilized with the end piece removed. In our clinical practice we have observed significant clinical difficulty in the use of the HealthScan flow meter. We have found the HealthScan flow meter unreliable in daily clinical monitoring of flow rates. We have been satisfied with the results when using either the Vitalograph pulmonary monitor or the mini-Wright peak flow meter with preference toward the former because of its lower cost to the patient.

In conclusion, we concede that the HealthScan peak flow meter may be highly accurate and reproducible in vitro, but it has a serious deficiency when used in the clinical setting.

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REFERENCES


Peak Flow Meters

To the Editor:

It was with great interest that we read the recent article by Eichenhorn et al (Chest 1982; 82:306-09), as we have been assessing the clinical utility of the three flow meters described in their article. We have observed significant clinical difficulty in the use of the HealthScan flow meter distributed by Organon. This flow meter has a removable end piece together with two flow scales so that flows ranging from 80 to 520 liters per minute can be determined. One scale reads 80 to 260 liters per minute and is used when the end piece is in place. The second scale reads 260 to 520 liters per minute and is utilized with the end piece removed. In our clinical practice we have observed significant clinical difficulty in the use of the HealthScan flow meter. We have found the HealthScan flow meter unreliable in daily clinical monitoring of flow rates. We have been satisfied with the results when using either the Vitalograph pulmonary monitor or the mini-Wright peak flow meter with preference toward the former because of its lower cost to the patient.

In conclusion, we concede that the HealthScan peak flow meter may be highly accurate and reproducible in vitro, but it has a serious deficiency when used in the clinical setting.

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To the Editor:

We simply have not had the experience which you described in your letter wherein individuals are able to reach the maximum level of the lower scale, but are unable to move the indicator on the higher scale. In most cases, spasm is related to the catheter tip and is promptly relieved following removal of the catheter and administration of sublingual nitroglycerin. We report here a case of catheter-induced spasm which persisted following removal of the catheter and administration of sublingual nitroglycerin, but was promptly relieved by intracoronary nitroglycerin.

Figures 1 and 2: Right coronary artery angiogram in the left anterior oblique projection following administration of sublingual nitroglycerin (0.6 mg). This demonstrates persistent severe proximal spasm.

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scale with the end piece removed. In our testing procedure, we purposely tested flows at 300 liters per minute because it would represent a low end-point on the higher scale provided by Health Scan, as well as because of its clinical applicability.

I find it difficult to understand how the instrument could be accurate at 300 liters per minute, and be relatively inaccurate at the range of 260-300 liters per minute, but have no data to support this contention. In any case, I doubt that the number of individuals falling in this category would be very large.

Based on my own studies, I have difficulty accepting the use of either the Vitalograph pulmonary monitor or the mini-Wright peak flow meter for the reasons stated in our article.

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Knotting of a Swan-Ganz Catheter in the Pulmonary Artery

To the Editor:

Complications resulting from the use of flow directed, balloon-tipped pulmonary artery catheters have been well described. Among these, intracardiac knotting of the catheter and techniques for knot removal have also been reviewed. All of the previously described cases of catheter knotting have involved looping of the catheter in either the right atrium or right ventricle.

We recently had a case in which a pulmonary artery catheter was inserted and an adequate pulmonary artery wedge pressure obtained. Central venous, right ventricular and pulmonary artery tracings while introducing the catheter had also been unremarkable. Initial cardiac outputs, however, varied markedly, and were difficult to reproduce. The chest x-ray film revealed the problem (Fig 1). Obvious looping of the catheter in the left pulmonary artery outflow tract had occurred, with knot formation. Despite this, the catheter tip was in adequate position, and wedged easily. The catheter was subsequently removed without complications.

Cardiac outputs obtained via thermodilution techniques were inconsistent probably secondary to the artificially reduced distance from the injection port to the thermistor. This was caused by the loop in the catheter, and most likely resulted in inadequate venous mixing of the injectate.

To our knowledge, this case represents the first documented report of knotting of a balloon-tipped pulmonary artery catheter in the pulmonary artery. Further, it gives another possible etiology for erroneous cardiac output measurements.

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REFERENCES
2 Pace NL. A critique of flow-directed pulmonary arterial catheterization. Anesthesiology 1977; 47:453-65

Cardiac Involvement in Mixed Connective Tissue Disease

Pathomorphologic Changes

To the Editor:

We described a patient with mixed connective tissue disease and cardiac conduction disturbances, (Chest 1982;81:257-59), who has since died. The ultimate cause of death was pulmonary thromboembolism.

This communication serves two aims: it provides evidence that histopathologic changes in this patient were consistent with the diagnosis of mixed connective tissue disease and it reports on pathomorphologic changes of the heart with a special emphasis on the sinus node.

The diagnosis of the underlying collagenosis was corroborated by the following findings: benign lymphoepithelial lesion of the salivary glands, fibroplastic pleuropneumonitis, diffuse interstitial pulmonary fibrosis, chronic interstitial focal myositis, chronic lymphocytic adrenalitis, and submucous fibrosis with mononuclear cells of the small intestine. The heart was enlarged, the cavities hypertrophied and dilated. Microscopic examination revealed small foci of chronic lymphoplasmocytic infiltration and interstitial fibrosis.

In the sinus node, there was a diffuse, moderate fibroelastosis, more pronounced peripherally, and affecting more severely the

Figure 1. Intracardiac knotting of a Swan-Ganz catheter in left pulmonary artery.