

Inadvertant Coronary Artery Air Embolism Complicating Selective Coronary Angiography

By

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Introduction

The use of coronary angiography in the evaluation of patients with chest pain syndromes and coronary artery disease has increased by geometric proportion during the last decade. In addition to firmly establishing the low incidence of morbidity and mortality, there have been numerous published reports which have discussed the complications associated with this procedure¹⁻²⁻³. In reviewing the literature, however, we were unable to find more than a casual mention of coronary artery air embolism as a complication of selective coronary angiography⁴. As a result, this study was undertaken to elucidate the clinical features, hemodynamic, and electrocardiographic consequences of inadvertant coronary artery air embolism. In addition, the etiology and various methods of prevention are commented upon.

Materials and Methods

The last 1000 cardiac catheterizations at Grady Memorial Hospital were examined

retrospectively. Among this group, there were 750 patients in whom selective coronary angiograms were performed (approximately 5250 selective injections). Of this number, there were six instances in which air was inadvertently injected directly into a coronary artery. Criteria for selection included independent verification of an air bubble(s) outlined by contrast material within the lumen of the coronary artery by each of the authors. Air emboli travelling through a coronary artery filled with contrast material are radiographically very distinctive, and rarely escape the attention of an experienced angiographer. In the larger proximal segments of the vessel the small radiolucent air bubbles form nearly a perfect sphere. The lucent body can be seen to break up into smaller spheroids, or take on an oblong configuration to conform to the dimensions of the smaller distal vessels. (see Figure 1). Finally, they are seen to disappear into vessels so small as to be beyond the level of resolution. In addition, in every instance the written record was supplemented by verbal information obtained from at least one of the

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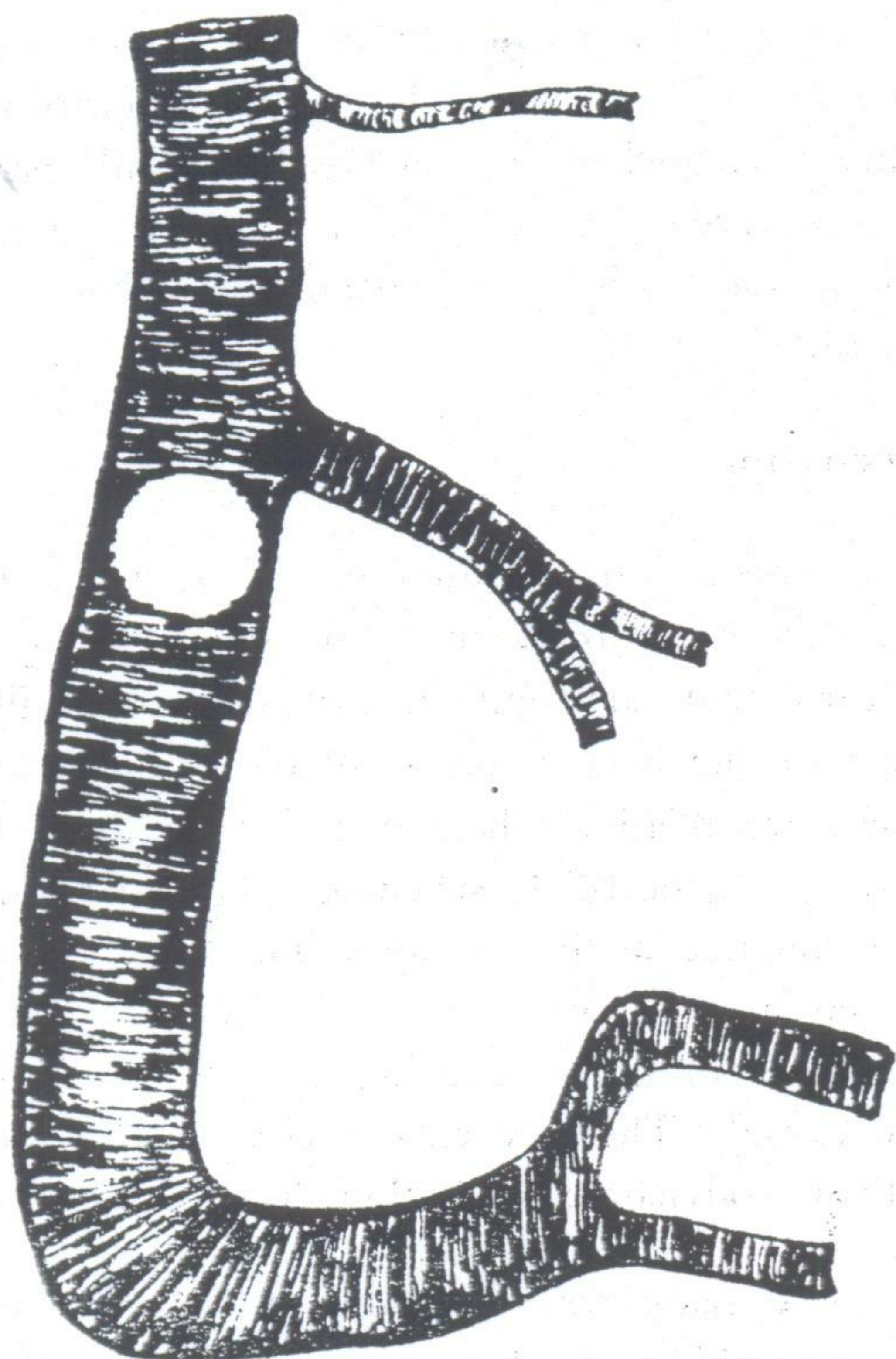


Fig. 1:-Typical appearance of an air bubble as seen in this proximal right coronary artery diagram.

physicians who performed the catheterization. The amount of air injected was estimated by tracing the outline of the "bubble", which closely approximated a circle in the proximal segment of the coronary artery, measuring the diameter, correcting for magnification error, and employing the standard formula for volume of a sphere $\frac{4}{3} \pi R^3$. Data from in vitro and in vivo animal experimental studies plus additional case reports were collected from the literature.

Results

All six cases were studied for diagnostic evaluation of a chest pain syndrome. One patient,

age thirty five, had a previously documented, inferior, transmural myocardial infarction. Five of the six cases had no angiographic evidence of obstructive coronary artery disease, even though, the patient mentioned above had an inferior akinetic area on ventriculogram. The sixth patient had significant disease in both the left and right coronary systems. Among the group of 750 patients who had selective coronary angiograms for chest pain syndromes, approximately 37% had normal coronary angiograms. Therefore, the fact that five of the six patients with coronary air emboli had normal coronary angiograms was indeed fortuitous.

The overall occurrence rate for coronary air emboli was 0.8% per patient or 0.1% per injection. The age range was thirty five to fifty five. Five of the cases were males.

Of the three patients with emboli to the right coronary artery, patients number one and two had a single bubble embolize to a posterior left ventricular branch. Patient number three had two bubbles go down the posterior descending branch. The remaining three patients had left coronary artery emboli: in patient number four a single bubble and in patient number five, two bubbles went down an obtuse marginal branch of the circumflex artery. In the sixth patient the left anterior descending branch as well as a diagonal branch with an obstructing lesion causing 75% diameter reduction were embolized.

In each case, subsequent injections of contrast material into the same coronary artery failed to demonstrate visible coronary artery obstruction or spasm.

The estimated total volume of air embolized ranged from 0.02 to 0.05 ml. Five of six cases

had chest pain compatible with myocardial ischemia, beginning between 15 and 60 seconds after injection of the air embolus and lasting one to five minutes. In three instances, nitroglycerin was given with no difference in course from the three instances in which it was not given. The clinical manifestations of the air emboli in the sixth patient with obstructive coronary disease did not differ from those of the patients with normal coronary arteries.

Electrocardiographic changes included sinus tachycardia in two and ST and T wave changes, in four. In three of the four cases where ST and T wave changes were present, there was ST depression and T wave inversion on a lead II monitor. In the fourth case involving embolism to the right coronary artery, there was transient ST elevation in lead II. There were no other arrhythmias or conduction disturbances. There was no relation between the coronary artery embolized and the type of ECG changes observed (see Table 1).

There were no long term adverse effects in any of the six cases. Follow-up electrocardiograms were unchanged from baseline in all cases, and myocardial enzymes drawn post catheterization in one patient demonstrated no abnormal elevation.

Discussion

Although the limitation of such small series is recognized, in these six patients it appears that air emboli, which occasionally occur during hand injection of the coronary arteries, are relatively benign and result in only transient myocardial ischemia. The manifestations include angina pectoris immediately preceded by ST and T wave changes which are indistinguishable to those resulting from ischemia of any cause⁵. These events were unassociated with hemodynamic or rhythm disturbances.

The major reason for the absence of significant morbidity is the extremely small volume of the air emboli when compared to that employed

TABLE I: Summary Patient Data

Patient and age	Coronary Artery Embolized	Volume of Air (ml)	Chest Pain	Acute ECG Changes	
				ST-T	Sinus Tachycardia
#1 - 55 y.o.	RCA	0.03	+	+*	+
#2 - 35 y.o.	RCA	0.02	-	+	-
#3 - 35 y.o.	RCA	0.03	+	+	-
#4 - 52 y.o.	LCA	0.04	+	-	-
#5 - 35 y.o.	LCA	0.05	+	+	+
#6 - 51 y.o.	LCA†	0.03	+	-	-

* ST elevation, transient; all others ST depression and T wave inversion.

† 75% discrete diameter reduction of the first diagonal branch of the LAD and 75% lesion in the right coronary artery.

in the majority of animal experiments, and to pathologically documented human case reports⁶⁻⁸. In his classic paper, Rukstinat in 1931⁶, using the canine model, found that when 15-20 ml of air was injected directly into the left coronary artery, acute cardiac dilatation followed by ventricular fibrillation uniformly occurred. On the other hand, when 2.5-5 ml was injected, the only significant finding was a transient increase in heart rate. The speed of injection also appeared to play a role. When 15-20 ml of air was delivered over 40 seconds the animals demonstrated no untoward effects.

Also operative in our series is the fact that one and not both coronary arteries was embolized. Kent and Blades⁷, again using the canine model, embolized both coronary arteries by introducing 1 ml of air into a pulmonary vein. In each case, ventricular fibrillation and death occurred. 0.25 ml injected in the same manner was always well tolerated.

A complete discussion of the variety of clinical situations (such as spontaneous or iatrogenic pneumothorax, trauma, cardiac surgery, etc.) in which fatal coronary artery air emboli have been pathologically documented is beyond the scope of this paper. However, in these situations, death appeared to result from profound myocardial ischemia with resultant left ventricular dysfunction and fibrillation. Again, the volume of air that gained access to one or both coronary arteries in the above noted situations was much greater than that which occurred in our series, or which could conceivably occur in the hands of an experienced angiographer.

The ischemic manifestations of air emboli are for the most part secondary to mechanical

obstruction of small vessels. There is some data, however, to suggest that a local reactive vasoconstriction occurs⁸⁻⁹. This would help to explain why the ischemic effects are sometimes greater than would be expected on the basis of the size of the embolus alone. As pointed out by Eiseman¹⁰, gas bubbles that are of a size equal to or greater than that of the blood vessel result in columns of blood interspersed with "slugs" of air. In this "slug flow system" as long as perfusion pressure and constant flow was maintained there was no increase in resistance to flow. However, air slugs which became wedged in small vessels or at points of constriction created a stationary system wherein resistance was increased and flow was significantly impeded.

In the current series the air "slugs" were seen to move freely through the coronary tree and disappear into vessels beyond the resolution of our angiographic equipment, i.e., $<100 \mu$. Therefore, it appears that air emboli of a size that occur during selective coronary angiography could, if at all, result in a static "slug flow system" only in vessels beyond the level of resolution of the current cineangiographic equipment. Even if local vasoconstriction occurs, the resultant area of ischemia would be quite small, and as noted, likely to result in no significant hemodynamic or electrical instability.

In five of the six cases in this retrospective series the air emboli fortuitously occurred in patients with normal coronary arteries. Although there was no significant differences in the clinical manifestations of patient number six (air emboli to a critically stenosed, large, diagonal branch of the left anterior descending), it cannot be stated that an air embolus as small as 0.03 mls (average) is necessarily benign in all patients with

significant coronary artery disease. It is plausible that a large caliber vessel with a critical stenosis could have pressure and flow characteristics such that an air bubble could become wedged and create a total obstruction. The hemodynamic effects would then become dependent upon a number of factors including the adequacy of collateral flow and the state of the myocardium distal to the obstruction.

In each of the six cases, the cause of the air embolus could be traced to a technical error by the operator. There was no evidence that cavitation at the catheter tip occurred¹¹. The common denominator was a failure to aspirate the contents of the manifold system and the catheter back into the syringe (followed by re-filling of the system with contrast material) between each injection of a coronary artery. By performing this basic maneuver, all air which may have gained access to the manifold or catheter would be eliminated. Further, each time the coronary syringe is refilled with contrast, the manifold should be "cleared" in the same manner.

The primary source by which air entered the system was the rotating swivel attachment located at the distal end of the manifold to which the catheter is secured. The nondisposable manifolds can, if they become worn, allow air to enter the system if any torque is applied to the swivel during manipulation of the catheter or syringe. A second important source is the air which almost invariably becomes trapped in the syringe during the procedure due to leakage around the plunger and cavitation phenomena. If the syringe is not held in a semivertical position or is completely emptied during an injection, the air will either embolize or become trapped

within the catheter. Other sources by which air may gain access to the system are at the points of attachment of the catheter, flush solution line, contrast material line, pressure line, and the syringe to the manifold. Failure to secure these attachments may result in air embolism if the catheter and manifold are not "cleared" before each injection.

It is uncertain what procedure should be followed if an air bubble becomes wedged in a proximal vessel. Maneuvers to increase intracoronary pressure and flow such as having the patient cough or, as suggested by one angiographer, to vigorously inject the coronary artery with dye; may help to dislodge the bubble or break it up. The role of these maneuvers or the use of nitroglycerin, however, has not been established. In cases such as ours where the embolus is seen to go down a vessel unobstructed to a branch beyond angiographic resolution, observation and withholding further manipulations until ECG changes and chest pain resolve appears to be all that seems needed.

Summary

Six cases of inadvertant coronary artery air embolism complicating selective coronary angiography are reported. The incidence of this complication was 0.8% (6/750). In three cases, the right coronary artery was embolized and in three, the left. All six instances proved to be relatively benign. The major result was transient myocardial ischemia manifest by angina pectoris and ST-T changes which lasted one to five minutes. There were no rhythm or hemodynamic disturbances except for sinus tachycardia, nor were there any long term sequelae. The major reason for the absence of significant

morbidity was the extremely small volume of the air emboli (avg. 0.03 mls). The source of the air emboli as well as techniques to avoid this complication are discussed.

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