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Central Venous Catheters in the Management of Air Embolism: Whether As Well As Where

IN THIS ISSUE of ANESTHESIOLOGY, Bunegin *et al.*[1] present evidence obtained from an *in vitro* model that precise anatomical placement of a central venous catheter is a critical determinant of one's ability to efficiently recover air which has entered the venous circulation. These workers begin with the assumption that a central venous catheter is indicated whenever there exists a high risk for the occurrence of venous air embolism (most commonly a neurosurgical patient undergoing surgery in the sitting position) and therefore concern themselves with "where" not "whether" a catheter should be placed. The latter issue of "whether" is not a dead one and deserves consideration before examining their conclusions concerning "where."

As so often occurs, the initial recognition in 1965 that a central venous catheter may be useful in the management of air embolism was serendipitous. For reasons now forgotten, central venous pressure was being measured in a series of neurosurgical patients at the Mayo Clinic. In the space of six months, two of these patients (both in the sitting position) developed ominous hemodynamic signs compatible with the diagnosis of air embolism. In each instance large volumes of air were withdrawn from the catheter and both patients recovered without incident. (Interestingly, postoperative chest x-rays showed the catheter to be in the superior vena cava on both occasions.) This stimulated a prospective study to evaluate the effectiveness of central venous catheters for the diagnosis and treatment of venous air embolism in patients undergoing surgery in the sitting position. The encouraging findings of that study resulted in the recommendation that central venous catheters should be inserted in all such patients when technically possible.[2] The decision to localize the catheter in the mid right atrium was without a scientific basis. Rather it was an intuitive choice, and possibly an erroneous one.

Since that time the routine use of right atrial catheters in patients at risk has achieved an almost sacrosanct stature. Enthusiasts have been heard from the lectern to label the failure to use catheters as malpractice. Individual practitioners commonly cancel surgery if a catheter cannot be placed in the preoperative period. Such positions represent the extreme and fail to recognize the considerable progress that has been made in the diagnosis of air embolism and the prevention of clinically significant episodes. The introduction of the Doppler converted our perception of air embolism from that of being an unusual but clinically significant complication to one that is common but rarely significant.[3] Thus, where once the catheter was a route whereby large volumes of air were often recovered, today it is unusual to recover more than a few bubbles. This simply reflects the extreme sensitivity of the Doppler and the readily effective means for preventing further entry of air once the complication is recognized. The catheter thus has become useful primarily as a confirmatory diagnostic tool which aids considerably in learning those Doppler sounds that are characteristic of air. Once learned, the catheter is not needed for diagnosis.

Why then continue to use catheters at all? For therapeutic reasons the catheter offers assistance in perhaps two circumstances. In the event of the abrupt aspiration of large volumes of air, such as can occur with inadvertent opening of a major dural sinus, the catheter may be lifesaving. Those who deny the potential occurrence of such a complication are in error; that they have not experienced such a complication simply reflects its infrequent occurrence. Secondly, there is the nagging concern that some of the embolized air may cross to the systemic cir-

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culation with possibly disastrous cardiac or cerebral consequences.[4] Since this must occur both as the result of an available route (*e.g.*, a patent foramen ovale) and as a direct function of the volume of embolized air *not* recovered, the potential to recover embolized air via a catheter should not be abandoned.

Admittedly, the risk of either of these occurrences is low, but to the degree that the risk of inserting a catheter is low, a reasonable effort should be considered and is still generally recommended. What constitutes a "reasonable effort" is a function of the skill and experience of the operator. For the author this means that if a central venous catheter cannot be passed via one of the arm veins no further effort is made. Experience indicates that this should be successful 90 per cent of the time and should require no more than 10–15 min.[5] In the event of failure the case is rarely cancelled, rather the surgeon is informed and, with a perhaps somewhat higher level of anxiety, the operation proceeds. A decision to utilize an internal jugular or subclavian vein may not be unreasonable but does introduce added risk which must be weighed against the risk of proceeding without a catheter. Failure to establish a central venous catheter should not be labeled malpractice nor should such failure categorically be considered grounds for cancellation of the surgical procedure.

The opposite extreme is also represented; namely that a catheter is not necessary and routine insertion of one represents a waste of time and effort. Thus, Jackson reported 461 successive sitting cases without central venous catheters (or Doppler) and without complications related to air.[6] In three of these cases clinical signs of air embolism were recognized but were corrected without difficulty. Although these are impressive results, those of us who have witnessed the devastating complications that can result from air embolism cannot help but view Jackson's recommendation as somewhat cavalier.

If there is some difference of opinion regarding "whether," the question of "where" is even more unsettled. Thus, the work by Bunegin *et al.*[1] is important since it represents the first effort to provide a rational basis for the optimal placement of a catheter. In addition, they provide evidence that a multiorificed catheter will, in general, perform better than a single-orificed one. Demonstration in their model that the optimal location for the proximal port of the multiorificed catheter (or the tip of the single-orificed catheter) is just above the right atrium is convincing as are the explanations they offer for this observation. Still lacking, however, are the data in humans to confirm their findings. This will require a large series of patients to compare the volume of air recovered with either mid-atrial or supra-atrial placement of the catheter, as well as to compare the multiorificed and single-orificed catheters. Unfortunately, *in vivo* evidence in experimental animals cannot provide the answer because of the many critical anatomical differences.

It should also be recognized that their results suggest the need for quite precise localization of the catheter tip in relation to the SVC-atrial junction. This cannot be done with pressure monitoring nor is such precision possible using routine chest x-rays. However, reasonable precision is possible using the single-orificed catheter as an exploring ECG electrode and identifying the location of the sinoatrial node (by the characteristic P wave changes[7]). If, however, one uses a multiorificed catheter, this precision may be lost or at least blunted since the exploring "tip" now includes several orifices extending over the distance of several centimeters. Thus, the active electrode is no longer a discrete point but rather a diffuse one. Experience with the multiorificed catheter will be required before firm recommendations can be made as to how best to localize the proximal orifice.

Finally, it should be noted that the debate over "where" extends beyond the right atrium to include the pulmonary artery. There is evidence that a pulmonary artery catheter is useful as an indicator of the volume of air embolized as reflected by the magnitude of increase in the pulmonary artery pressure.[8] Whether this is a more quantitative measure than that provided by monitoring expired CO_2 has not been established. Most proponents of the pulmonary artery catheter recommend that a double-lumen catheter be used with the second orifice located at the level of the right atrium. This would permit aspiration from both the right atrium and the pulmonary artery while at the same time providing semiquantitative information regarding the magnitude of the problem. Once again the risk as well as the cost of routine pulmonary artery catheterization must be weighed against the stated benefits.

In summary, it seems premature to judge the use of central venous catheters for the management of air embolism as obsolete, as does it seem extreme to condemn those who view them as something less than essential. The catheter is no longer a primary diagnostic tool and now rarely plays an important role in therapy. But on those few occasions that it does become important the "bother" of a routine reasonable effort to place the catheter seems well worth it. To this end any effort to identify the optimal location for such a catheter is welcomed.

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Anesthesiology
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Is Anesthesia Beneficial for the Ischemic Heart? II

IN A STUDY by Bland and Lowenstein[1] we were told, "Do let the blood pressure drop and do use myocardial depressants."[2] In defense of this thesis, Dr. Hamilton and co-workers have now demonstrated that this approach is indeed feasible.[3] They have treated 12 patients who responded to surgical stress with increased systemic, pulmonary arterial, and left ventricular filling pressures with the myocardial depressant anesthetics halothane and enflurane. Six of their patients also showed evidence of myocardial ischemia during the stress by ST-segment analysis which reverted to normal with the anesthetic treatment. Thus, anesthesia, specifically halothane and enflurane, clearly was beneficial for those ischemic hearts.

However, the same Dr. Lowenstein, with different coworkers, now suggests that the decrease in blood pressure and global ventricular function seen with increasing concentrations of halothane, although clearly not detrimental to the normal heart, may be associated with regional myocardial ischemia in a heart with coronary stenosis and "may be an important mechanism for perioperative infarction."[4] Clearly such an effect is not beneficial for the ischemic heart!

Can we resolve this quandary? In fact, I do not believe that there is any contradiction between the two studies. As noted previously, "tachycardia, sympathetic hyperactivity and perhaps even increased output of hormones such as angiotensin and vasopressin are deleterious to the ischemic heart."[5] Although tachycardia was not a significant part of the syndrome treated by Roizen *et al.*,[3] they documented sympathetic hyperactivity with increased plasma norepinephrine concentrations. Thus, they were restoring the myocardial oxygen supply-demand balance by decreasing demand with the depressant anesthetics. However, as Hamilton has suggested, other pharmocologic regimens in other people's hands may be just as beneficial.[2]

On the other side of the coin, "if myocardial perfusion and oxygenation are adequate without anesthesia, then the production of decreased coronary perfusion pressure by anesthesia in a heart where the flow may be highly pressure dependent is unlikely to benefit that heart."[5] Lowenstein *et al.* intentionally prepared an animal with a "critical stenosis" (so that no ischemia was evident at low anesthetic concentrations and high perfusion pressures) in the left anterior descending coronary artery leaving the circumflex coronary artery normal. As anesthetic concentration was increased and diastolic arterial pressure decreased, progressive dysfunction appeared in the region supplied by the stenosed coronary artery signifying myocardial ischemia. On the other hand, there was no such dysfunction in the segment of myocardium supplied by the normal circumflex artery. Thus, Lowenstein and co-workers have demonstrated for the first time that the oxygen supply-demand balance of myocardium supplied by a stenosed artery is not well-preserved during increasing concentrations of halothane. On the basis of the present work, it seems likely that the decreased perfusion pressure was responsible for this imbalance. This hypothesis is supported by the observations of Behrenbeck *et al.* who observed no difference in regional myocardial function between ischemic and non-ischemic areas as halothane concentration was increased when perfusion was controlled at the different anesthetic concentrations by an extra corporeal pump.[6]

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