# Comparing percutaneous tracheostomy with open surgical tracheostomy

*Both will coexist until robust evidence becomes available*

Tracheostomy is one of the most frequent surgical procedures carried out in critically ill patients.[1] Traditionally, open surgical tracheostomy has been done by surgeons in the operating room, and in many institutions it remains that way. In the past 50 years, however, several methods of doing percutaneous tracheostomy at the bedside have been introduced. Some of these methods did not get far because of high complication rates. The most popular technique today is the percutaneous dilatational tracheostomy described by Ciaglia in 1985.[2] This technique uses serial dilators over a guide wire and is usually done at the bedside in the intensive care unit under bronchoscopic guidance. Ciaglia later introduced a single tapered dilator to replace the serial dilators, further simplifying the technique. In experienced hands, percutaneous tracheostomy can be done in five to 10 minutes and will rarely require more than 15 minutes. The low cost of percutaneous tracheostomy initially was an important reason that led to its popularity in the United States and elsewhere. It is likely to thrive, unless well designed prospective studies show that open surgical tracheostomy is clearly superior. Moreover, both open surgical tracheostomy and percutaneous tracheostomy will coexist, as long as non-surgeons continue to do tracheostomies.

The trend towards minimally invasive surgery and the development of interventional services in non-surgical specialties spurred considerable interest in bedside percutaneous tracheostomy. When it was first introduced its exponents pointed to its ease of performance, a safety profile comparable to open surgical tracheostomy, significantly lowered hospital charges, and more efficient use of intensive care unit resources. The cost was low because there were no operating room charges or anaesthetists fees.

Percutaneous tracheostomy as a bedside procedure in critically ill patients opened the door for open surgical tracheostomies at the bedside. These have developed in the past decade, with reports of comparable safety.[3–5] The surgeon's fee for tracheostomy is the same, regardless of where or how it is done. The shorter operating time needed for the percutaneous method is not a cost advantage when done at the bedside. Most percutaneous tracheostomies are now done using disposable kits under bronchoscopic guidance. These increase the cost, rendering percutaneous tracheostomy more expensive than open surgical tracheostomy when both are done at the bedside.[3]

Many studies comparing the safety and outcome of percutaneous dilatational tracheostomy with standard open surgical tracheostomy lack rigorous design, making useful comparisons impossible. Two recent meta-analyses have compared percutaneous tracheostomy and open surgical tracheostomy.[6 7] Dulguerov et al did a meta-analysis that included observational as well as prospective studies and studies which used different percutaneous tracheostomy techniques.[6] They found that percutaneous tracheostomy had more perioperative complications, in particular perioperative death and cardiorespiratory arrest. Freeman et al included only prospective studies comparing percutaneous tracheostomy done by Ciaglia's technique with open surgical tracheostomy.[7] They found potential advantages for percutaneous tracheostomy in ease of performance and a lower incidence of peristomal bleeding and postoperative infection. Both meta-analyses are limited by the heterogeneity of the studies they cite.

The status of percutaneous tracheostomy has undergone several ups and downs in many institutions. It is common to see initial zest as percutaneous tracheostomy is introduced followed by dismay at unacceptably high complication rates; then it is replaced by open surgical tracheostomy. Often the high complication rates reflect inadequate training and lack of familiarity with the technique, especially during the learning curve. Expertise in open surgical tracheostomy does not necessarily confer safety and expertise in percutaneous tracheostomy. Therefore training is essential even for experienced surgeons. Ideally today's surgical trainees need training in both open surgical and percutaneous tracheostomy. The ability to convert a percutaneous method to an open surgical procedure if needed has always been an advantage that surgeons have over non-surgeons.

Almost every case scenario that was previously reserved for open surgical tracheostomy has been successfully managed with percutaneous tracheostomy, including emergency tracheostomy, a history of prior tracheostomy, obesity, short neck, coagulopathy, and bleeding diathesis.

Tracheostomy is done mostly in critically ill patients, many of whom do not survive. This makes it difficult to study its long term complications. We still do not know the long term complication rates of tracheostomy itself—notably tracheal and subglottic stenosis, and tracheomalacia. A confounding factor in assessing these complications is the possible airway injury caused by translaryngeal intubation usually done before the tracheostomy. No study has attempted to define these complications and prospectively study long term survivors after tracheostomy. Using bronchoscopy to guide percutaneous tracheostomy provides the advantage of visualising and recording tracheal mucosal injury, tracheal wall abnormalities, and vocal cord and subglottic injury present prior to tracheostomy. Documenting these may be useful in the prospective evaluation of long term complications.

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# Screening for inherited metabolic disease in newborn infants using tandem mass spectrometry

*Further assessment of performance and outcome is needed*

Although individually rare, inborn errors of metabolism represent a potentially preventable cause of death and disability. Screening for phenylketonuria (birth prevalence 10 per 100 000) was introduced in the United Kingdom over 30 years ago. It has proved successful in preventing severe mental retardation. The development of tandem mass spectrometry enables a wide variety of additional compounds to be assayed on the dried blood spots routinely collected from newborn infants.[1] The combined birth prevalence of disorders, excluding phenylketonuria, which could be detected by screening is about 20 per 100 000. Of these, medium chain acyl CoA dehydrogenase deficiency is one of the most important. However, despite experience of screening over a million infants, many questions about screening for this disorder remain unanswered.

In the United Kingdom between 5 and 11 per 100 000 live born infants have medium chain acyl CoA dehydrogenase deficiency, which is about 35 to 70 children each year.[2] This recessively inherited disorder classically presents during infancy and early childhood with a severe illness characterised by encephalopathy and hypoglycaemia. This is usually precipitated by a minor febrile illness, particularly gastroenteritis, and fasting. Of those presenting clinically, up to a quarter will die and about a third of survivors will have irreversible neurological damage.[3 4] In a significant proportion there is a history of previous sudden unexplained death or encephalopathy in a sibling.[4] However, the presentation varies widely, with some individuals not presenting until they are adults and an unknown number remaining undiagnosed or asymptomatic. In people of northern European descent, over 80% of clinically diagnosed patients are homozygous for one mutation—G985A. Simple heterozygotes have no symptoms. The mainstay of treatment is a high carbohydrate diet, orally or intravenously during fasting or intercurrent infection.[5] This seems to be effective, with few of the 162 children reported in the two largest series having further episodes of encephalopathy.[3 6]

The outcome for siblings diagnosed prospectively also seems good.[6] Given this clinical course and response to treatment, medium chain acyl CoA dehydrogenase deficiency has been identified as a potential candidate for early detection through newborn screening.[7 8]

Several centres outside the United Kingdom have introduced newborn screening for medium chain acyl CoA dehydrogenase deficiency by using tandem mass spectrometry to measure acyl carnitines.[9] Carpenter et al have recently reported identifying 11 babies with definite medium chain acyl CoA dehydrogenase deficiency among 275 000 screened, a birth prevalence of 4 per 100 000[10]—which was lower than expected. Their publication highlights many of the questions and uncertainties that remain about performance and outcome.

Differences in the choice of metabolite as well as in thresholds used to define a positive result limit direct comparison of test performance between centres. A further issue is the criteria used to confirm a diagnosis of medium chain acyl CoA dehydrogenase deficiency. In one study from the United States 62 infants were considered to have medium chain acyl CoA dehydrogenase deficiency solely on the basis of "pathological acyl carnitine profiles."[9] By contrast, Carpenter et al applied explicit independent diagnostic criteria to 23 infants with positive screening results and diagnosed definite medium chain acyl CoA dehydrogenase deficiency in 11, with one further probable mild case.[10] A striking finding in this report is that of the remaining 11 babies who screened positive but did not meet the diagnostic criteria for medium chain acyl CoA dehydrogenase deficiency (false positives), four died in the neonatal period. This is consistent with observations that infants and young children who are ill for any reason may have abnormal patterns of acyl carnitines.[11] In a retrospective study based on 100 600 dried blood spots, all but one of those with false positive results were preterm babies.[12]

The false negative rate is difficult to determine, as none of the prospective studies included a rigorous scheme to identify those who might have escaped detection. Babies who have rapidly become carnitine depleted may be missed. It is already clear that newborn screening identifies some individuals whose history is not known and who may be treated unnecessarily. In both the Australian and the US study the frequency of the common mutation was lower than expected, and the proportion of A985G heterozygotes higher.[9 10]

To be maximally effective screening needs to be done and acted on very soon after birth. Up to one third of those with medium chain acyl CoA dehydrogenase deficiency have been reported to

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