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A risk stratification tool for exacerbations of COPD: time to switch to DECAF

Richard W Costello, Breda Cushen

To paraphrase Henry Kissinger's infamous quote ascribing the vicious nature of academic politics to their lack of importance, it seems that precisely because exacerbations of COPD are so important, they are so infrequently studied. COPD is one of the most common reasons that people are admitted acutely to hospital. An OECD (Organisation for Economic Co-operation and Development) report indicates that the rate of admissions is 164 for women and 251 for men per 100 000 population.[1] The median length of stay is 5 days and 5%–7% of people die during the course of the admission. While many recover to be discharged, 30% are readmitted within the subsequent 90 days.[2] Clinically, compared with all patients with COPD, those who experience exacerbations suffer increased morbidity, reduced quality of life and have shortened life expectancy. One important feature of COPD exacerbations about which we have relatively little information is which clinical features, on presentation, indicate that an individual person is at risk of a poor outcome. In this month's *Thorax*, work from Dr Bourke's group have addressed this important question and report a validation study of a previously described risk assessment score, termed DECAF.[3]

To put this study into context, it is worthwhile reviewing the nature of an exacerbation of COPD. Most exacerbations are precipitated by infections, both viral and bacterial. As there is a seasonal pattern to these exacerbations, temperature change, variations in environmental pollution levels and poor adherence to preventer inhalers are additional causative factors.[4] Locally, the airway responds to these insults with increased production of protective mucus and a resulting increased cough frequency. Infection increases the individual's metabolic demand and the resulting increase in VO_2, leads to an increase in respiratory rate. In obstructed airways, the increased rate of ventilation and the impairment of airflow, from both mucus impaction and airway inflammation, lead to a state of dynamic hyperinflation with reduced respirable volumes and a sensation of progressive dyspnoea.[5] Dynamic hyperinflation has a number of direct consequences on the cardiac and respiratory systems. For example, for a cough to be sufficient to clear mucus, a flow of >300 L/s must be generated. Hyperinflation reduces cough efficiency, which compounds the deteriorating situation as mucus is retained.[6] Increased intrathoracic pressure, from dynamic hyperinflation, impairs venous return and increases pulmonary vascular resistance which may in turn cause cardiac dysrhythmia, in particular atrial fibrillation.[7] Hyperinflation also reduces the peak inspiratory flow that an individual can generate when using an inhaler, thereby reducing the effectiveness of rescue medications.[8 9] In short, relatively small changes in metabolic demand from sepsis along with the development of dynamic hyperinflation may have important direct clinical effects that cause the patient to seek emergency care.

Once the patient seeks help, the clinical team needs to provide care in a setting that is appropriate to the patient's needs. Over time, pathways have been established to provide care for particular clinical circumstances. For example, less severe exacerbations may be treated by home from hospital care programmes, while more severe cases, in which increased dead space ventilation results in acidotic hypercapnic respiratory failure, require ventilatory support in a monitored area.[10 11] However, there is no validated prognostic tool that can be reliably used to direct where care for patients with an exacerbation may best be provided. Further, there is no valid reliable clinical tool that could be used to inform patients and their families of the risk of death during the exacerbation.

The current paper is the third of a 'trilogy' of studies that the authors have performed to address this question. The basis of their work was a large prospective observational study of 920 consecutive patients with a primary diagnosis of an exacerbation of COPD who had presented to two general hospitals. A particular strength of the study was the large sample size, the sensible liberal inclusion criteria that included people with exacerbations of COPD and coexisting pneumonia and the comprehensive ascertainment of patients, even those with a short hospital stay.[12] The in-hospital mortality rate was 5.8% in those without evidence of pneumonia but 20% in those with both an exacerbation of COPD and coexisting pneumonia. This paper also described the particular value of an extended Medical Research Council (MRC) dyspnoea score, termed eMRCD. An important difference between the eMRCD and the traditional MRC score is that an eMRCD score of 5 identifies only those patients who, in a stable state, are unable to leave their home unassisted. Patients at this level are then further divided into those who can carry out self-care activities, such as washing and dressing, independently (eMRCD 5a) or require help (eMRCD 5b). Stratification of patients using the eMRCD, was a better discriminator of mortality than CURB-65[13] or the traditional MRC dyspnoea score. This initial report highlighted two important clinical features of an exacerbation of COPD, namely that coexisting consolidation and a lower baseline capacity markedly impact the outcome.

In a follow-up paper, using data from this same cohort, statistical modelling of all admission variables was performed to devise a composite risk score.[14] The variables studied were comprehensive and included socio-demographic characteristics of the patients, the presence of comorbid conditions, markers of COPD severity and laboratory data available on admission including arterial blood gas values. From the dataset, they identified five categorical values that performed strongly in predicting in-hospital mortality. These features were the stable-state level of dyspnoea, the presence of eosinopenia, consolidation on chest X-ray, respiratory acidosis and atrial fibrillation, collectively this is easily remembered as the DECAF score. It is interesting to note that these parameters reflect features that could be attribute to the effect of either sepsis or hyperinflation. The receiver operator characteristic (ROC) for this score in predicting in-hospital mortality was 0.86, above 0.8 is considered to be a very reliable test. Further, compared with other instruments, such as APACHE II,[15] CURB-65 and others, DECAF performed significantly better in predicting in-hospital mortality.

Department of Respiratory Medicine, Royal College of Surgeons in Ireland (RCSI), Beaumont Hospital, Dublin, Ireland

Correspondence to Professor Richard W Costello, Department of Respiratory Medicine, RCSI Clinical Research Centre, Smurfit Building, Beaumont Hospital, Dublin 9, Ireland; rcostello@rcsi.ie

A clinical prognostic tool requires robust validation in an independent group of patients and in their current paper, Echevarria and colleagues have performed a validation of the DECAF score.[3] A new cohort of 880 patients from the original two hospitals and an independent cohort of 845 patients who were admitted with an exacerbation of COPD to four additional hospitals were included, giving a total cohort of 1725. In this study, the ROC for predicting in-hospital mortality was 0.82, similar to the original baseline study, an important confirmatory finding. A particular strength of this cohort is that it is representative of the national population with a wide geographical spread including urban and semirural populations. The overall mortality rate, 7.7%, was similar to that reported in the 2008 British Thoracic Society COPD audit[16] although slightly higher than that reported from the 2014 audit,[17] 4.3%, which the authors attribute to case ascertainment and a higher proportion of patients with pneumonic exacerbations and poor baseline status in the study. The other novel feature of this study is that the large dataset permitted the authors to derive a 0–6 point prognostic scale. Those with a score of 0–1 had low mortality, while a score of ≥3 was associated with a stepwise increase in in-hospital mortality. In those with a pneumonic exacerbation, DECAF again proved to be a stronger predictor of in-hospital and 30-day mortality compared with the commonly used CURB-65. This was especially true among those patients deemed low risk by each score. The study has obvious value for clinicians in practice as a guide to triage patient care, for example to early discharge services or to more specialist care beds. Of those with a DECAF score of 5–6, 40% died in hospital with median time to death of 2 days. The DECAF may therefore also help clinicians communicate risk to patients and their families and inform healthcare managers to allocate services appropriate to patient needs.

As is the case with decaffeinated coffee, it is important to know that some things are missing from the DECAF studies. Two areas that were not assessed in the derivation of the DECAF score are variations in the quality of care and their impact on clinical outcomes. For example, the length of time that the patient spent in an inappropriate care setting, such as in the emergency department, or the inappropriate use of high-flow supplemental oxygen, both of which impact outcomes. The other is to know why patients with low DECAF scores present with an exacerbation. This may be an important point because even if a patient has a low risk of dying, the frightening symptoms of an exacerbation of COPD may be reason enough to admit the patient to hospital, where palliation of symptoms may be required. On the positive side, besides being a catchy mnemonic, DECAF, is a robust and carefully conceived clinical tool that will help guide clinicians in the management of this common condition.

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