In My Own Time

Respiratory failure 30 years ago

E J M CAMPBELL

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Before 1950, respiratory failure as we know it today was scarcely recognised. Barcroft, Haldane, Henderson, and others had provided sound frameworks for understanding anoxia, respiratory acidosis, and the functional disorders which caused them. These topics were part of the undergraduate physiology curriculum, which in my case had extra emphasis during a BSc year. But none of this theoretical framework was put to use in the wards.

These reflections are particularly concerned with respiratory failure in chronic lung disease, and my memory of the story we learnt as students (helped by reference to a few popular medical texts of the time) is as follows. People who suffer repeated attacks of acute (infective) bronchitis develop persistently infected bronchi. This condition is called chronic bronchitis, and it leads on to destruction of the lungs, causing emphysema—which is manifested by breathlessness. Emphysema puts a strain on the right heart, which eventually fails and causes cor pulmonale with oedema. Throughout this natural history the patient is liable to attacks of secondary infection, during which he becomes anoxic. After the development of emphysema the prognosis is poor, and once cor pulmonale has developed it is grave.

Not much notice was taken of these patients in London teaching hospitals. Neither the junior nor the senior staff regarded them as "interesting," and their treatment was unrewarding. The chest physicians of the time were nearly all specialists in tuberculosis, which was naturally and correctly seen as a much more pressing problem. Blood-gas measurements were so impractical as to be non-existent, and oxygen therapy was clumsy and unpredictable. If a patient was put into an oxygen tent his distress might be lessened; he might become pink and peaceful, but quite frequently he entered a slumber which was irreversible.

Light through the fog

Then in the early 1950s the smogs brought the problem into the limelight. Barach, Donald, and others had given us hints, but, like many others, I was helped most by Keith Westlake and Tom Simpson, who had large numbers of patients with respiratory failure to deal with at Chase Farm Hospital in Enfield. For many years Tom had taken a lonely interest in the problem, and Keith became his house physician in time for the winter crop of 1950-1. Although not as infamous as 1952, from their point of view it was a good year. Whenever I visited

Chase Farm there were two or three patients with respiratory failure in the wards and, on occasion, there might be as many as a dozen. Ironically, the busier the hospital became the less easy it was to be sure of the diagnosis because there was not time to measure the blood gases.

Westlake became engrossed in the problem and stayed on at Chase Farm with the support of a British Medical Association Ernest Hart memorial fellowship. With Simpson and Kaye, Westlake performed a heroic feat of clinical investigation. They had a large and busy general medical service, which was further strained by a great many patients with respiratory failure. They had no technical help, and their equipment—as good as any then available—was primitive by today's standards. Using a temperamental pH meter and a Van Slyke apparatus, they followed the changes in PCO_2 and in cerebrospinal fluid pressure on giving oxygen. They recorded changes in PCO_2 , which at least one physiologist proclaimed must be incorrect because the values were incompatible with life. Clinicians were less surprised—I suspect because they hadn't even heard of PCO_2 .

The late '40s and early '50s were great years in clinical research—when the application of physiology greatly improved understanding and management of renal failure, liver failure, electrolyte disturbances, and so on. The techniques were often simple or even crude by today's standards, but such studies were an emotional strain. The patients were very ill; they came into hospital with little warning; and it was difficult to mobilise help in studying them. Hence for all these reasons it was difficult to observe a protocol, and, furthermore, one was often exploring in the dark, without the guidance of a clear hypothesis. In our work on respiratory failure we would usually study patients in the late evening or at night, and postpone those measurements that could wait. This approach might mean doing a dozen Van Slyke measurements of plasma CO_2 at the weekend.

Victims of enthusiasm

By 1951, the dilemma of acute on chronic respiratory failure was generally appreciated: the patients were at risk of death from anoxia, but oxygen administration might cause CO_2 narcosis. The fate of the patients swung rapidly. From being victims of neglect they became victims of enthusiasm. Oxygen was given by various devices according to various routines. One routine was to give it intermittently—with the idea that spells without it would stimulate the breathing and blow off the CO_2 . This routine was quite popular, despite Haldane's sensible dictum that intermittent oxygen therapy is like bringing a drowning man to the surface—occasionally.

We tried other approaches haphazardly: intravenous bicarbonate, to raise the blood pH; artificial ventilation; stimulants; and tracheostomy. The old tank ventilators left over from the polio epidemics, and the newer but primitive positive-pressure machines, did not seem to be able to cope with these patients, who "fought" them—probably because the machines did not

Ambrose Cardiorespiratory Unit, McMaster University, Hamilton, Ontario, Canada L8S 4J9

E J M CAMPBELL, MD, professor of medicine

provide enough ventilation. Furthermore, positive pressure was impossible to apply by mask to these restless, confused patients. Stimulants were nasty, but seemed sometimes to reverse the drift into CO₂ narcosis. The dramatic step, of course, was tracheostomy.

Tracheostomy in these patients was—and often still is—a miserable business. The trachea jerked up and down causing shearing forces between the tracheostomy tube, the trachea, and the soft tissues. A bruised, infected neck often resulted or, worst of all, erosion of the trachea. We now know that some damage to the trachea with stricture is common. Few patients survived tracheostomy, but we suspected that the poor results were due to delay in performing it. A surgical aphorism that acquired a certain currency was, "The right time to do a tracheostomy in these patients is when you first think of it." It was all very unsatisfactory and unpredictable, but then we couldn't measure anything to see what we were doing.

I spent 1955 at Johns Hopkins and returned able to do Riley's bubble method for PCO₂ and PO₂; I also had a grasp of the new understanding of the processes of gas exchange and their disturbances. In the bubble method, a very small bubble of gas was equilibrated with the blood in a 1 ml Roughton-Scholander syringe. This was a tuberculin syringe which had a 100 mm capillary on the nozzle. After equilibration, the bubble was gently moved into the capillary and its length was measured. The CO₂ and O₂ were then successively absorbed, and the changes in length enabled the partial pressures to be calculated. The whole procedure was delicate, because the bubble was liable to break up when being moved into the capillary; each analysis took nearly an hour and a disproportionate amount of nervous energy. The demands of the bubble method engendered a healthy respect for the value of blood gases which, old-fogey-like, I do not think is present in this time of easy electrode methods.

Late in 1955 Keith Westlake came to the Middlesex and we decided to try to tackle the problem of acute on chronic respiratory failure systematically, starting with stimulants. I wanted to try controlled oxygen, but the equipment for the bubble method would not be available for a few months and Keith was persuaded that the most important factor in the development of narcosis was central respiratory depression, which he thought, on the strength of a few preliminary studies, might be reversed by nikethamide or aminophylline.

Studies at the Middlesex

Our routine was to inspect the admissions record each morning and evening to see if any potentially suitable patients had been admitted. This inspection would be followed by a tour of the wards to see if any patient admitted as heart failure, pneumonia, or some other label was in fact suffering from undiagnosed respiratory failure. If we suspected that this was so, we would seek out the patient's consultant and obtain permission to proceed. Some of the consultants were much more dubious about arterial puncture than about the administration of drugs. Indeed, many of them regarded tracheostomy as perfectly laudable, but arterial puncture as quite unjustifiable. We would also try to have the patient moved to our so-called laboratory and persuade our ward sister to lend us a nurse for a couple of hours. Then, usually in the early evening, the study would begin.

Were this a Hollywood film script all this action would be shrouded in dense yellow fog, but in fact most of the patients came into hospital some days after the fog had lifted. Nevertheless, there were occasional night studies when all parts of the hospital were murky; one could not see one end of the corridor from another, and patients and staff were all coughing.

The protocol of our research was simple. A Riley arterial needle was inserted and two control samples of blood were taken. Their pH was measured and 5 ml were put in a screw-top bottle in the refrigerator for Van Slyke analysis later that night,

on the morrow, or whenever we could find time. Then the drug was given. In the case of aminophylline, we gave a large but standard dose. In the case of nikethamide, we had nothing to guide us and had to resort to pushing the dose until the patient's behaviour suggested that a stimulant level had been reached. Then there was often a hectic spell when the patient became agitated and restless. The twitches of the respiratory failure would rapidly change to much more extensive jerks, which made it difficult to keep the needle in place and very difficult to draw an arterial sample. (Today's indwelling catheters make life much easier.) I would try to take samples every two or three minutes during the peak of the drug action, and Keith would analyse them as quickly as he could. This frequency depended on the mood of the pH meter. If it was drifting or was difficult to balance, we might be able to get a credible reading only every five minutes or so. We had great difficulty in earthing both the machine and the operator, so that Keith had to stand barefooted in a tray of saline. Usually, either Keith was chastising me for my failure to get blood samples or I was niggling him for his failure to analyse them quickly enough. Occasionally, all went well and something like a protocol could be said to have been observed; more often the study had to be abandoned.

We managed only a handful of completed studies that winter. Keith died and I wrote them up. I am grateful to the *British Medical Journal* for publishing the paper. Today the study would have no chance of being supported by a granting agency, cleared by an ethical committee, accepted by a statistician, or published by a journal. Even so, it was certainly persuasive in showing that the use of stimulants was not the answer to the problem, so I set off to explore controlled oxygen. Technically, the difficulties of this study were, if anything, greater, and, physically, they were more exhausting, because they went on so long and the bubble method was very demanding. But I do not remember these studies with the same distaste as the stimulants which made both the patients and ourselves miserable, and which at no stage gave us any sense of discovery. However, nights spent with vacuum cleaners driven backwards into bags hanging over a patient's head (the prelude to the HAFUE principle and the Venturi mask) cannot really be taken as typical of the hospital experience of a patient in respiratory failure in the early '50s. It's another story.

STRANGE ENCOUNTERS

Post mortem—Superbeast

The film *Superbeast* was the last of last summer's late-night horror movies on BBC television. It concerned a "famous pathologist" who, in her search for the explanation of some evidently unprecedented necropsy findings, flew to the Philippines, where the nonsensical plot was played out against occasional glimpses of superb location backgrounds. The colour photography was excellent. Although the initial pathological observations were not described in any detail—perhaps because a predominantly non-medical audience would not yet understand them readily—the necropsy was not left to our imagination but illustrated by two sequences filmed during a genuine PM.

The body of a somewhat obese coloured woman was shown while the midline incision from manubrium to hypogastrium was made; in the second sequence, gas-distended loops of small bowel were seen in situ, with much bloody fluid in the peritoneal cavity. Later, "Dr Alix," the pathologist, was seen in a laboratory, peering at some viscera in a clear cylindrical jar: they included a heart that may well have been human. The actress's expression seemed to convey the resigned vexation of any competent, serious pathologist who finds that she is expected to make sense of organs that have been stuffed into too small a container.

Is it ever appropriate that a real PM be shown on broadcast television or in a public cinema? Of course not, and to include such shots in a fictional presentation is not only unnecessary but tasteless and possibly harmful. Who provides the facilities that enable entertainers to record the procedure?—WILL MACREDIE.