Short reports

Deoxyhaemoglobin concentrations in the detection of central cyanosis

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Central cyanosis is an important clinical sign because it indicates the presence of appreciable arterial hypoxaemia. According to classical textbook teaching, concentrations of deoxyhaemoglobin of about 5 g/dl are necessary before central cyanosis is clinically detectable. This figure has recently been questioned and thought to be a considerable overestimation. The aim of this study was to re-examine the relationship between deoxyhaemoglobin concentration and central cyanosis and to determine the minimum deoxyhaemoglobin concentration required for the detection of central cyanosis.

Methods

Eighty normothermic patients (60 of them male) with various acute and chronic respiratory disorders referred for routine arterial blood gas analysis were studied during normal working hours. Most patients were breathing air when the samples were taken but 20 were receiving supplemental oxygen. Pigment disorders (methaemoglobin or sulphaemoglobin) were not present in any patient. Arterial blood was collected in preheparinised syringes by standard techniques and delivered immediately to the laboratory in ice for analysis. Oxygen and carbon dioxide tensions and pH were measured with a blood gas analyser (Radiometer BMS Mk2). Haemoglobin concentration and arterial oxygen saturation were measured with a haemoximeter (Radiometer OSM2). All measurements were made at 37°C. Both instruments were recalibrated and variation in repeat measurements did not exceed 0.1 g/dl (haemoglobin), 0.1% (saturation), and 2 mm Hg (0.3 kPa) (oxygen tension).

Each patient was examined independently by two observers for the presence of central cyanosis in ordinary ward daylight conditions. The observers had no prior knowledge of the patients' measured results. Central cyanosis was defined as being present if the tongue and buccal mucous membranes appeared blue and the patient was not breathing by mouth.

Results

Twenty-nine of the 80 patients studied were assessed as being centrally cyanosed. In only three cases did the observers disagree about the presence or absence of cyanosis. After re-examination two patients (arterial oxygen tension (Pao2) 60 mm Hg (8.5 kPa), deoxyhaemoglobin (deoxyHb) 1.0 g/dl) and Pao2 81 mm Hg (11 kPa), deoxyHb 0.9 g/dl) were recorded as not displaying central cyanosis and the third as being cyanosed (Pao2 57 mm Hg (8 kPa), deoxyHb 1.6 g/dl).

Cyanosed patients were significantly more hypoxaemic and had a significantly higher deoxyhaemoglobin concentration than non-cyanosed patients (mean(SD) for cyanosed: Pao2 75.0 (10.4) mm Hg (8.1 kPa), deoxyHb 1.9 g/dl; and for non-cyanosed: Pao2 81.3 (12.7) mm Hg (11.5 kPa), deoxyHb 0.5 g/dl; p < 0.001). In both groups the mean haemoglobin concentration was 12.7 g/dl. A plot of deoxyhaemoglobin concentration against arterial oxygen tension is shown in figure 1; the minimum deoxyhaemoglobin concentration in any centrally cyanosed patient was 1.1 g/dl and the maximum concentration for the non-cyanosed patients was 1.3 g/dl. There was no relationship between total haemoglobin and deoxyhaemoglobin concentrations (figure 2) and, at least within the haemoglobin concentration range 16-20 g/dl, the presence of cyanosis could not be related to the total haemoglobin concentration.

![Deoxyhaemoglobin concentration vs arterial oxygen tension](image)

Fig 1 Relationship between deoxyhaemoglobin concentration and arterial oxygen tension.

1 mm Hg = 0.133 kPa.
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Deoxyhaemoglobin conc. (g/dl)  
• cyanosed  
○ non-cyanosed

![Graph](https://example.com/graph.png)

Fig 2  Relationship between deoxyhaemoglobin and total haemoglobin concentration.

Discussion

The results of this study show that patients with central cyanosis were significantly more hypoxaemic and had significantly higher concentrations of deoxyhaemoglobin than patients who were not cyanosed. Our results also show that central cyanosis can be detected when deoxyhaemoglobin levels are 1-1 g/dl or greater; at concentrations of 1-5 g/dl or more central cyanosis was recognised in all patients.

In their monograph on cyanosis Lundsgaard and von Slyke concluded that a deoxyhaemoglobin concentration of about 5 g/dl was necessary before cyanosis could be detected. Flenley, however, has pointed out that analysis of the haemoglobin oxygen dissociation curve leads to the conclusion that this is a considerable overestimate. He argued that a deoxygenated haemoglobin concentration of about 1-5 g/dl is a more realistic estimate because at this concentration arterial saturation will be about 90% (given a haemoglobin level of 15 g/dl) and arterial oxygen tension 60 mm Hg (8-3 kPa). Barnett and coworkers reported the presence of central cyanosis in 20 of 178 hypoxaemic patients studied under normal ward conditions. They found that the cyanosed patients were significantly more hypoxaemic and had significantly higher deoxyhaemoglobin concentrations (mean 3-48 g/dl) than the non-cyanosed group (mean 1-02 g/dl). These results showed for the first time that central cyanosis could be detected reliably at deoxyhaemoglobin concentrations of less than 5 g/dl. These workers reported a 95% confidence limit for the threshold of detection at about 2-38 g/dl.

The results of our study are not unexpected. They confirm the previously reported and well known relationships between central cyanosis and both arterial hypoxaemia and the concentration of deoxyhaemoglobin. The important new finding in our study was that central cyanosis can be detected reliably at deoxyhaemoglobin concentrations of 1-5 g/dl or more, thus confirming Flenley's conclusions, with a threshold deoxyhaemoglobin concentration for detecting central cyanosis in the range 1-0–1-5 g/dl. These values are lower than the concentration suggested by Barnett et al. The likely explanation for the discrepancy is that they calculated the threshold concentration from group mean results and a 95% confidence limit, whereas we have specifically plotted our data to identify threshold values. We could also argue that this discrepancy might be due to differences in haemoglobin concentrations. In both studies, however, the mean haemoglobin concentrations were similar, and furthermore we were not able to identify any effect of the haemoglobin concentration on the detection threshold within the haemoglobin concentration range 11–16 g/dl (fig 2). Further studies are needed to examine this relationship in polycythaemia and anaemia. Figure 1 shows that deoxyhaemoglobin concentrations of 1-0–1-5 g/dl were found in both cyanosed and non-cyanosed patients. Although we believe that the actual critical threshold lies at the lower end of this range a “grey zone” is not unexpected, given the well recognised physical and perceptive limitations in the detection of cyanosis.

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References