# Pulse Oximetry in CO Poisoning—Additional Data

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## A Kinky Catheter

*To the Editor:*

Complications when using the balloon-tipped, flow-directed pulmonary artery catheter (PAC) abound. We wish to report an unusual episode of acute angulation of the PAC in the right internal jugular vein (IJV).

A 68-year-old man with poor left ventricular function (ejection fraction, 30%) was scheduled for elective coronary artery bypass grafting. Preinduction, both the central venous pressure catheter and PAC (7.5F thermodilution catheter, model 1755HD, 21–000007-93C; Biosensors International Pte Ltd; Singapore) were inserted into the right IJV via a double-puncture technique. We were unable to float the PAC into the pulmonary artery. On the third attempt, it was noted that the PAC had been advanced to the 50-cm mark. Subsequent withdrawal of the catheter was met with a definite resistance at the 20-cm mark. No waveform was obtained, and the aspiration of blood was unsuccessful. The chest radiograph (Fig 1) revealed an acute angulation of the PAC at the end of its introducer in the right IJV. After discussion with the surgeon, a decision was made to unfurl the PAC by opening the right atrium intraoperatively. The kink in the PAC is shown in Figure 2. Surgery proceeded uneventfully.

This case highlights the possibility of acute angulation of the PAC loop in the IJV itself. Acute angulation has been described when a PAC was inserted into the external jugular vein (EJV).[1] This is attributed to the variations in the EJV-subclavian vein (SCV) course. Theoretically, this angulation also could occur using the right SCV.

Boyd et al[2] reported an incidence of 0.1% for the inability to wedge the PAC and an incidence of 0.2% for catheter looping. As is widely reported, multiple attempts at floating the PAC and floating too long a section of the PAC should be avoided. In our case, we postulate that the PAC had looped in the right ventricle and retraced its path back into the right IJV. During withdrawal, the loop tightened, causing the acute angulation that resulted in complete occlusion of the PAC.

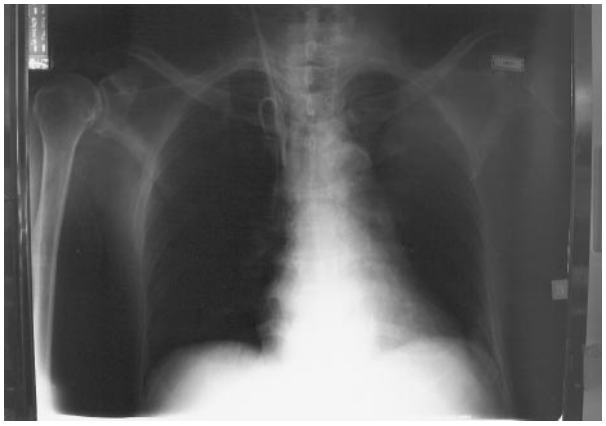

FIGURE 1. Chest radiograph showing the acute angulation of the PAC in the right IJV at the end of the sheath.

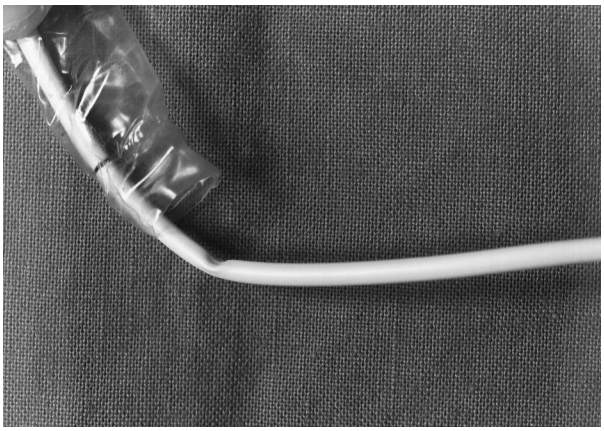

FIGURE 2. The kink in the PAC.

We considered using a guidewire to push the PAC into the right atrium then straightening it, but chose to induce the patient and solve the problem by opening the right atrium intraoperatively.

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## Knowledge and Beliefs of Samples of the General Population About Asthma

*To the Editor:*

Asthmatic teenagers belong to a high-risk population, mainly because they usually have a low level of therapeutic compliance.[1] Many case-control studies have exemplified personal characteristics that could be responsible for this behavior.[2,3] In this context, it seems useful to know better the beliefs of teenagers about asthma compared to other acute and chronic diseases.

This survey was performed in the framework of the International Study of Asthma and Allergies in Childhood (ISAAC).

In the Marseille area, 12 secondary schools (165 classes) were randomly selected, consisting of 4,186 teenagers. Of these, 3,495 (83.5%) agreed to participate to the study. After they had completed the standard ISAAC questionnaire in class, teenagers were asked to complete a questionnaire concerning their beliefs about six diseases: asthma, cancer, tuberculosis, influenza, the common cold, and chicken pox. Teenagers had to provide a grade between 1 (not at all) and 10 (very much) for rating the disease on the following attributes: severe, contagious, hereditary, patient is responsible for it, bashful, well-known, unusual, curable, disgusting, disabling in everyday life, and annoying for other people. The response rate was fair, between 68.5% and 81.4% according to the disease, except for questions on tuberculosis, which were answered by only 35% of the study group.

Figure 1 provides the average (± SD) rating for asthma. Asthma is seen as a rather common, severe, well-known, and hereditary disease, which are attributes that fit well with the disease. Boys (49.6% of the study population) had a significantly more optimistic view about asthma: less severe disease, greater probability of cure, and less impact on everyday life. There was no difference in rating according to age, which ranged between 12 and 15 years, and according to socioeconomic status. Asthmatic children see the disease as more hereditary but, paradoxically, are more optimistic about the possibility of a cure. Figure 2 shows how asthma compares to other diseases with regard to beliefs and knowledge. Teenagers' responses about these diseases also fit with general opinion.

No survey has been devoted to this topic in the international literature. In 1992, we performed a street survey among 800 individuals visiting a public exhibition in Marseille.[4] These visitors, who were mainly young and middle-aged adults, were asked to complete a short questionnaire dealing with their beliefs about asthma. Respondents stated that asthma is a common, severe, and long-lasting disease.

In conclusion, this survey demonstrates that knowledge of and beliefs about asthma in a large group of teenagers are fair.

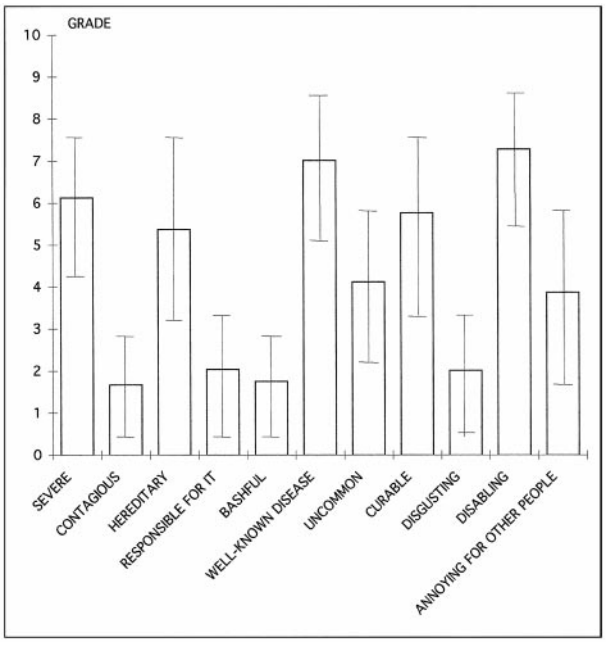


FIGURE 1. Shows beliefs of teenagers about asthma graded (mean ± SD) on a scale of 1 (not at all) to 10 (very much).

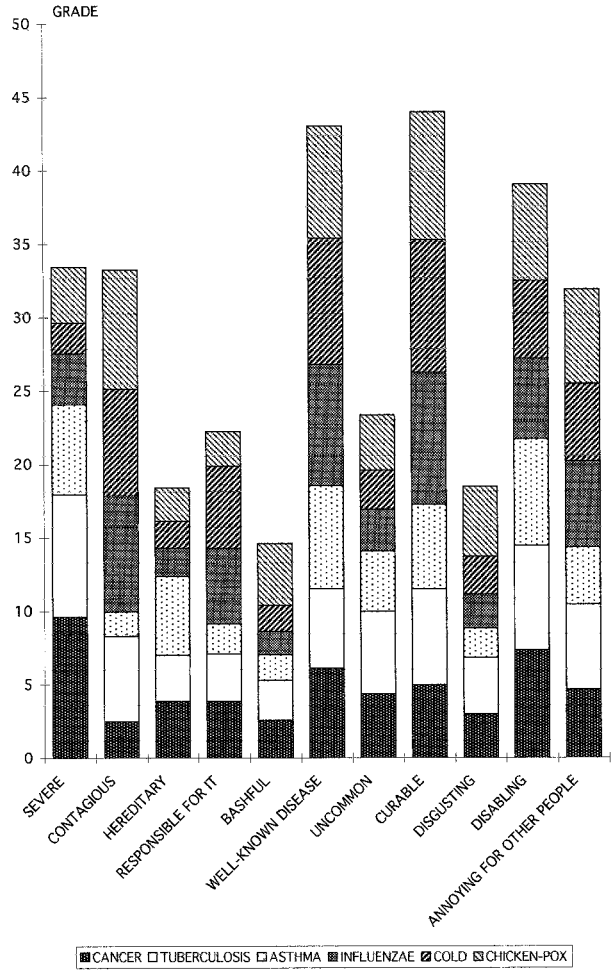


FIGURE 2. Shows beliefs about asthma compared to other diseases graded (mean ± SD) on a scale of 1 to 10. Total score equals 50 because each gradation runs from 1 to 10.

There are no major mistakes or misapprehensions in their knowledge and beliefs about the disease. Thus, health education is unlikely to have a major impact on asthma management in teenagers.

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## Sarcoidosis and IFN-α Treatment

*To the Editor:*

I read with interest the report by Pietropaoli et al (August 1999)[1] of a woman who developed sarcoidosis during treatment of chronic myelogenous leukemia with interferon (IFN)-α. I recently treated a similar patient and found two others reported in the literature.[2,3]

My patient was a 39-year-old woman who was admitted to Memorial Hospital in May 1999 for fever, polyarthralgias, and erythema nodosum. Four weeks earlier she had completed a 1-year course of IFN-α (3 million units tiw), given with 6 months of ribavirin (1 g qd) as treatment for hepatitis C. In the past she had a self-limited thyroiditis. Her chest radiograph demonstrated new hilar adenopathy without parenchymal disease. The results of a skin biopsy were compatible with erythema nodosum. Her angiotensin-converting enzyme (ACE) level was 155 U/L. The results of pulmonary function tests were normal. A transbronchial needle aspiration of the right hilar lymph node revealed granulomas, as did endobronchial and transbronchial biopsies. She received prednisone, 40 mg daily, giving her rapid relief from fever and joint pain. The dosage of prednisone has been tapered. After 8 weeks, her ACE level was 46 U/L.

In the two additional cases in the literature, one patient had received IFN-α for hepatitis C.[2] She had pulmonary and cardiac involvement with the development of complete atrioventricular block. The other patient had been treated with IFN-β for multiple myeloma.[3]

As noted by the authors, α- and β-interferon have not been classified in the pathogenesis of sarcoidosis, but *in vitro* they can activate alveolar macrophages in sarcoidosis patients.[1]

Our patients seem to have had an iatrogenic stimulation of the immunologic pathway of sarcoidosis. Whether they have a genetic disposition or have had an exposure to the presumed antigen is speculative. It would be interesting to have them undergo Kveim testing.

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## Histamine $PC_{20}$ Extrapolation

*To the Editor:*

We read with interest the article by Jokic et al (December 1998)[1] because we also have investigated simple linear extrapolation for the estimation of the provocative concentration of a substance causing a 20% fall in $FEV_1$ ($PC_{20}$) in histamine and methacholine challenge tests. Our conclusions were remarkably similar to those of Jokic et al, although our method of calculation was slightly different.

In our study, we reviewed 490 consecutive histamine challenges performed according to the tidal breathing method of Cockcroft et al.[2] In the 184 positive challenges ($PC_{20}$, $< 8$ mg/mL) we compared the conventional $PC_{20}$ using linear interpolation (PCint) to the $PC_{20}$ calculated by linear extrapolation of the dose-response curve (PCext). The PCext was calculated in the same manner as in the study by Jokic et al, in that we assumed that the last histamine concentration (the one that induced a fall in $FEV_1$ of $> 20\%$) had not been administered, then we extrapolated using the last two remaining data points. The same equation was used for both calculations of $PC_{20}$.

Generally, there was a poor correlation between the interpolated and extrapolated $PC_{20}$ calculations ($R^2 = 0.25$). However, if we selected only those subjects with a fall in $FEV_1$ of $> 10\%$ to the penultimate histamine concentration (n = 123), the correlation between the $PC_{20}$ calculations improved ($R^2 = 0.79$) (Fig 1). When only subjects with a fall in $FEV_1$ of $> 15\%$ to the penultimate histamine concentration were included in the analysis (n = 55), the relationship between the $PC_{20}$ calculations tightened ($R^2 = 0.95$) (Fig 2).

In this group, the extrapolated $PC_{20}$ tended to slightly overestimate the interpolated value by approximately 15%. Correction according to the equation $PC_{20}int = 0.81*PC_{20}ext + 0.16$ accounted for this overestimation. Using this correction in calculations for the 55 subjects, there was almost no difference in the categorization of airway hyperresponsiveness between the two methods. All six subjects with severe airway responsiveness ($PC_{20}$, $< 0.25$ mg/mL) based on interpolated $PC_{20}$ remain in that category if the corrected extrapolated $PC_{20}$ is substituted. The same was found in all 18 subjects categorized as having moderate airway responsiveness ($PC_{20}$, 0.25 to 2 mg/mL). Only one subject in the mild category was redefined when the extrapolated $PC_{20}$ was used. This subject had a borderline increase in airway responsiveness with an interpolated $PC_{20}$ of 7.5 mg/mL and a corrected extrapolated $PC_{20}$ of 8.1 mg/mL.

We conclude that linear extrapolation using the last two data

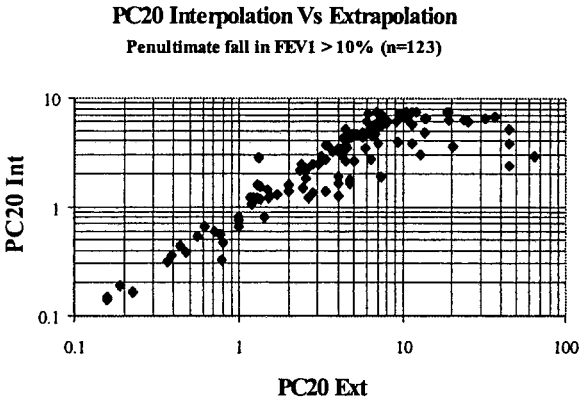


FIGURE 1. The correlation between the $PC_{20}$ calculations with an $R^2$ of 0.79.

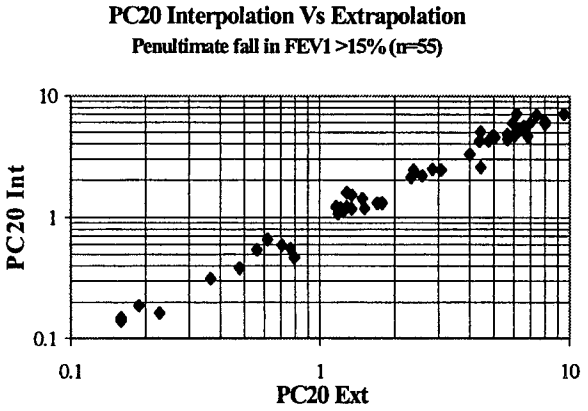


FIGURE 2. The correlation between the $PC_{20}$ calculations with an $R^2$ of 0.95.

points should be used only if the fall in $FEV_1$ has exceeded 15%. A correction formula can be used to compensate for the overestimation of the true $PC_{20}$ by the extrapolated $PC_{20}$. Use of this method will negate the necessity of inducing large and potentially dangerous falls in $FEV_1$, which sometimes are required to calculate an interpolated $PC_{20}$, with negligible effect on the interpretation of the challenge test. It will also allow calculation of an accurate $PC_{20}$ in research trials in those subjects who fail to meet the 20% fall in $FEV_1$ criteria.

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*To the Editor:*

We were pleased to read the commentary by Eckert and Mitchell regarding our article (December 1998)[1] on the extrapolation of the provocative concentration of a substance causing a 20% fall in $FEV_1$ ($PC_{20}$). The data presented in the bottom graph of Figure 1 in our article were similar to their data, *ie*, we used the penultimate fall in $FEV_1$ ($\geq 15\%$, $< 20\%$) and the antipenultimate fall in $FEV_1$ to extrapolate the methacholine $PC_{20}$, and we compared this to the conventional interpolated $PC_{20}$. When analyzed in the same way as Eckert and Mitchell's data, the Pearson correlation coefficient (r) was 0.91. The interclass correlation coefficient (ICC), a more appropriate test to look for *equivalence* as opposed to *correlation*,[2] was 0.74. The geometric mean extrapolated $PC_{20}$ was 12% larger than the interpolated value. However, our data suggest that this was not a systematic overestimation but rather was due to a large overestimation in a small number of individuals (8 of 100) who had a very shallow slope on the extrapolation line (see Fig 1 in our article[1]). In fact, it was this reason that prompted us to look at a one-point extrapolation using only the penultimate fall in $FEV_1$. This approach to extrapolation proved to be a better approach to approximating the interpolated $PC_{20}$ ($r = 0.995$; $ICC = 0.97$).[1] There was a slight tendency (8%) for this approach to underestimate $PC_{20}$. The accuracy of extrapolation will decrease the smaller is the fall in the $FEV_1$. Extrapolation will be unreliable for falls in $FEV_1 < 10\%$ and will be of borderline reliability with falls between 10% and 15%. Our data as well as that of Eckert and Mitchell document that extrapolation is a reliable way to estimate $PC_{20}$ when the fall in $FEV_1$ is $\geq 15\%$.

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# Pulse Oximetry in CO Poisoning—Additional Data

*To the Editor:*

I read with interest Dr. Hampson's retrospective review (October 1998)[1] of a decade of experience with severe carbon monoxide (CO) poisoning. His series revealed 15 patients who had concurrent pulse oximetry and spectrophotometrically measured arterial oxygen saturation readings. The pulse oximetery gap, which is produced by carboxyhemoglobin and represents the difference between the pulse oximetry reading and the actual oxyhemoglobin saturation, is demonstrated to be a real and clinically important phenomenon. The report is well written and contributes to an area that historically has had a dearth of experimental data.

Unfortunately, there was a significant omission in the "extensive English-language literature search" performed. This omission led the author to incorrectly assess that only seven humans previously had been demonstrated to have a pulse oximetry gap with high levels of carboxyhemoglobin. This search did not reveal our existing report, published the year before, of 124 patients with CO exposure and a determination of the pulse oximetry gap by essentially the same methodology.[2] Twenty-two of these patients had carboxyhemoglobin levels from 20 to $> 50\%$. With a greater number of data points, we were able to show a very clear linear relationship between CO levels and the pulse oximetry gap. Our series included mild, moderate, and severe cases of CO poisoning. Dr. Hampson's report confirms this finding in severe cases and adds valuable additional data to our collective experience with this condition.

Physicians who may treat CO intoxication must be aware of the profound limitations of pulse oximetry in this setting. High-flow oxygen should be administered to all patients suspected of

significant CO exposure until direct measurement of CO levels can be performed, regardless of pulse oximetry readings.

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2 Bozeman WP, Myers RAM, Barish RA. Confirmation of the pulse oximetry gap in carbon monoxide poisoning. Ann Emerg Med 1997; 30:608–611

*To the Editor:*

Dr. Bozeman expresses concern that his study,[1] published in November 1997, was not noted in my article (October 1998).[2] As indicated on the first page of my article, my manuscript was submitted in its original form on October 2, 1997. It is only possible to reference material that is published at the time of manuscript preparation. I would like to assure Dr. Bozeman that I would have referred to his excellent study had it been available.

I am, however, concerned that Dr. Bozeman's interpretation of his own data continues the perpetuation of the myth that my investigation clearly disproved. It often has been stated that pulse oximeters overestimate true arterial hemoglobin oxygen saturation by the amount of carboxyhemoglobin (COHb) present. Examining 30 patients with extreme elevations of COHb (> 25%), I demonstrated that this difference (the "pulse oximetry gap") was less than the COHb concentration in 19 patients (73%). While pulse oximeters overestimate the arterial hemoglobin oxygen saturation, the amount is not equal to the COHb level.

Reviewing Dr. Bozeman's graphed data in Figure 2 of his article, 13 individuals appear to have COHb levels > 25%. Among these patients, nine (69%) appear to have pulse oximetry gap values that are lower than the COHb level. In my article, I used as an example an individual with a COHb level of 50% and indicated that the pulse oximetry gap would be approximately 5% less than the COHb level (*ie,* 45%). Using the regression equation published in Dr. Bozeman's paper, he would predict a pulse oximetry gap value of 45.85%.

Thus, while pulse oximeters do overestimate true arterial hemoglobin oxygen saturation, it is by an amount that is *less* than the COHb level. Dr. Bozeman's data confirm my finding in this regard. It should not be surprising that the two values are not equal. Pulse oximeters rely on the differential absorption of light to make their measurement. The absorption spectra of carboxyhemoglobin and oxyhemoglobin are not identical at the wavelengths utilized.

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2 Hampson NB. Pulse oximetry in severe carbon monoxide poisoning. Chest 1998; 114:1036–1041

## Erratum

In the July 1999 issue of *CHEST*, the article "Lymphocyte Glutathione Levels in Children With Cystic Fibrosis" by Lands et al contained lymphocyte glutathione levels that were reported using the incorrect unit μmol/$10^6$ cells. The correct unit is nmol/$10^6$ cells, which is equivalent to μmol/L.