PAPER

Normal jugular bulb oxygen saturation

A Chieregato, F Calzolari, G Trasforini, L Targa, N Latronico

J Neurol Neurosurg Psychiatry 2003;**74**:784–786

See end of article for authors' affiliations

Correspondence to: Dr A Chieregato, Via Boccacanale S. Stefano no. 25, 44100 Ferrara, Italy; achiere@ausl-cesena.emr.it

Received 31 May 2002
In revised form 9 October 2002
Accepted 2 December 2002

Background: Normal values of the jugular bulb oxygen saturation were obtained in 1942 and in 1963. Correct catheter positioning was not confirmed radiologically.
Objectives: To replicate the measurements during angiographic catheterisation of the jugular bulb.
Methods: Oxygen saturation in the jugular bulb (SjO_2), inferior petrosal sinus ($SipsO_2$), and internal jugular vein was bilaterally measured in 12 patients with Cushing's syndrome undergoing selective bilateral catheterisation of the inferior petrosal sinus. In addition, data from the two old series were reanalysed for comparison.
Results: SjO_2 values (44.7%) were significantly lower than in the two old series, particularly concerning the normal lower limit (54.6% and 55.0% respectively). Comparative analysis suggests that contamination with the extracerebral blood of the facial veins and inferior petrosal sinuses was responsible for falsely high SjO_2 values in the two old series.
Conclusions: The normal lower SjO_2 limit is lower than previously recognised. This may have practical implications for treating severe head trauma patients.

Monitoring of jugular bulb vascular oxygen saturation (SjO_2) is currently suggested in patients with severe head trauma as a global measure of the adequacy of the cerebral blood flow (CBF) to brain metabolic requirements.[1] SjO_2 in normal volunteers was first obtained by Gibbs *et al* in 1942,[2] and then by Datsur *et al* in 1963.[3] In 1945 Gibbs *et al* also showed that the SjO_2 does not differ in simultaneous sampling of both right and left jugular bulbs.[4] However, these physiological data were obtained by direct puncture of the jugular veins without radiological confirmation of the needle's or catheter's tip position.

Substantial extracerebral contamination with high oxygen saturation values may occur if the blood is withdrawn from the facial vein, joining a few centimetres below the jugular bulb and possibly from the inferior petrosal sinus, joining immediately below the jugular bulb. Contamination may also occur if the catheter is correctly positioned and blood withdrawal is too fast. Therefore, extracerebral blood contamination could have occurred in the two old series, and falsely high oxygen saturation values could have been obtained.

We sought to replicate the SjO_2 measurements during angiographic catheterisation of the jugular bulb.

MATERIALS AND METHODS

The study was approved by the local review board, and written informed consent was obtained by the patients. The study was carried out on 12 patients (nine females; median age 39 years, range 25–72) with Cushing's syndrome, whose aetiology had to be defined. Selective bilateral venous sampling in the inferior petrosal sinus was necessary to distinguish those individuals with microadenoma of the pituitary from some paraendocrine tumours (for example, carcinoids or phaeochromocytoma) that ectopically produce corticotrophin releasing hormone (CRH) or adrenocorticotrophin hormone (ACTH).[5] We took advantage of this procedure, which was done using a digital subtraction angiography system, to obtain multiple bilateral samples from: (1) internal jugular veins at the level of the 5th cervical vertebra (midlevel) ($SmidjO_2$); (2) inferior petrosal sinuses ($SipsO_2$); and (3) dome of internal jugular vein bulbs (SjO_2). Correct position of the tip of the catheter was meticulously checked under direct angiographic observation with contrast medium injection. Blood was withdrawn slowly (1 ml per minute)[6] and venous blood was analysed for oxygen saturation with a co-oximeter (IL 482, CO-Oximeter, Instrumentation Laboratory, Milan, Italy).

Patients were excluded if they had abnormal arterial oxygen saturation (<97% in room air), anaemia of recent onset (Hb less than 100 g/l in the past 10 days), or haemodynamic instability. Chronic arterial hypertension is associated with an adaptation of CBF, and metabolic coupling is ultimately affected only by severe involvement of the cerebral vascular circulation.[3 7] Therefore, stable hypertension was not an exclusion criterion. Conversely, patients with hypertension of recent onset or with poorly controlled hypertension were excluded. Patients were also excluded if they had evidence of acute or chronic diseases affecting the central nervous system (CNS) other than microadenoma of the pituitary, or if they had hyperventilation (more than 25 breaths per minute for at least one minute) during the procedure.

Pain and agitation were carefully avoided because they could induce ACTH secretion and cerebral metabolic rate of oxygen ($CMRO_2$)/CBF changes, both of which were undesirable. The procedure was carried out in a noise free environment, and accurate local anaesthesia was performed before venous cannulation. Arterial blood pressure was measured at five minute intervals by using an automated device with cuff inflation. Arterial oxygen saturation (SaO_2) was continuously monitored using a pulse oximeter.

Statistical analysis

SjO_2 values are presented as mean, upper (mean plus 2 standard deviations (SD)), and lower (mean minus 2 SD) limit. Precision of each point estimate was evaluated by 95% confidence intervals (95% CI).[8] Comparison of the venous oxygen saturation in the jugular bulbs, inferior petrosal sinuses, and midlevel jugular veins was done using analysis of variance (ANOVA).

SjO_2 values from this series were compared to those from the two historical series by Gibbs and colleagues (50 healthy

Abbreviations: ACTH, adrenocorticotrophin hormone; ANOVA, analysis of variance; CBF, cerebral blood flow; $CMRO_2$, cerebral metabolic rate of oxygen; CNS, central nervous system; CRH, corticotrophin releasing hormone; SjO_2, jugular bulb oxygen saturation; $SipsO_2$, inferior petrosal sinus oxygen saturation

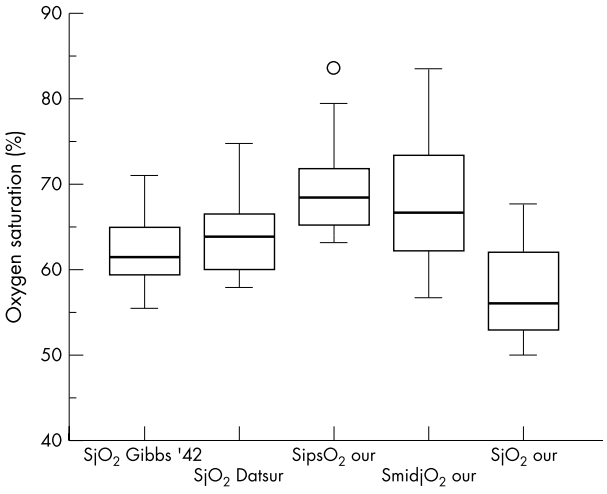

Figure 1 Oxygen saturation in the jugular bulb (SjO_2) in the series by Gibbs (1942) and Datsur, and in the inferior petrosal sinus ($SipsO_2$), midlevel jugular vein ($SmidjO_2$), and jugular bulb in the present series.

young volunteers)[2] and Datsur and colleagues (26 healthy elderly volunteers).[3] Data from the three series were compared by means of ANOVA.

Analysis of differences between right and left SjO_2 in simultaneous samples was done using paired Student's *t* test. In addition, the degree of agreement was evaluated according to Bland and Altman.[9] The same procedure was applied to the data published in 1945 by Gibbs *et al*, in which bilateral measurements were performed in 25 patients with various psychiatric disorders.[2] As suggested by the authors, seven patients were excluded because of data inconsistency.

In all cases two tailed tests were used and $p < 0.05$ was chosen to define statistically significant differences. Box plots were used for graphic presentation.

RESULTS

SaO_2 and arterial blood pressure remained stable during the procedure. In two patients simultaneous catheterisation of jugular bulbs was not possible because of severe ear pain, in one the procedure was interrupted due to a contrast medium anaphylactic reaction, and one had hyperventilation after CRH administration. Overall, 143 measurements were made in 12 patients. Haemoglobin was 130 g/l (SD 22).

Oxygen saturation was significantly lower in samples from the jugular bulb compared to inferior petrosal sinus and midlevel jugular vein samples ($F = 8.4451$, $p = 0.0014$). Interestingly, the SjO_2 values in the series by Gibbs and Datsur were intermediate between SjO_2 and $SipsO_2$–$SmidjO_2$ in the present series (fig 1).

SjO_2 values in the present series were significantly lower than in the two series by Gibbs and Datsur ($F = 9.4998$, $p = 0.0002$). Table 1 presents mean values, upper and lower limits, and 95% CI.

Differences between right and left SjO_2 in simultaneous samples were not statistically significant, neither in the present series ($t = 0.1978$, two tailed $p = 0.8481$) nor in the Gibbs series ($t = -0.1464$, two tailed $p = 0.8846$); however, limits of agreement were wider in the latter. Results, expressed as mean difference (95% CI), and upper (95% CI) and lower (95% CI) limits of agreement were as follows: present study: 0.2% (−1.2 to 1.7); 3.7% (1.2 to 6.3); −3.3% (−5.8 to −0.7); Gibbs: −0.1% (−1.3 to 1.1); 6.5% (4.4 to 8.6); −6.7% (−8.8 to −4.6).

DISCUSSION

We found significantly lower SjO_2 values in this series (44.7%) than reported in two old series (54.6%, 55.0%), the only ones in which SjO_2 was measured in normal volunteers. The difference was not accounted for by the fact that patients in the present series had Cushing's syndrome. In fact, all patients had a syndrome of recent onset, whose pathogenesis, whether due to microadenoma of the pituitary or paraendocrine tumours, had yet to be defined. In addition, patients with severe unstable hypertension, in whom uncoupling of CBF and $CMRO_2$ may occur, were excluded.

Three facts are relevant to explain our results. First, we used a digital angiography system and vascular catheters were exactly positioned in the dome of the jugular bulb. In the Gibbs and Datsur series the catheter positioning in the jugular bulb was blind, and cervical *x* ray confirmation of the final catheter position was not performed. Therefore, extracerebral blood contamination from the facial veins,[10] and as shown in this study, from the inferior petrosal sinuses may have occurred in some patients. Our results support this hypothesis showing that the SjO_2 values in the old series were intermediate between SjO_2 and $SipsO_2$–$SmidjO_2$ (extracerebral) in the present series. Second, blood samples in our study were withdrawn slowly, a factor that has been showed to avoid extracerebral blood contamination, provided that the catheter is correctly positioned in the jugular bulb.[6] Third, we measured haemoglobin oxygen saturation by mean of a co-oximeter, the reference method based on three wavelength spectrophotometry,[11] while Datsur *et al* used a two wavelength spectrophotometric method,[12] and Gibbs *et al* the Van Slyke manometric method.[13] The two latter methods overestimate oxygen saturation compared with the co-oximeter, particularly for the lower range of oxygen saturation, as is the case in venous samples.[11]

Normal SjO_2 limit lower than previously recognised: does it matter?

SjO_2 values below the lower normal limit have been defined as jugular desaturation—that is, situations of critical inadequacy of CBF to $CMRO_2$, "from norms defined by Gibbs and coworkers",[14] and have been proven in a single study to independently affect the outcome of severe head trauma.[15] However, the methods on how to summarise insult data such as jugular desaturation have only recently been defined,[16] while in previous studies the jugular desaturation was simply dichotomised as present or absent, based on Gibbs' data. Therefore, a new limit could modify the result. More

Table 1 Oxygen saturation values in the jugular bulb (SjO_2)

	SjO_2		
	Mean (95% CI)	Upper limit (95% CI)	Lower limit (95% CI)
Present series	57.1 (52.3 to 61.6)	69.5 (61.2 to 77.7)	44.7 (36.5 to 53.0)
Gibbs (1942)	62.0 (61.0 to 63.1)	69.4 (67.6 to 71.2)	54.6 (52.8 to 56.5)
Datsur (1963)	64.3 (62.4 to 66.2)	73.7 (70.4 to 76.9)	55.0 (51.7 to 58.2)

importantly, it is suggested that jugular desaturations, variously defined as SjO_2 less than 50% or 54%,[1] are treated aggressively, although benefits are unproven. A new normal limit further weakens the legitimacy of such treatments.

This study included a small sample of patients and therefore conclusions should be taken cautiously. Due to the lack of invasiveness that can be attributed to the method itself, our results can be easily replicated. If the normal lower SjO_2 limit is confirmed to be as low as 46%, redefinition of "normality" applied to SjO_2 may have diagnostic, prognostic, and therapeutic implications.

Authors' affiliations
A Chieregato, L Targa, Department of Anesthesiology-Intensive Care, Section of Neurocritical Care, Ospedale Civile M. Bufalini, Cesena, Italy
F Calzolari, Department of Neuroradiology, Arcispedale S. Anna, Ferrara, Italy
G Trasforini, Department of Clinical Endocrinology, University of Ferrara, Italy
N Latronico, Institute of Anesthesiology-Intensive Care, University of Brescia, Italy

Competing interests: none declared

REFERENCES

1 **Macmillan CSA**, Andrews PJD. Cerebrovenous oxygen saturation monitoring: practical considerations and clinical relevance. *Intensive Care Med* 2000;**26**:1028–36.
2 **Gibbs EL**, Lennox WG, Nims LF, *et al*. Arterial and cerebral venous blood. Arterial-venous differences in man. *J Biol Chem* 1942;**144**:325–32.
3 **Datsur DK**, Lane MH, Hansen DB, *et al*. Effects of aging on cerebral circulation and metabolism in man. In: Birren JE, Butler RN, Greenhouse SW, *et al*, eds. *Human aging. A Biological and behavioral study*. Washington, DC: US Government Printing Office, 1963:59–76.
4 **Gibbs EL**, Lenox WG, Gibbs FA. Bilateral internal jugular blood. Comparison of AV differences, oxygen-dextrose ratios and respiratory quotients. *Am J Psychiatry* 1945;**102**:184–90.
5 **Miller DL**, Doppman JL. Petrosal sinus sampling: technique and rationale. *Radiology* 1991;**178**:37–47.
6 **Matta BF**, Lam AM. The rate of blood withdrawal affects the accuracy of jugular venous bulb. Oxygen saturation measurements. *Anesthesiology* 1997;**86**:806–8.
7 **Aaaslid R**, Lindegaard KF, Sorteberg W, *et al*. Cerebral autoregulation dynamics in humans. *Stroke* 1989;**20**:45–52.
8 **Altman DG**. Principles of statistical analysis. In: *Practical statistics for medical research*. Boca Raton, FL: Chapman & Hall, 1991:152–78.
9 **Bland JM**, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986;**ii**:307–10.
10 **Lassen NA**, Lane MH. Validity of internal jugular blood for study of cerebral blood flow and metabolism. *J Appl Physiol* 1961;**16**:313–20.
11 **Maas AHJ**, Hamelink ML, De Leeuw RJM. An evaluation of the spectrophotometric determination of HbO_2, HbCO and Hb in blood with the CO-Oximeter IL 182. *Clin Chim Acta* 1970;**29**:303–9.
12 **Deibler GE**, Holmes MS, Campbell PL, *et al*. Use of triton X-100 as a haemolytic agent in the spectrophotometric measurement of blood O_2 saturation. *J Appl Physiol* 1959;**14**:133–6.
13 **Van Slyke DD**, Neill JM. The determination of gases in blood and other solutions by vacuum extraction and manometric measurement. I. *J Biol Chem* 1924;**61**:523–73.
14 **Cruz J**, Miner ME, Allen SJ, *et al*. Continuous monitoring of cerebral oxygenation in acute brain injury: injection of mannitol during hyperventilation. *J Neurosurg* 1990;**73**:725–30.
15 **Gopinath SP**, Robertson CS, Contant CF, *et al*. Jugular venous desaturation and outcome after head injury. *J Neurol Neurosurg Psychiatry* 1994;**57**:717–23.
16 **Signorini DF**, Andrews PJ, Jones PA, *et al*. Adding insult to injury: the prognostic value of early secondary insults for survival after traumatic brain injury. *J Neurol Neurosurg Psychiatry* 1999;**66**:26–31.