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Cerebral Venous Oxygen Content During Carotid Thrombintimectomy *

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THE MAINTENANCE of cerebral oxygenation during carotid thrombintimectomy and its enhancement as a result of operation are items of primary concern during the surgical treatment of occlusive cerebrovascular disease. Of the various methods proposed for the evaluation of cerebral oxygenation, it has seemed to us that the cerebral A–V O_2 difference was the most practical and the most direct. In the clinical situation wherein the arterial saturation may be maintained at 100 per cent, the per cent saturation of venous blood with oxygen becomes a sufficient index of cerebral oxygenation even though it can not differentiate between variations in oxygen consumption, alterations in blood flow, or combinations of these factors.

Present technics permit the continuous recording of the directly determined per cent oxygen saturation of venous blood from the lateral sinus with an average lag time of 1½ minutes. The observations reported here are derived from an experience with 50 patients undergoing carotid thrombintimectomy with local or general anaesthesia during the past two years.

Methods

1. Cannulation of the Lateral Sinus. After exposure of the internal jugular vein and division of the common facial vein, the tip of a 5-foot length of P.E. No. 50 tubing is introduced through a No. 18 needle into the jugular vein. It is then threaded upward as far as it will go to gain a position in the lateral sinus. The tubing is anchored to the wall of the vein and to a skin margin, flushed with heparin-saline and the distal end is passed off the table. We routinely give 50–75 mg. of heparin to the patient at this time.

2. The Oxygen Monitor. The Clark [1, 2, 5] apparatus consists of a proportioning pump, cuvettes, magnetic stirrers, a polarizing

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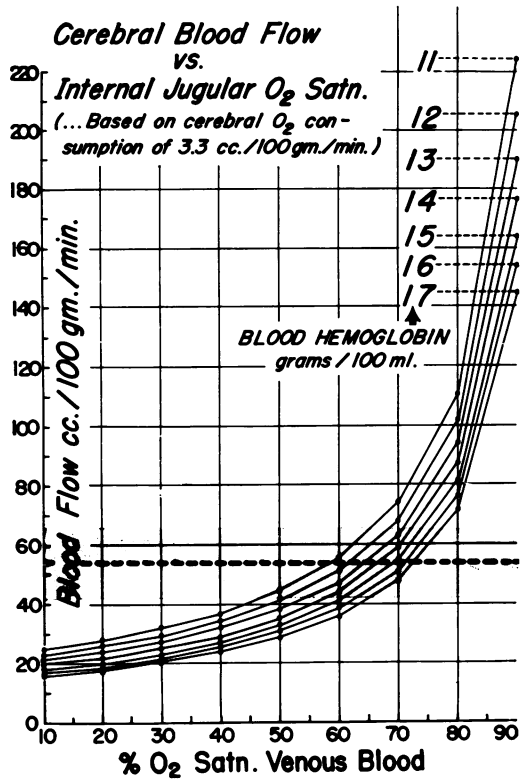

FIGURE 1.

circuit, and recording potentiometers. Blood is pumped from the lateral sinus at a rate of 0.3 ml. per minute, mixed with 20 parts of hemolyzing ferricyanide solution (de-aerated 3% ferricyanide containing saponin) in an anaerobic micro-cuvette equipped with a Clark oxygen electrode and a magnetic stirrer, and the oxygen tension is continuously recorded. The details of the design, theory and use of electrodes have been recorded elsewhere.[7, 8] This apparatus has been used continuously for as long as four days in cardiovascular patients. No complications have been recognized as the result of jugular cannulation.

3. *Standardization.* Since the O_2 content of blood varies with the hemoglobin content, a 6 ml. sample of blood is drawn and fully oxygenated in a rotating flask. Blood from this reservoir is repeatedly sampled to identify 100 per cent saturation during the period of monitoring. Arterial pO_2, pCO_2 and pH may be intermittently or continuously monitored to assure the validity of the observed per cent saturation of venous blood as an index of the A–V O_2 difference.

4. *Calculations.* Cerebral blood flow rates in an unanesthetized patient may be calculated from venous oxygen values on the basis of the original work done by Kety and Schmidt.[3] These calculations have been reduced to graphic form in Figure 1. It is apparent that venous oxygen saturation of 60 per cent usually reflects an optimal flow rate.

Observations

1. *Effect of Variation of Arterial pO_2 and pCO_2 in "Normal Patients."* In patients undergoing incidental neck operations under local anaesthesia, the effects of hyperventilation, oxygen breathing, and inhalation of 5% CO_2—95% O_2 were compared. A brief (45 seconds) period of hyperventilation produces a significant decrease in cerebral flow requiring four minutes for recovery as shown in Figure 2. The oxygen content of venous blood rises appreciably on oxygen breathing (Fig. 3) but is increased even more by adding 5% CO_2 to the oxygen (Fig. 4).

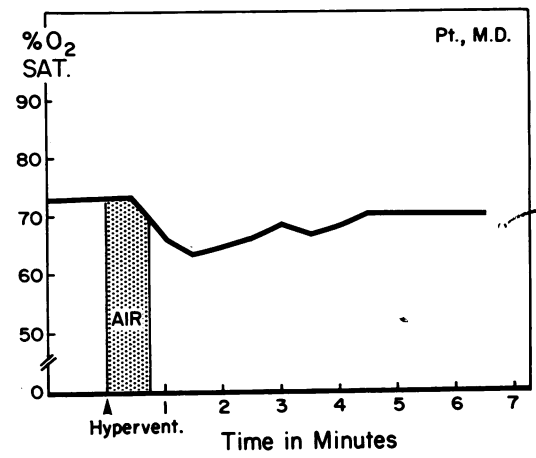

FIG. 2. The per cent O_2 saturation in jugular venous blood after hyperventilation for ¾ minute.

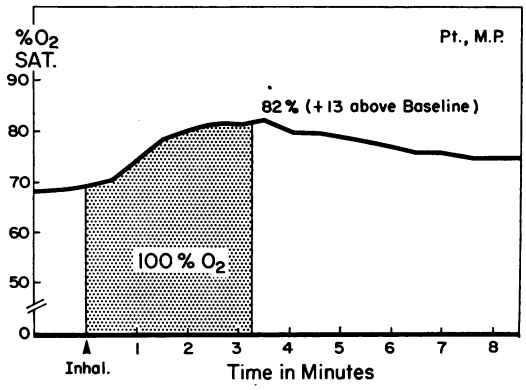

FIG. 3. The per cent O_2 saturation in jugular venous blood after breathing 100% O_2 for 3¼ minutes.

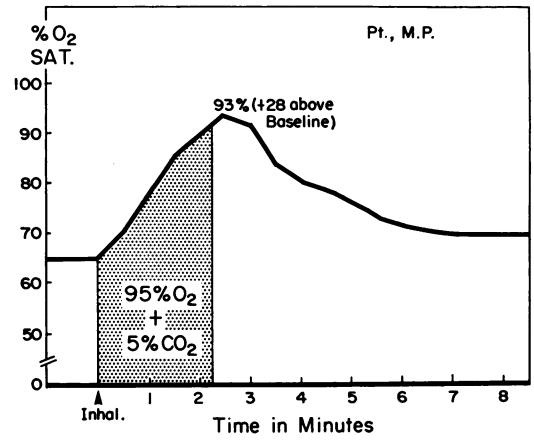

FIG. 4. The per cent O_2 saturation in jugular venous blood after breathing 95% O_2 plus 5% CO_2 for 2¼ minutes.

2. Effect of Blood Pressure. The effect of blood pressure on venous oxygen values was observed in a hypertensive *normal* patient with local anaesthesia during spontaneous recovery from a syncopal episode (Fig. 5). There is an almost linear relationship between per cent oxygen saturation and systolic pressure. In patients with occlusive disease, induced hypertension with vasopressors often increases venous oxygen content even after carotid occlusion.

3. Effect of Arteriography. The intra-arterial injection of renograffin was found to depress venous oxygen significantly at the first injection, but subsequent injections were less effective (Fig. 6). The recovery time from this depressant effect is about ten minutes and we have utilized this interval between injections in clinical practice.

4. Effect of Carotid Occlusion. Following release of an occluded carotid, the venous oxygen content rises suggesting a transient reflex cerebral hyperemia. This effect may be temporarily mitigated by a transient fall in blood pressure.

5. Effect of Carotid Thrombintimectomy. The evaluation of enhanced flow is often possible in patients whose operation is done under local anaesthesia. An especially

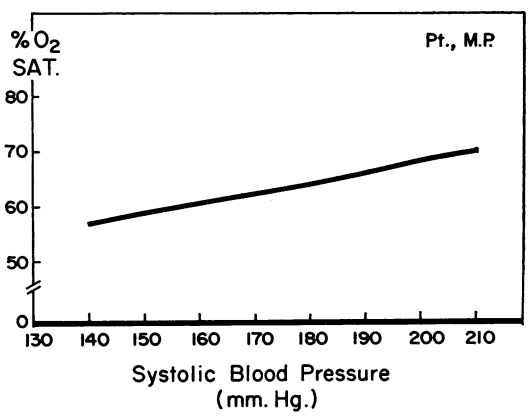

FIG. 5. The per cent O_2 saturation in jugular venous blood during recovery from syncopal episode.

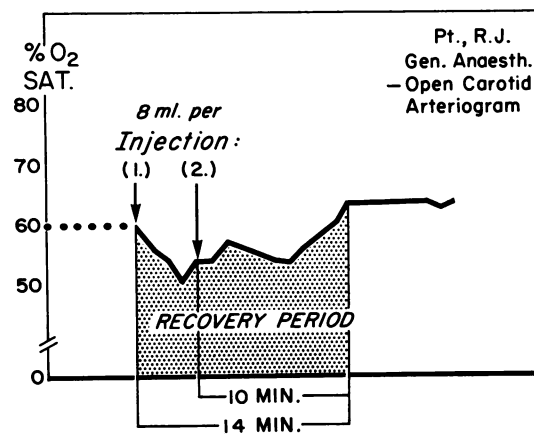

FIG. 6. The effect of intra-arterial injection of 60% renograffin upon jugular O_2 saturation.

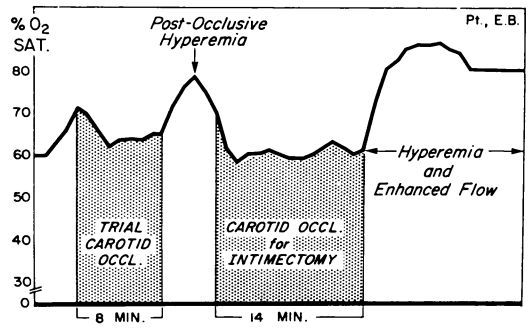

FIG. 7. Jugular venous O_2 saturation during operative reconstruction of stenotic internal carotid artery.

gratifying result is presented in Figure 7, wherein an increase of 20 per cent in venous oxygen saturation indicated a two-fold increase in flow. The efficacy of an internal shunt is well illustrated in Figure 8. This figure also confirms the fact that less than 50 per cent venous oxygen saturation identifies critical cerebral hypoxia with associated transient neurologic deficit in this patient.

6. Effect of General Anesthesia. The well known depressant effect of general anesthesia upon the cerebral metabolic rate of oxygen consumption results in unsuually high venous oxygen values provided arterial pCO_2 values of 35–40 mm. Hg and systolic blood pressures of 170–180 are maintained. When there is abundant collateral circulation, values up to 98 per cent saturation have been observed during carotid occlusion (Fig. 9). On the other hand, it is often possible to identify significant cerebral hypoxia under general anesthesia. Figure 10 presents such an instance in a patient with the *subclavian steal* syndrome. Obstruction of the subclavian distal to the vertebral increased venous oxygen saturation from 65 per cent to 90 per cent, and this higher value was maintained after surgical repair. It is important to identify the fact that the jugular venous oxygen values here reflected a deficiency of *basilar* circulation.

Discussion

The observations as regards the effect of systemic blood pressure, arterial pCO_2 and arterial pO_2 confirm previous observations [4] and the utility of the method herein reported. It is of considerable interest that patients with local anesthesia develop neurologic deficits when the venous oxygen saturation drops below 50 per cent for any period of time. This observation qualifies the method as a useful technic for monitoring patients during operation. Meyer *et al.* [6] have correlated electroencephalographic and jugular pO_2 changes in experiments in primates. It was noted that small regional cortical changes might be detected by the E.E.G. and missed in the venous blood, but the converse was true in vertebral artery occlusion. The correlation was quite satisfactory when cerebral metabolic changes were diffuse. The venous method has obvious virtues as regards quantitation and interpretation, but its major point of superiority over the EEG is in the detection of deficient vertebral-basilar flow.

Patients undergoing carotid thrombintimectomy, under either local or general anesthesia, exhibit considerable variation in blood pressure and often have significant variations in arterial pCO_2. When such variations occur, the venous oxygen record identifies variations in cerebral blood flow of considerable magnitude. Unless the sys-

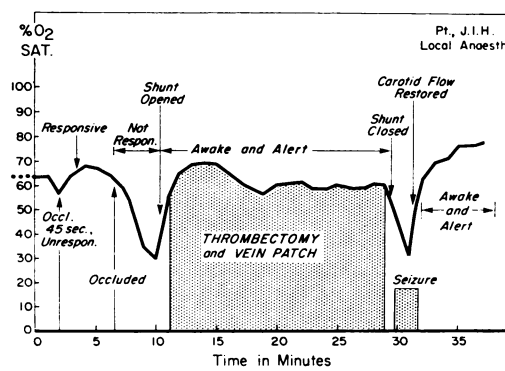

FIG. 8. Bilateral carotid stenosis and vertebral occlusion.

temic pressure and arterial pCO_2 be comparable, flowmeter studies before and after thrombintimectomy are meaningless. The venous oxygen method identifies flow changes nicely in unanesthetized patients, but does not permit such deductions with equal certainty when the cerebral rate of oxygen consumption is depressed by general anesthesia.

There is now general acceptance of the concept that the best protection against anemic infarction during carotid occlusion is provided by general anesthesia with oxygen-rich gas mixtures, mild hypercarbia and induced systemic hypertension. Wells, Keats and Cooley [9] have reported their experience with this technic utilizing an E.E.G. monitor, and state that shunts, by-passes and hypothermia are no longer necessary. In a small series, we have had one patient with bilateral carotid stenosis and absence of posterior communicating arteries who did require a shunt during occlusion of the first side. Although the program of general anesthesia with hypercarbia and hypertension certainly minimizes the need for internal shunting during occlusion, it seems to us

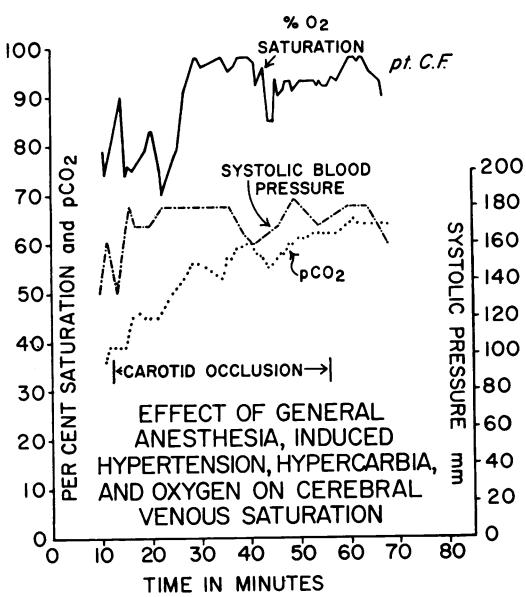

FIGURE 9.

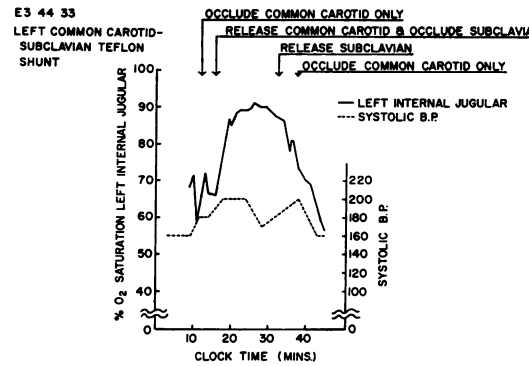

FIG. 10. "Vertebral Steal" effect on venous oxygen—ipsilateral internal jugular.

essential that some type of monitoring be employed. Although the E.E.G. is probably adequate during carotid occlusion, it is not reliable during vertebral occlusion. Since we are increasingly concerned with repair of vertebral lesions, the venous oxygen method seems preferable.

It would be unwise to attempt too many conclusions from our experience with this method to date. We believe that cerebral A–V O_2 difference is a reliable index of cerebral oxygenation and intend to pursue studies in this area toward the end that the vagaries of cerebral flow and the effect of drugs upon cerebral metabolism may be more completely understood. Our current monitor measures venous pCO_2 and pH in addition to pO_2. The technic is adaptable to the management of patients with progressive strokes.

Conclusions

A technic for continuously monitoring the cerebral A–V O_2 difference as an index of cerebral oxygenation has been developed.

A 50 per cent oxygen saturation of cerebral venous blood has been defined as a minimally acceptable level for assured cerebral oxygenation.

The venous oxygen monitor is superior to an E.E.G. monitor in that it identifies reduced flow in the basilar-vertebral system as well as in the carotid system.

Evidence is presented that cerebral oxygenation is enhanced by surgical treatment of atherosclerotic lesions of the extracranial vessels.

Acknowledgment

We are indebted to Edward R. Strong, Jr., research assistant, for technical supervision of the Clark apparatus.

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Discussion

Dr. Richard Shackelford (Baltimore): Dr. Lyons has just presented a beautiful piece of work, I think. I was unaware that it was on the program until this morning, and we have been doing work along somewhat similar lines, but from a different angle.

His procedure has a great advantage in that he is continuously monitoring cerebral blood flow. Dr. Stephen Hegedus and I, working in the Research Laboratory at the Perry Point VA Hospital, have been working on cerebral blood flow using Kety's nitrous oxide differential adsorption method, and have done experiments on over two hundred dogs and a sizable number of patients. Our findings are similar to those of Dr. Lyons, except, of course, ours are at specific moments, while his is a continuous monitoring. I think his procedure has certain advantages.

However, our method is quite accurate quantitatively. We have measured cerebral blood flow in milliliters/100 Gm. of brain weight/minute, which is a quantitative number that we can put down and compare with other readings.

It is a very accurate method, and is reproducible within less than a 3 per cent error after the team gets used to it.

However, we have been uncertain as to whether studying cerebral blood flow is actually the most important thing in cerebral circulation from the standpoint of neurologic deficits. Is the amount of blood that runs through the main vessels the important fact? Does that tell how much is distributed to the cells that are impaired by relative ischemia? The Nile River carries a great deal of water, and yet the land around it is arid unless irrigation ditches are established to distribute that water in the soil.

Our experiments so far have demonstrated the following, in an attempt to find what is important. Bilateral cervical sympathectomy does not affect the cerebral blood flow in normal animals. It does increase the spinal fluid pressure.

The same is true in dogs with both internal carotids ligated—which produces an 11 per cent reduction in cerebral blood flow—and it is true in patients with carotid obstruction.

Inhalation of 5 per cent carbon dioxide increases the cerebral blood flow from 30 to 35 per cent in both of the above groups during inhalation, but as soon as the inhalation is stopped it returns to its former rate.

We now have animals living in a carbon dioxide environment to see what permanent changes we can effect.