Effect of Valsalva Maneuver on Oxygen Saturation in Patients with Intracardiac Shunts

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Oxygen saturation of arterial and central venous blood was measured continuously during the Valsalva maneuver in patients with and without intracardiac defects. The majority of patients with atrial septal defects and patients with ventricular septal defects and high right ventricular pressure showed a sharp decrease in saturation of radial artery blood shortly after the maneuver. These changes were not recorded accurately by ear oximetry. The patients with shunts uniformly showed a rapid increase in saturation of pulmonary artery blood during the initial period of increased airway pressure. This effect was not observed in patients without shunts. These reactions may be of special diagnostic value under certain circumstances.

Although under "resting" conditions the pressure gradient between the 2 atria is predominantly from left to right, temporary reversals during short periods of the cardiac cycle have been demonstrated in patients\(^1,2\) and in experimental animals.\(^3-5\) Demonstration by dye-dilution curves of small right-to-left shunts of 5 per cent or less in the majority of patients with atrial septal defects who do not show evidence of gross arterial desaturation\(^6\) confirmed the theory that these brief periods of reversed gradient are capable of producing shunting of small volumes of venous blood to the left atrium. In 1950, Brecher and Opdyke\(^7\) demonstrated that various respiratory maneuvers can accentuate this reversal in dogs with experimental atrial septal defects, and in the same year Burchell\(^7\) reported a pronounced decrease in oxygen saturation of arterial blood during breath holding in a patient with an atrial septal defect associated with a veno-arterial shunt. Variations in oxygen saturation associated with the respiratory cycle have been described in the venous blood of normal persons.\(^8\) These variations are accentuated in patients with left-to-right shunts and may be observed also in the arterial blood of some of these patients.\(^9\)

In 1954 Lee and co-workers\(^10\) showed that, during the first few heartbeats following the release of increased intrathoracic pressure at the end of the Valsalva maneuver, the right atrial pressure increased above the pulmonary artery wedge pressure and therefore presumably above the left atrial pressure. In 1957 Lee and Gimlette\(^11\) reported a diagnostic test for interatrial communications utilizing this effect of the Valsalva maneuver. By means of an ear oximeter, they observed a drop in arterial oxygen saturation which occurred shortly after the period of increased intrathoracic pressure. This was ascribed to the occurrence of a temporary right-to-left shunt.

Since the ear oximeter may not provide a reliable index of arterial oxygen saturation under conditions such as the Valsalva maneuver which cause large changes in the blood content of the ear, the present study was undertaken to confirm the report of Lee and Gimlette\(^11\) by means of measurements made directly on arterial blood and to include observations on venous blood.
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MATERIAL AND METHODS

The effect of the Valsalva maneuver on the circulation was investigated in 11 patients with atrial septal defects, 6 with ventricular septal defects, and 10 without shunts, by means of continuous and simultaneous recordings of the oxygen saturation of blood withdrawn both from a systemic artery and from various vessels and chambers of the heart accessible by right heart catheterization. The persons without intracardiac shunts, who served as a control group, included 6 patients referred to the laboratory because of possible cardiovascular lesions and 4 patients with mitral regurgitation. The 6 patients were found to have normal cardiovascular systems on the basis of clinical and catheterization data. Of the 4 patients, 1 patient had severe mitral regurgitation, 1 was studied after a successful mitral valvuloplasty, and the remaining 2 were thought to have mild mitral regurgitation with slight disability. In the latter 2 patients a slight elevation of the pulmonary artery wedge pressure with the V wave representing the maximal pressure was the only abnormality observed in the data obtained by catheterization of the right side of the heart. The 11 patients with atrial septal defects included 4 with the usual type of defect; 4 with so-called superior vena cava syndrome, which consisted of a high atrial septal defect and anomalous pulmonary venous connection of the right upper and middle lobes to the superior vena cava or right atrium near the juncture of this chamber and the superior vena cava; and 3 patients with atrial septal defect who had unusual dye-dilution curves and may have had partial anomalous pulmonary venous connection in addition to the atrial septal defect. One patient in this group had severe pulmonary hypertension. In the 5 patients who underwent operation, the catheterization findings were confirmed.

The 6 patients with ventricular septal defects included 2 with uncomplicated ventricular septal defect with only slight elevation of the right ventricular systolic pressure and 4 patients with marked right ventricular systolic hypertension, which was due to infundibular pulmonary stenosis in 3 patients and to severe pulmonary hypertension in 1. The ratios of systolic pressures in the right ventricle to those in the radial artery in these 4 patients averaged 0.90 (range 0.81 to 1.02), indicating that the right and left ventricular systolic pressures were essentially equal.

Cardiac catheterization was performed in all patients by methods previously described. These included pressure, oxygen saturation, and dye-dilution studies. The patients rested in the supine position. They had a light meal prior to the procedure and received premedication of 90 mg. (1 1/2 gr.) of secnade sodium and 30 mg. (1/2 gr.) of codeine.

The Valsalva maneuver was performed as follows: the patients were asked to take a deep breath and to blow from their lungs (glottis open) into a mouthpiece connected by tubing to a strain-gage and to an aneroid manometer (Tyco), so as to maintain a pressure of 40 mm. Hg as indicated on the dial of the aneroid manometer which was placed in their field of vision. This increased airway pressure was maintained for about 15 seconds. The precise measurements of the photographic records of the airway pressure revealed that the average mean pressure during the pressure plateau was 37 mm. Hg (range 21 to 48) and, excluding 2 patients with extreme values, the range was 31 to 44 mm. Hg. The mean duration of the pressure rise was 17 seconds (range 12.3 to 20.6 seconds).

For about 30 seconds prior to the beginning of the maneuver, during the period of increased airway pressure, and for 20 to 40 seconds following the maneuver, arterial oxygen saturation was recorded continuously by an ear oximeter and also directly in arterial blood being withdrawn continuously through a cuvette oximeter attached to an indwelling no. 20-gage needle in the radial artery. In addition, in most cases the oxygen saturation was recorded continuously in blood being withdrawn through a second cuvette oximeter via a catheter the tip of which was placed at selected sites in a vessel or chamber on the right side of the heart. The average rate of flow through the cuvette oximeters was 20.0 ml. per minute with a range of 10.2 to 37.7 ml. per minute. The variables, which included airway pressure, respiration, electrocardiogram, heart rate, and the oxygen saturation of blood in the ear, radial artery, and in most cases from the right side of the heart, were recorded by a photokymographic assembly on paper 18 inches wide moving at a speed of 0.5 cm. per second. Each milliliter of blood flow through the cuvette oximeters was signaled on the photographic record. The time required for the blood to traverse the “dead space” volume between the sampling site in the vascular system and the detecting element of the cuvette oximeters was calculated from the flow rate and the volume of the needle-oximeter or catheter-oximeter assembly. With the use of this correction factor the curves of blood oxygen saturation recorded from the right side of the heart and the radial artery were aligned, with respect to time, with the other recorded variables, so that simultaneously occurring values could be compared throughout the period.
of observation. The average volume of the sampling systems from the needle tip in the radial artery up to and including the detecting chamber in the cuvette oximeter was 0.19 ml. (range 0.18 to 0.30 ml.), whereas the volume for the cardiac catheter-cuvette systems averaged 1.53 ml. (range 1.28 to 2.48 ml.). The time corrections for these arterial and venous sampling systems averaged 0.6 second (range 0.3 to 1.2 second) and 4.9 seconds (range 2.7 to 8.6 seconds) respectively.

The values for blood oxygen saturation were measured in all records at selected points with reference to the period of increased intrathoracic pressure. These included maximal, minimal, and average saturation values during the control period prior to the beginning of the maneuver; the values at 5, 10, and 14 seconds following the onset of increased airway pressure; and the values at 10, 20, and 30 seconds following the end of the period. In addition, when present, the following saturation values and the time at which they occurred were measured: (1) the maximal saturation reached during the maneuver, (2) the onset of the decrease (dip) after the release of the increased intrathoracic pressure, and (3) the minimal value during this decrease. Because of cyclic respiratory changes in oxygen saturation during the control period, decreases or increases in saturation of less than 1 per cent were not considered to be significant in relation to variations induced specifically by the Valsalva maneuver.

The appearance times of dye-dilution curves recorded at the radial artery following the injection of the indicator into the superior or inferior vena cava in patients with right-to-left shunts were measured and correlated with the onset of the decrease in the radial artery saturation following the Valsalva maneuver. In patients in whom a left-to-right shunt but no right-to-left shunt was evident in the dye-dilution curves, the appearance time of a "potential" right-to-left shunt was estimated by subtracting the pulmonary recirculation time measured from the curve from the appearance time at the radial artery following the injection into a vena cava.

The sites in the central circulation from which the blood oxygen saturation was recorded included the pulmonary artery, pulmonary vein, right atrium, and superior vena cava. The number of maneuvers with records of saturation from these various sites in the 3 groups of patients is shown in Table 1. Recordings of the oxygen saturation of radial artery blood during the Valsalva maneuver were obtained in all patients and those of pulmonary artery blood in all but 3.

### RESULTS

Typical changes in the oxygen saturation in arterial blood recorded by the ear and cuvette oximeters in 2 patients with atrial septal defects are shown in figure 1. In one of these patients the ear oximeter curve followed closely the actual pattern of changes in arterial saturation as recorded by the cuvette oximeter directly from radial artery blood while in the other, the ear oximeter record did not accurately reflect the changes evident in the radial artery saturation. The latter was the case in about 50 per cent of the experiments and therefore the ear oximeter records were not analyzed further.

The average values and changes in oxygen saturation of radial and pulmonary artery blood and their variability before, during, and after the Valsalva maneuver in patients without shunts and in those with atrial septal defects are shown in figure 2. Examples of original recordings from individual patients are shown in figure 3. An increase in systemic arterial saturation occurred uniformly during the period of increased intrathoracic pressure and was similar in magnitude in all 3 groups. It amounted to 1.1 per cent (range 0.5 to 4.5 per cent), 1.5 per cent (range 0.5 to 4.0 per cent), and 1.8 per cent (range 0.5 to 3.5 per cent) above the maximal control value for patients without shunts and with atrial and ventricular septal defects respectively. Usually within 10 seconds after the release of the increased pressure a sudden decrease was clearly evident in radial artery saturation in patients with atrial septal defects, whereas
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(♀, 17 years - Atrial Septal Defect) (♀, 57 years - Atrial Septal Defect)

**Fig. 1 Top.** Comparison of changes in oxygen saturation of systemic arterial blood recorded continuously during the Valsalva maneuver by an earpiece oximeter and a cuvette oximeter connected to a radial artery. The patient whose record is shown in the left panel may possibly have anomalous pulmonary venous connection in addition to the atrial septal defect. The patient whose record is shown in the right panel has a high atrial septal defect with anomalous connection of the right upper and middle lobes to the junction of the right atrium and superior vena cava. Note that the recordings from the earpiece and cuvette oximeter in the right panel correspond closely, but the earpiece record in the left panel fails to show the changes actually occurring in oxygen saturation of arterial blood as indicated by the cuvette oximeter.

**Fig. 2 Bottom.** Average and variability of the changes in oxygen saturation of blood withdrawn from the radial and pulmonary arteries in 10 subjects without intracardiac shunts and 10 patients with atrial septal defects. Black circles, average of the group calculated from changes in per cent saturation from the average control value for each patient. Numbers in brackets, number of patients, if the given point is not based on all the patients of the group. Vertical and horizontal bars, 1 standard deviation for the saturation and time values respectively. The points that precede the onset of the increase in airway pressure represent the maximal, minimal, and average control saturation for the group. Open circles and triangles, the average saturation values of radial and pulmonary artery blood of individual patients during the control period. Note the dip in the radial artery saturation shortly after the end of the increased airway pressure and the biphasic response in the pulmonary artery saturation in patients with atrial septal defects and the absence of these changes in patients without shunts.
the saturation in the cases without shunts generally exhibited a slow return toward control levels. An early decrease in saturation of 1 per cent or more following the Valsalva maneuver was found in 8 of 11 patients with atrial septal defects and in 3 of 10 patients without shunts. One of the 3 patients with atrial septal defects who did not show the usual response showed a larger decrease in saturation during a subsequent maneuver, and the lack of a typical response in another patient may have been related to a 16-second period of apnea that followed the Valsalva maneuver. In the 3 patients without shunts who showed a 1-per cent decrease in saturation after the maneuver, the decrease was more gradual than in patients with interatrial communications in whom the decrease was sharp, the contour of the change in saturation resembling somewhat that of an indicator-dilution curve. In 6 of 8 patients with atrial septal defects who exhibited this characteristic response, the sudden decrease in saturation ranged from 2.5 to 4.5 per cent, whereas it amounted to 1 per cent in the remaining 2 patients and in the 3 patients without shunts.
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When the magnitude of the decrease in per cent saturation was divided by the time in seconds from the onset to the minimal value during the "dip," the values for the 3 patients without shunts fell between 0.10 and 0.20 and those for the 8 patients with atrial septal defects fell between 0.25 and 1.25; these values confirmed the visual impression of a more rapid decrease in saturation in patients with atrial septal defects. The type of atrial septal defect and the presence or absence of anomalous pulmonary venous connection did not seem to influence the response.

The responses of 2 patients with ventricular septal defects are also shown in figure 3. The patient with right ventricular systolic hypertension showed a sharp dip in the radial artery saturation after the maneuver. This type of response was found in all 4 patients with right ventricular systolic hypertension, whether due to pulmonary stenosis or pulmonary hypertension, and the magnitude of the decrease ranged from 1.5 to 9 per cent. The 2 patients without ventricular hypertension showed only a gradual slow decrease in the radial artery saturation similar to the response seen in the patients without shunts (fig. 3).

The relation of the time from the end of the Valsalva maneuver to the onset of the dip in radial artery saturation, to the appearance time of an actual or "potential" right-to-left shunt of a dilution curve recorded at the radial artery in patients with intracardiac defects is illustrated in figure 4. There is a general correlation of the 2 variables suggesting that this dip in arterial oxygen saturation may be caused by the transient occurrence of, or increase in, an existing right-to-left shunt.

Striking differences in the changes in oxygen saturation of pulmonary artery blood associated with the Valsalva maneuver were observed between the patients with and those without intracardiac shunts. The average values and their variability obtained in the groups without shunts and with atrial septal defects are shown in figure 2. The characteristic biphasic response in patients with defects and its absence in patients without shunts is well illustrated by original records from individual patients shown in figure 3. All the patients with atrial and ventricular septal defects exhibited a definite increase in pulmonary artery saturation above the maximal control value during the initial period of increased intrathoracic pressure, which averaged 4.0 per cent (range 1.0 to 7.0 per cent), and 4.2 per cent (range 2.0 to 9.5 per cent) respectively. In 8 of 10 patients with atrial septal defects the increase in saturation of pulmonary artery blood was preceded by a definite small dip below the minimal control value occurring just before, or approximately at the time of, the onset of increased airway pressure. This phenomenon was seen in only 1 patient with ventricular septal defect. The peak saturation during the maneuver was followed by a decline that reached saturations below minimal control values in 4 of 15 pa-

![Fig. 4. Correlation of the appearance time of the right-to-left shunt deflection in dye-dilution curves recorded following injection of indicator into the vena cava with the time from the cessation of the Valsalva maneuver to the onset of the dip in arterial oxygen saturation after the Valsalva maneuver. Note the general correlation of the 2 variables suggesting that the dip in arterial oxygen saturation in these patients is due to the transient occurrence or increase in a pre-existing right-to-left shunt that occurs concomitantly with the cessation of the Valsalva maneuver.](http://circ.ahajournals.org/content/579.4172)

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patients with intracardiac defects during the later stages of the period of increased intrathoracic pressure. The second part of the biphasic response was a sudden further decrease in saturation to levels below the minimal control values beginning at the time of the release of the increased airway pressure. This further decrease was observed in all patients with atrial septal defects and in 4 of 5 cases of ventricular septal defect and averaged respectively 6.4 per cent (range 3.5 to 16.0 per cent), and 3.2 per cent (range 1.0 to 6.0 per cent).

In contrast to the patients with intracardiac shunts, those without shunts showed only a decrease in saturation of pulmonary artery blood during the period of increased intrathoracic pressure. In 7 of 9 cases this decrease was interrupted by a temporary return toward the control value followed by a second more pronounced decrease to lower saturation levels (figs. 2 and 3). All patients in this group exhibited a wave of increase in saturation starting usually at the time of release of increased airway pressure before the saturation returned to a steady plateau. The maximal saturation of this wave was below, above, or equal to the control values in various patients.

The oxygen saturation of blood withdrawn from the superior vena cava was recorded during the Valsalva maneuver in 8 patients with atrial septal defects. A pronounced increase in the saturation ranging from 3.5 to 12.5 per cent occurred during the period of increased intrathoracic pressure in 5 of the 8 patients, whereas in the remaining 3 there was only a decrease in saturation very similar to the change that took place in the pulmonary artery of patients without shunts. Two patients who showed this increase and one who did not show it, had an anomalous pulmonary venous connection at the junction of the superior vena cava and the right atrium suggesting that this increase in saturation may have been due to backflow of arterialized blood into the superior vena cava from the right atrium.

A significant increase in the saturation of right atrial blood was not observed during the Valsalva maneuver in the 2 patients with ventricular septal defect in whom this was recorded.

The oxygen saturation of blood withdrawn from a pulmonary vein was recorded during the Valsalva maneuver in 5 patients with atrial septal defect. In 4 cases these veins were anomalously connected. In 2 cases, 1 of which is illustrated in figure 5, there was no sudden decrease in oxygen saturation of blood in the pulmonary vein shortly after the maneuver, although a definite dip in the radial artery saturation could be seen. One patient did not show this decrease at either sampling site, whereas in the remaining 2 the dip was present in the pulmonary vein and in the radial artery saturation.

The reproducibility of the saturation changes in response to the Valsalva maneuver can be gauged from figure 6, which shows the magnitude and the time of maximal saturations in the radial artery from successive maneuvers in the same patients. These data are based on patients with shunts, in several of whom the saturation in the radial artery was recorded 2 or 3 times simultaneously, with the saturation from different sampling sites reached by the cardiac catheter. Most of the points fall on or close to the line of identity, usually within 0.5 per cent saturation and 2.0 seconds. Occasionally, however, larger differences were observed.

**DISCUSSION**

Continuous recording of the oxygen saturation of blood being withdrawn simultaneously from a systemic artery and from various sites on the right side of the heart allows a better understanding of the circulatory changes that occur in patients with intracardiac shunts during the Valsalva maneuver than has been possible previously. It also permits a better evaluation of diagnostic tests that utilize this maneuver. 11

Although a sharp decrease in radial artery saturation was found shortly after the end of
(♀, 51 years - Atrial Septal Defect with Pulmonary Hypertension)

**FIG. 5** Top. Comparison of changes in the oxygen saturation of pulmonary vein and radial artery blood during the Valsalva maneuver. Note the definite dip in saturation of radial artery blood shortly after the release of increased airway pressure and its absence in pulmonary vein blood indicating that this decrease in saturation of arterial blood is due to a right-to-left shunt rather than to a change in oxygenation of the blood traversing the lungs.

**FIG. 6** Bottom. Reproducibility of the magnitude and time of maximal saturation of radial artery blood recorded during successive Valsalva maneuver in the same patients.
the Valsalva maneuver in the majority of patients with atrial septal defects as reported by Lee and Gimlette, ear oximetry, which they utilized, was found to be unreliable for this purpose. In about half of the experiments ear oximeter records did not reflect accurately the saturation changes evident in the radial artery blood and were impossible to interpret. This would be expected under the conditions of large and rapid changes in blood content of the ear that are produced by the Valsalva maneuver. Because compensation for the changes in quantity of transilluminated blood is not complete, artifacts are produced in the oxygen saturation values indicated by the ear oximeter under these circumstances.

Continuous recording of oxygen saturation of systemic arterial blood withdrawn through a cuvette oximeter during the Valsalva maneuver may provide a screening test for the presence of certain types of defects. However, even the demonstration of a typical response does not differentiate between patients with ventricular septal defects with high right ventricular pressure and patients with atrial septal defects. It is probable that patients with aortopulmonary communications and pulmonary hypertension also may exhibit this type of response. Furthermore, the test would not provide information as to whether the interatrial communication is of the os- tum primum or secundum type and whether or not it is associated with anomalous pulmonary venous connection. The latter type of information is often essential in planning the surgical attack on the underlying lesion.

A temporary right-to-left shunt shortly after the end of the Valsalva maneuver might be expected to occur on the basis of changes in intrathoracic pressure gradient as demonstrated in human beings and in experimental animals. The finding that in 2 patients the dip in the radial artery saturation occurred while there was no sharp decrease in the pulmonary vein saturation suggests that the dip in systemic arterial saturation is not due to an effect of the maneuver on the oxygenation of blood during its passage through the lungs. Reversal of flow in the vena cava and its tributaries probably accounts for the finding of the decrease in saturation in the anomalously connected pulmonary veins in the other 2 patients. Correlation between the appearance time of the deflection caused by the right-to-left shunt of a dilution curve recorded at the radial artery and the time of onset of the dip in the radial artery saturation following the Valsalva maneuver also supports the concept that the dip in the radial artery saturation is due to a temporary appearance of, or an increase in magnitude of, a right-to-left shunt.

Present experiments confirmed the finding of the increase in systemic arterial saturation during the period of increased intrathoracic pressure, which Lee and Gimlette ascribed to a decrease in the pre-existing right-to-left shunt. However, the fact that this increase was of similar magnitude in patients with and without intracardiac defects suggests that other factors must play a part in most cases. Possible causes of the increase in arterial oxygen saturation include (1) increase in alveolar PO2 resulting from the increase in intrathoracic pressure and augmented ventilation of some of the alveoli associated with the deep inspiration preceding the maneuver, and (2) decrease in carbon dioxide content of arterial blood due to this deep inspiration which would shift the hemoglobin saturation curve to the left.

Changes in oxygen saturation of the blood withdrawn from various sites on the right side of the heart are of interest. All patient with atrial septal defects exhibited similar changes in the pulmonary artery saturation during the Valsalva maneuver. The initial dip in saturation is probably due to an increased inflow of the blood from the systemic veins associated with the deep inspiration that preceded the increase in airway pressure, whereas the rise in saturation during the period of increased intrathoracic pressure must be related to the decrease in systemic venous return well known to occur during the Val- salva maneuver. The increase in saturation could result if the volume of blood shunted from left to right increased, re-
mained unchanged, or decreased but to a lesser degree than the decrease in systemic venous return. The decline in pulmonary artery saturation later during the period of increased intrathoracic pressure could result from an increase in systemic venous return, a decrease in the volume of the left-to-right shunt, or both.

The sudden further decrease in the pulmonary artery saturation, practically coincident with the release of increased pressure, must be due to the sudden onrush of systemic venous blood to the right atrium which increases the right atrial pressure, decreases the absolute volume of the left-to-right shunt, and creates a temporary right-to-left shunt as indicated by the changes in the radial artery saturation.

The sharp rise in the superior vena cava saturation that occurred in 5 of 8 patients with atrial septal defects, 3 of whom did not have anomalous pulmonary veins connected close to the superior vena cava, is probably due to the reversal of flow known to occur in the vena cavae during the Valsalva maneuver.19, 20

The patients without shunts showed only a decrease in the pulmonary artery saturation during the period of increased intrathoracic pressure. This may be due to the decrease in the return of the blood from the vena cavae and thus an increase in the proportion of the low-saturation blood from the coronary sinus contributing to the total pulmonary flow, since a decrease in the coronary return would not be expected initially due to the increase in the intrathoracic pressure. The temporary return of pulmonary artery saturation toward, but not above, the control values during the early part of the period of increased intrathoracic pressure might be related to phasic changes in the contribution of the superior and inferior caval blood of different saturations to the total venous return.

In accord with the findings of Lee and Gimlette,11 the 2 patients with ventricular septal defects without pronounced elevation of the right ventricular pressure did not show the characteristic dip in radial artery saturation found in patients with atrial septal defects. However, the differential, diagnostic value of this finding is limited, since a definite decrease was found in all 4 patients with ventricular septal defects associated with right ventricular systolic hypertension. In such patients an increase of right ventricular pressure above that of the systemic arteries may occur during the Valsalva maneuver.26 All 6 patients with ventricular septal defects exhibited an increase in pulmonary artery saturation similar to that found in patients with atrial septal defects; however, this increase in saturation was not observed in the right atrium.

The findings of the characteristic saturation changes on the right side of the heart during the Valsalva maneuver may be of some diagnostic help during right heart catheterization since, by repeating the maneuver with continuous sampling at various sites, it should be possible to demonstrate and localize the left-to-right shunt. As demonstrated in 2 cases of ventricular septal defect, the characteristic increase in oxygen saturation of right heart blood should be present at and downstream to the site of the shunt, but not upstream to it in the absence of valvular regurgitation. This method is not likely to prove more sensitive than other ways of detection of left-to-right shunts in the majority of cases. However, in patients in whom the left-to-right shunt may be too small to demonstrate by other methods under normal resting conditions, either due to the extremely small size of the defect or due to severe pulmonary hypertension resulting in a balanced right-to-left and left-to-right shunt, temporary "unbalancing" of the shunt in the left-to-right direction by increasing the airway pressure may prove to be of special diagnostic value.

Summary

Changes in blood oxygen saturation during the Valsalva maneuver were recorded continuously and simultaneously in blood being withdrawn from the radial artery and from various sites on the right side of the heart in patients with and without intracardiac defects.
A transient decrease in oxygen saturation of radial artery blood was clearly evident shortly after the termination of the increased airway pressure in 8 of 11 patients with atrial septal defects and in 4 patients with ventricular septal defects and right ventricular systolic hypertension. This response was not observed in 10 patients without shunts nor in 2 patients with ventricular septal defects without marked elevation of right ventricular pressure.

However, ear oximeter records did not reflect accurately the saturation changes evident in the radial artery blood under these circumstances and hence were of limited diagnostic value. This is probably because of incomplete compensation of this instrument for the large and rapid changes in the blood content of the ear caused by the Valsalva maneuver.

Correlation between the appearance time of the deflection due to the right-to-left shunt of dye-dilution curves and the time of onset of the decrease in arterial saturation following the Valsalva maneuver, and comparison of the changes in oxygen saturation of pulmonary vein and radial artery blood during the maneuver suggest that the decrease in the systemic arterial saturation is due to the temporary occurrence of, or a transient increase in the magnitude of, a right-to-left shunt.

All the patients with intracardiac defects showed characteristic biphasic changes in the oxygen saturation of pulmonary artery blood consisting of clearly evident rapid increase in saturation shortly after the onset of increased airway pressure followed by a decrease which was sharply accentuated shortly after the release of airway pressure. In contrast only a decrease in saturation was observed in patients without shunts.

These characteristic changes in oxygen saturation of blood from the pulmonary artery in patients with left-to-right shunts were absent when venous blood was sampled from chambers in the right heart upstream to the defect. These responses allow a better understanding of the changes in the circulation of patients with intracardiac defects during the Valsalva maneuver and may be of diagnostic value in detecting and localizing cardiac defects under certain circumstances.

**Acknowledgment**

These studies were made possible by the unstinting co-operation of many technical and professional colleagues to whom the authors are indebted.

**Summario in Interlingua**

Alteraciones del saturation oxygenic esseva registrate continue et simultaneamente durante le effectuation del manovra de Valsalva in specimens de sanguine prendite ab le arteria radial e ab varie sitos al latere dextere del corde de patientes con e sin defectos intracardiaci.

Un reduction transiente del saturation oxygenic del sanguine ab le arteria radial esseva claramente evidente un breve tempore post le termination del augmentate pression in le viaes aeree in 8 del 11 patientes con defectos atrio-septal e in 4 patientes con defectos ventriculo-septal e hypertension systolic dextero-ventricular. Iste responsa non esseva observate in 10 patientes sin shunting intracardiaci e non in 2 patientes con defectos ventriculo-septal non associate con marcate grados de elevation del tension dextero-ventricular.

Tamen, lecturas del oxymetro de aure non reflecte accuratamente le alterationes del saturation oxygenic que es evidente in le sanguine del arteria radial sub iste circumstantias. Per consequente illos es de paucu valor diagnostic. Le causa de iste facto es probablemente le incomplete compensation que iste instrumento provide pro le grande e rapide alterationes del contento de sanguine in le aure que es causate per le manovra de Valsalva.

Le correlation inter le tempore del apparition del deflexion effectuate in curvas de dilution de colorante per shunting dextero-sinistre e le tempore del declaracion del reduction in le saturation arterial que occurre post le manovra de Valsalva, insimul con un comparation del alterationes in le saturation oxygenic del sanguine in le venas pulmonar con illos del sanguine in le arteria radial durante le manovra, suggere que le reduction del saturation oxygenic in le sanguine del
arterias systemic es causate per un occurren-
tia temporari de un shunting dexteroinsinistre
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tion del pression in le vías aeree. Per contrasto
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sanguine habeva non ancora passate per le
defecto. Le responsas noteate permite un plus
precise comprehension del alterations in le
circulation de patientes con defectos intrac-
cardiae, occurrencente durante le manovra de
Valsalva, e es possibilemente de valor diag-
nostic in le detection e localisation de defectos
cardiae sub certe circumstancias.

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Cardiac output and related measurements
and pressure values in the right heart and
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Medical Eponyms

By Robert W. Buck, M.D.

Bell's Palsy and the respiratory nerve of Bell were described by Charles Bell (1774-1842) in a communication read July 12, 1821, by Sir Humphry Davy before the Royal Society of London, entitled “On the Nerves, giving an account of some experiments on their structure and functions, which lead to a new arrangement of the system.” This appears in the Philosophical Transactions of the Royal Society of London, second part for 1821, pp. 398-424.

After describing the “respiratory nerve of the face, being that which is called the portio dura of the seventh” and detailing his experiments, he says:

“We have proofs equal to experiments, that in the human face the actions of the muscles which produce smiling and laughing, are a consequence of the influence of this respiratory nerve. . . .

“Cases of this partial paralysis must be familiar to every medical observer. It is very frequent for young people to have what is vulgarly called the blight; by which is meant, a slight palsy of the muscles on one side of the face, and which the physician knows is not formidable. Inflammations of glands seated behind the angle of the jaw will sometimes produce this. All such affections of the respiratory nerve will now be more easily detected; the patient has a command over the muscles of the face, he can close the lips, and the features are duly balanced; but the slightest smile is immediately attended with distortion, and in laughing and crying the paralysis becomes quite distinct. The knowledge of the sources of expression teaches us to be very minute observers.”
Effect of Valsalva Maneuver on Oxygen Saturation in Patients with Intracardiac Shunts

STEFAN A. CARTER, NEWTON C. BIRKHEAD and EARL H. WOOD

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