Frontiers in Health Informatics
ISSN-Online: 2676-7104

www.healthinformaticsjournal.com

2024; Vol 13: Issue 3

Open Access

Scientific Breakthroughs From Siggaard–Andersen Nomogram To Latest Developed Graphical Tool Representation For Arterial Blood Gas (ABG) Interpretation

[1]Dr.T.Rajini Samuel

1Professor , Department of Biochemistry Shri Sathya Sai Medical College and Research institute, SBV Chennai Campus Sri Balaji Vidyapeeth Deemed to be University

Corresponding Author: **Dr.T.Rajini Samuel**

Cite this paper as: Dr.T.Rajini Samuel (2024) Scientific Breakthroughs From Siggaard– Andersen Nomogram To Latest Developed Graphical Tool Representation For Arterial Blood Gas (ABG) Interpretation. *Frontiers in Health Informatics, 13* (3), 11213-11229

ABSTRACT

Arterial Blood Gas (ABG) interpretation plays an immense role in critical care medicine. Various approaches are available to interpret ABG data. The most commonly used approaches are the physiological approach using the bicarbonate-carbon dioxide buffer system and the base excess approach developed by Astrup and Siggard Anderson. Physico-chemical approach is used to explain the causative mechanism of metabolic acid base disturbances. The graphical representation may serve as an useful tool for easier and quicker interpretation of ABG data. But only few graph methods are proposed and devised for depicting the various respiratory and metabolic acid–base disturbances. They are used for teaching purposes only and these are not practically convenient to use in clinical settings. Graphical methods for ABG interpretation like Siggaard- Andersen chart (S-A chart), Davenport or Bicarbonate-pH diagram and Grogono diagram are not used frequently in the clinical practice due to certain practical difficulties and providing inaccurate diagnosis in few of the ABG data. A newer graphical method using 4 quadrant method was constructed and proposed by the current author. The purpose of this review is to discuss in detail the construction, interpretation, advantages, clinical utility and the limitations of the various proposed graphical tools.

KEY WORDS: Graphical Tool, Arterial Blood Gas, Siggaard-Andersen chart, Davenport diagram, Grogono diagram, 4 quadrant Graphical Tool

INTRODUCTION:

Blood Gas Analyzer remains an indispensable medical equipment in emergency department and critical care medicine. **Arterial Blood Gas (ABG)** interpretation plays an immense role in emergency health conditions. Various approaches are available to interpret ABG data.[1] The commonly used approaches are the **physiological approach** using bicarbonate and the **base excess** approach developed by Astrup and Siggard Anderson.

The most commonly used physiological approach is based on the bicarbonate-carbon dioxide buffer system(based on pCO_2/ carbonic acid/bicarbonate equilibrium).[2,3]

The contribution to the advancement of clinical acid-base chemistry by **Siggaard-Anderson** is significant. He implemented a method based on the **Van Slyke equation** which emphasizes the use of base excess (BE) or

deficit. In 1948, **Singer and Hastings** proposed the term **'buffer base'** to define the sum of HCO_3^- and the non-volatile weak acid buffers. A change in buffer base directly reflects the changes in the metabolic component. The methods for calculating the **change in buffer base** were later refined by many investigators to yield the base excess (BE) methodology.[4,5]

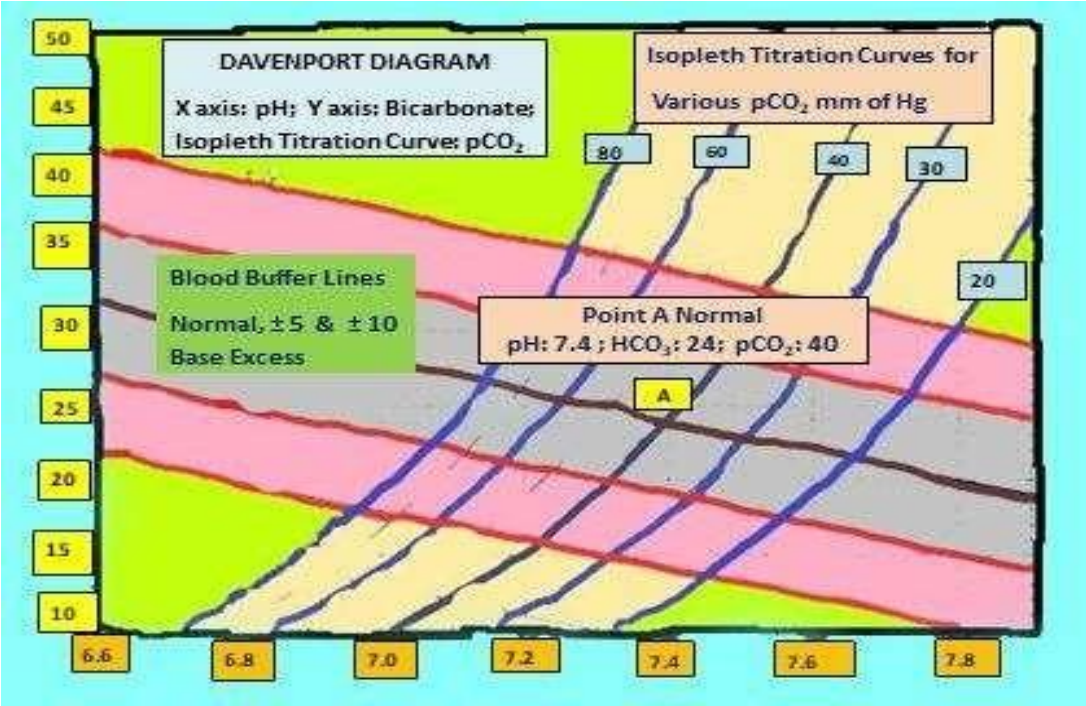
The **base excess calculation** was modified to **'standardize'** the effect of haemoglobin. A blood sample **diluted threefold** with its own plasma may serve as a **model of the extracellular fluid**.[4,5] **Standard base excess (SBE)** or extracellular base excess was developed to represent the in-vivo base excess which is the base excess at haemoglobin concentration of **5g/dl (or 3.1 mmol/L).**[6,7] *Thus the **Base excess** is defined as the amount of strong acid that must be added to each litre of **fully oxygenated blood** to return the **pH** to **7.40** at a temperature of 37°C and a **pCO_2** of **40** mmHg.[8,9,10]*

The graphical representation may serve as a useful tool for easier and quicker interpretation of ABG data. But only few graph methods are proposed and devised for depicting the various respiratory and metabolic acid–base disturbances. They are used for teaching purposes only and these are not practically convenient to use in clinical settings.[11,12] Graphical methods for ABG interpretation like **Siggaard-Andersen chart (S-A chart), Davenport or Bicarbonate-pH diagram** and **Grogono diagram** are not used frequently in the clinical practice due to certain practical difficulties and providing inaccurate diagnosis in few of the ABG data. A newer graphical method using **4 quadrant method** was constructed and proposed by the **current author (Rajini Samuel)**.[11-15] The knowledge of the scientific breakthroughs from Siggaard–Andersen nomogram to latest developed graphical tool representation for Arterial Blood Gas (ABG) interpretation may help us to understand the contribution of various researchers in the field of acid base clinical chemistry. The purpose of this review is to discuss in detail the construction, interpretation, advantages, clinical utility and the limitations of the various proposed graphical tools.

DAVENPORT DIAGRAM:

The plot of both the Henderson-Hasselbach Equation and the Van Slyke equation has been called ""Davenport or Bicarbonate-pH diagram". In the davenport diagram, **x-axis** represents the **pH** values**, y-axis** represents the plasma **bicarbonate** values and the **curved lines** (**isopleths**) denote the **pCO_2** values derived using the Henderson Hasselbach Equation(depicted in **figure 1**). These titration curves are called isopleths, because they are generated at a fixed partial pressure of carbon dioxide (shown in the **figure 1**). Each isopleth pCO_2 curved line has a **fixed pCO_2 value** for a given variable pH and calculated bicarbonate concentration.[16,17]

FIGURE 1: DAVENPORT DIAGRAM



The **blood-buffer line** is the relationship between HCO_3^- and pH as carbonic acid is added to whole blood. This **negative slope line** denotes the **buffering capacity** of blood which is achieved by varying pCO_2 through variation in alveolar ventilation.

CONSTRUCTION OF THE DAVENPORT DIAGRAM:

The **normal blood buffer line** goes through **point A (pH = 7.4, [HCO_3^-] = 24, pCO_2 = 40 mm Hg)** on the Davenport diagram (depicted in **figure 1**). If HCO_3^- is added or eliminated while maintaining the **pCO_2 fixed at 40 mmHg**, it will move **along** the **40 mmHg isobar away from point A**.

The blood sample from a healthy patient is collected and then placed in a chamber in which the partial pressure of carbon dioxide (pCO_2) is held at 40 mmHg. The pH and bicarbonate concentration are measured once equilibrium is reached,and then they are plotted on a diagrammatic chart. Then the pH of the blood sample is modified, first by adding a strong acid and then by adding a strong base but pCO_2 is kept constant in the chamber. A **titration curve** for a **given pCO_2** value is obtained by the changes in the pH and bicarbonate concentration for a given pCO_2 value (**40 mm Hg**). Similarly with a new, identical blood sample from the same patient the experiment is performed with the chamber reset to a pCO_2 **of 60 mmHg.** After equilibration, a new point is reached, indicating a new pH and a new bicarbonate concentration (shown in **figure 1**). The **bicarbonate** concentration at the newer, **higher pCO_2** will be **higher** than the **value** at **pCO_2** of **40** and the pH will be lower. [16,17] **A titration curve** for a **given** pCO_2 **value** is obtained by the changes in the pH and bicarbonate concentration for a given pCO_2 value (**60 mm Hg**). A series of isobars can be constructed depending on the pCO_2 values.[16]

The central **point A** (**figure 1**) is the normal value of pCO_2 (40mm Hg), pH (7.40) and bicarbonate (24 mmol/L). If this experiment is repeated at **various partial pressures of carbon dioxide**, with a new identical blood sample from the same patient **a series of points** will be obtained. A **line is drawn** through these points connecting the central point with other points representing the **buffer line**(clearly depicted in **figure 1**) which shows the possible values of pH and bicarbonate achieved by changing pCO_2 through changes in minute ventilation.

A series of **parallel blood buffer lines** can be constructed by changing the **Base excess values**. The normal buffer line passes through the base excess value of zero. The other parallel blood buffer lines drawn above and below the normal buffer lines depend on the **positive** and **negative** values of the base excess respectively (clearly shown in **figure 1**).

As pCO_2 is increased, the magnitude of the resulting change in pH is dependent on the buffering power of the non-bicarbonate buffers present in the solution. As pCO_2 increases, it reacts with water molecules to form carbonic acid which dissociates into hydrogen and bicarbonate ions. The albumin, hemoglobin, and phosphate buffer system help in buffering of hydrogen ions. So, the concentration of bicarbonate increases as pCO_2 also increases. The generation of a bicarbonate molecule is accompanied with release of a proton, so the pH is decreased. If **strong non-bicarbonate buffers** are present, then they will quickly absorb the vast majority of protons released by the generation of bicarbonate, and pH will change very little for a given rise in bicarbonate concentration. This results in a **buffer line** with a **very steep slope**. On the other hand, if **only weak non-bicarbonate buffers** are present (or **no non-bicarbonate buffer**), then a much larger change in pH will be observed for a given change in bicarbonate concentration, and the **buffer line** will have a **slope closer to zero**.[16]

The slope of the line will **never** actually reach **zero** (**horizontal**) under equilibrium conditions, even in the complete absence of non-bicarbonate buffers because the production of protons resulting from an increase in pCO_2 is concomitant with the production of bicarbonate ions. Thus, a decrease in pH resulting from an increase in pCO_2 always occurs with some minimal increase in bicarbonate concentration. Similarly, an increase in pH resulting from a decrease in pCO_2 must occur with some minimal decrease in bicarbonate

concentration.[16]

INTERPRETATION OF THE DAVENPORT DIAGRAM:

A **series** of **isobars** depending on the **pCO_2** values and a **series** of **parallel blood buffer lines** depending upon the **Base Excess** (BE) values are constructed in the davenport diagram. The **respiratory status** is assessed by the location on the **pCO_2 isobar** and the **metabolic status** is evaluated by the location on the **blood buffer line**. A **negative BE** (base deficit) indicates metabolic acidosis and a **positive BE** (base excess) denotes the presence of metabolic alkalosis. The concentration of bicarbonate corrected to pH of 7.4 is called the **Van Slyke Standard Bicarbonate (VSSB)** or **predicted bicarbonate value** (term used in some research articles). The difference between observed or actual bicarbonate (calculated from Henderson equation) and the predicted bicarbonate value (or Van Slyke Standard Bicarbonate) denotes the delta Vanslyke Standard Bicarbonate(**ΔVSSB)** which is numerically equal to the **base excess value**.[16,18,19]

An acid-base disturbance is characterized by two parameters namely the delta Vanslyke Standard Bicarbonate(**ΔVSSB)** and delta beta (**Δβ). ΔVSSB** denotes the **change in the position** of the Van Slyke curve and **delta beta** indicates the **change in the slope** of the Van Slyke curve. Delta VSSB measures the magnitude of a metabolic acidosis or alkalosis and is numerically equal to base excess. Delta beta indicating the buffer strength measures the change in the plasma's ability to resist perturbations of pH.

The observed $[HCO_3^-]$ can be calculated from the pH and pCO_2 values. The predicted $[HCO_3^-]$ is the value predicted from the normal blood buffer line and the observed pH which can be calculated using the given below equation.[16]

Predicted $[HCO_3^-]$ = 24 + β*(7.4-pH)

The buffer value β depends on the concentration of haemoglobin, albumin and phosphate values. Multiple formulas exist to calculate the same. Base Excess is defined as the difference between the observed and predicted $[HCO_3^-]$.

BE = observed $[HCO_3^-]$ - predicted $[HCO_3^-]$

= $[HCO_3^-]$ - {24 + β*(7.4-pH)}

= $[HCO_3^-]$ - 24 - β* (7.4-pH) or

BE = $[HCO_3^-]$ - 24 + β* (pH-7.4)

{(7.4 - pH) is changed to (pH - 7.4), so the sign is changed}

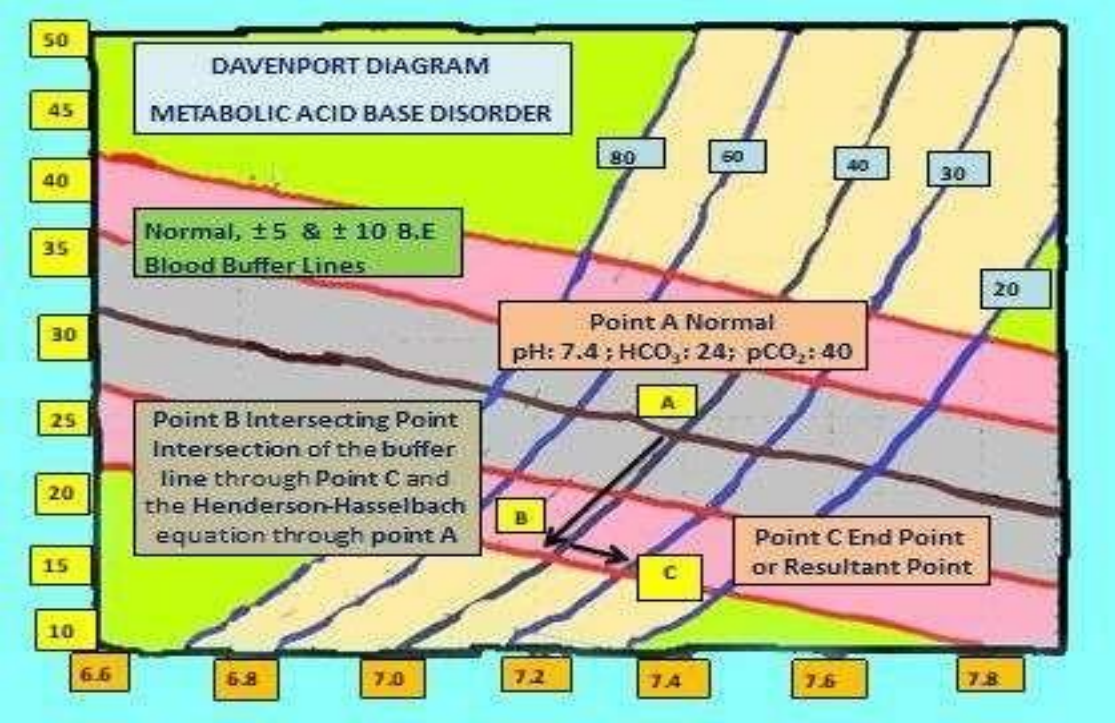
The above equation can be compared with the commonly used formulae to calculate the base excess values.

With a **pure metabolic disturbance** the point remains on the **normal pCO_2** (40 mm of Hg) isobar, but can develop **either a negative BE or a positive BE** by **moving** away from the normal point **along this isobar** (seen in **figure 1**). With a **pure respiratory disturbance**, the point remains on **the normal blood-buffer line,** but can **move along it** from **one isobar to another**. Movement along this line represents respiratory acidosis (decreasing pH) or respiratory alkalosis (increasing pH) which is clearly seen in **figure 1**. However, acid-base disturbances are rarely pure. A primary metabolic disturbance is compensated by respiratory mechanisms and vice versa. Also, it is not uncommon for two primary disturbances (mixed disorder) to coexist in the same patient at the same time. The components defining the acid base status can be graphically represented using the **relevant isobar**, as determined by the **pCO_2**, and the **relevant blood buffer line**, as determined by the value for **BE**. [16,17]

If a respiratory disturbance is superimposed on a non-respiratory (metabolic) one, movement from **one isobar(**pCO_2**) to another** along a blood buffer line that is **displaced from the normal line** by a distance equal

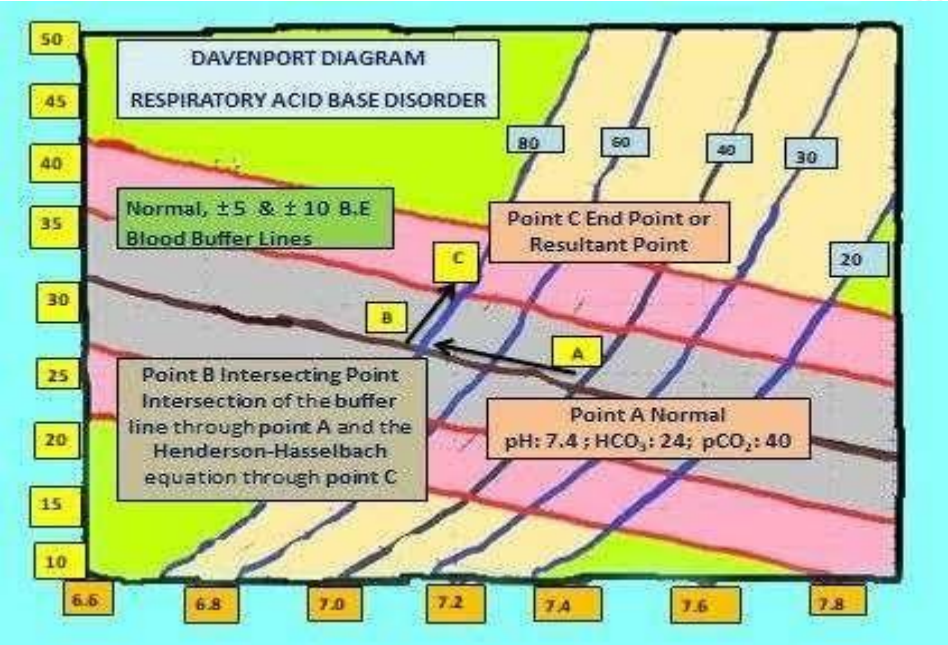
to the value for **BE**.The Henderson-Hasselbalch Curve of pCO_2 40 mm of Hg(**normal pCO_2 isobar**) and the **normal buffer line** passes through the **point A** which is the **starting point** with **normal** pH, pCO_2 and bicarbonate level**.** The **point C** is the **final result** of the measured pH, pCO_2 and bicarbonate values. (shown in **figure 2**)The **intersection** of the buffer line (**Van Slyke Curve**) with the **isopleth pCO_2 curve** (Henderson Hasselbalch Equation curve) yield the possible combinations of pH and bicarbonate concentration measured in the blood achievable by changes in alveolar ventilation **denoted** by the **point B**.[16,17,20] In case of **metabolic** acid base disorder, Point B is the intersection of the **buffer line** through **Point C** and the **Henderson- Hasselbach** equation through **point A** (clearly depicted in the **figure 2**).

FIGURE 2: Davenport Diagram-Metabolic Acid Base Disorder



In case of **Respiratory** acid base disorder **Point B** is the **intersection** of the buffer line through **point A** and the **Henderson-Hasselbach** equation through **point C** which is clearly shown in the **figure 3**.The resultant point C is the result of the **initial displacement** from the **normal point** A to the point B (**intersecting point**) and the **final displacement** from this intersecting point B to the end point C. The **initial** displacement indicates the **primary acid base disorder** and the **final** displacement represent the **compensatory mechanism** either respiratory compensations in metabolic acid base disorders or metabolic compensations in respiratory acid base disorders.[17]

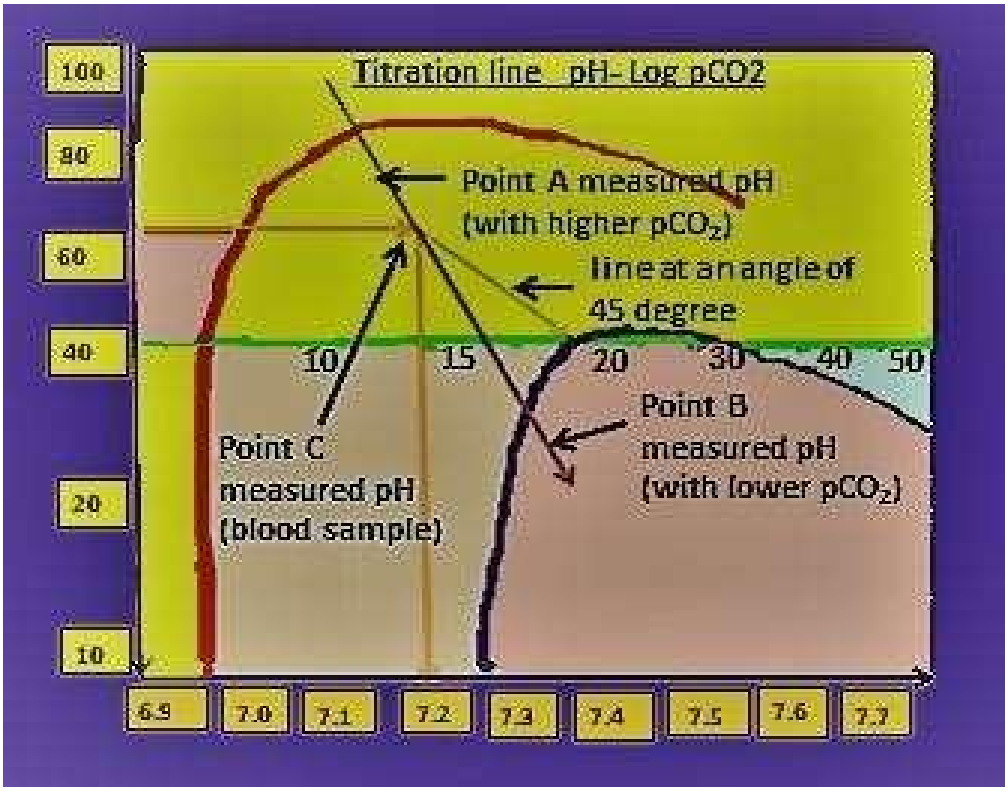
FIGURE 3: Davenport Diagram-Respiratory Acid Base Disorder



THE SIGGAARD–ANDERSEN CURVE NOMOGRAM:

This nomogram has **pCO_2** plotted on a **log scale** on the **vertical axis** and **pH** on the **horizontal axis**. Siggaard-Andersen used the procedure of equilibrium titration curves to determine the values of Buffer base and base excess. The two curves namely the **buffer base curve** and the **base excess curve** are constructed in this coordinate system using the determination of its **pH- log pCO_2 line** (depicted in the **figure 4**).The **pH** of the blood sample is measured **without** any **equilibration** in vitro. Then the pH of the blood sample is measured **after equilibration** with gas mixtures containing known values of **higher** and **lower pCO_2** values.[21]

FIGURE 4: pH-logpCO_2 line



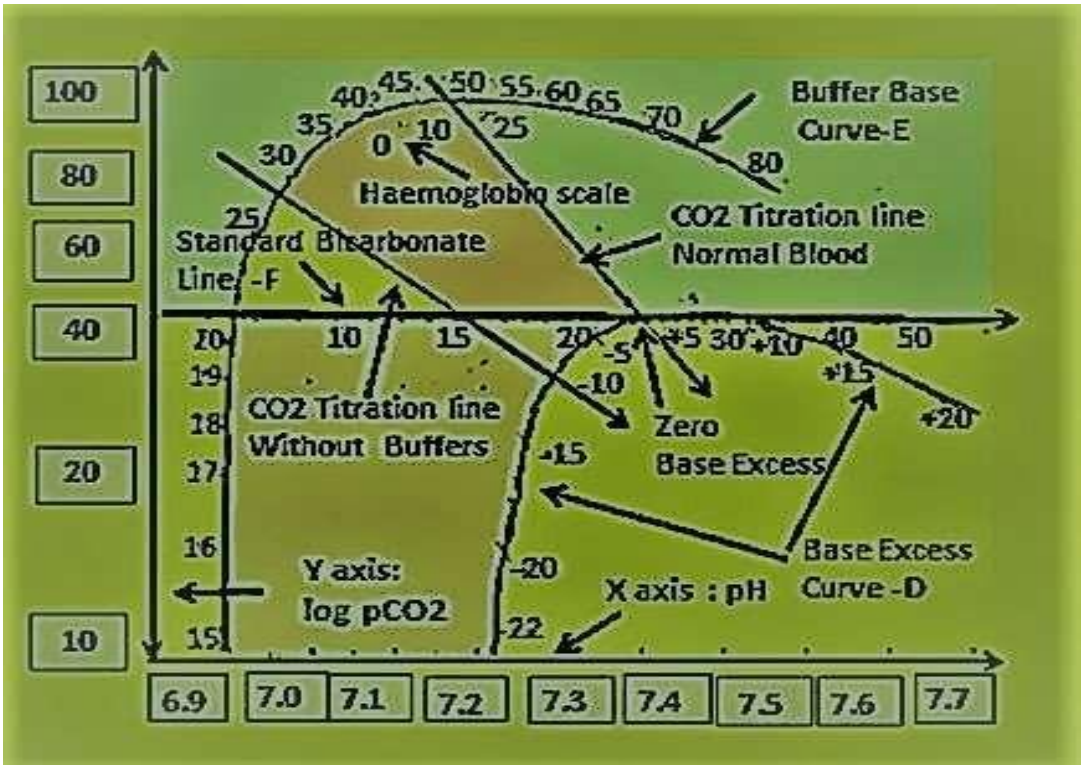
A – denote the measured pH after equilibration with higher pCO_2

B- denote the measured pH after equilibration with lower pCO_2

C- denote the measured pH of the blood sample (without in vitro equilibration)

A line is drawn connecting the points A and B. From the measured pH of the blood sample(without in vitro equilibration), a perpendicular line is drawn that meet the line connecting the points A and B. The value of pCO_2 of the blood sample is read off the ordinate.[21] This is the **Astrup method** of estimation of **pCO_2**.The **titration curves** (lines in the semi-logarithmic scale) have **different slopes** after blood titration with carbon dioxide, depending on **haemoglobin** concentrations. If **buffers** were present, the **slope** of the line would be **steeper.** For normal blood containing 15 g of haemoglobin/dL, the CO_2 titration line passes through the 15g/dL mark on the haemoglobin scale (on the underside of the upper curved scale) and the point where the pCO_2 equal to 40 mm Hg and pH equal to 7.40 lines intersect.

FIGURE 5: CO_2 TITRATION LINE WITH BUFFERS AND WITHOUT BUFFERS



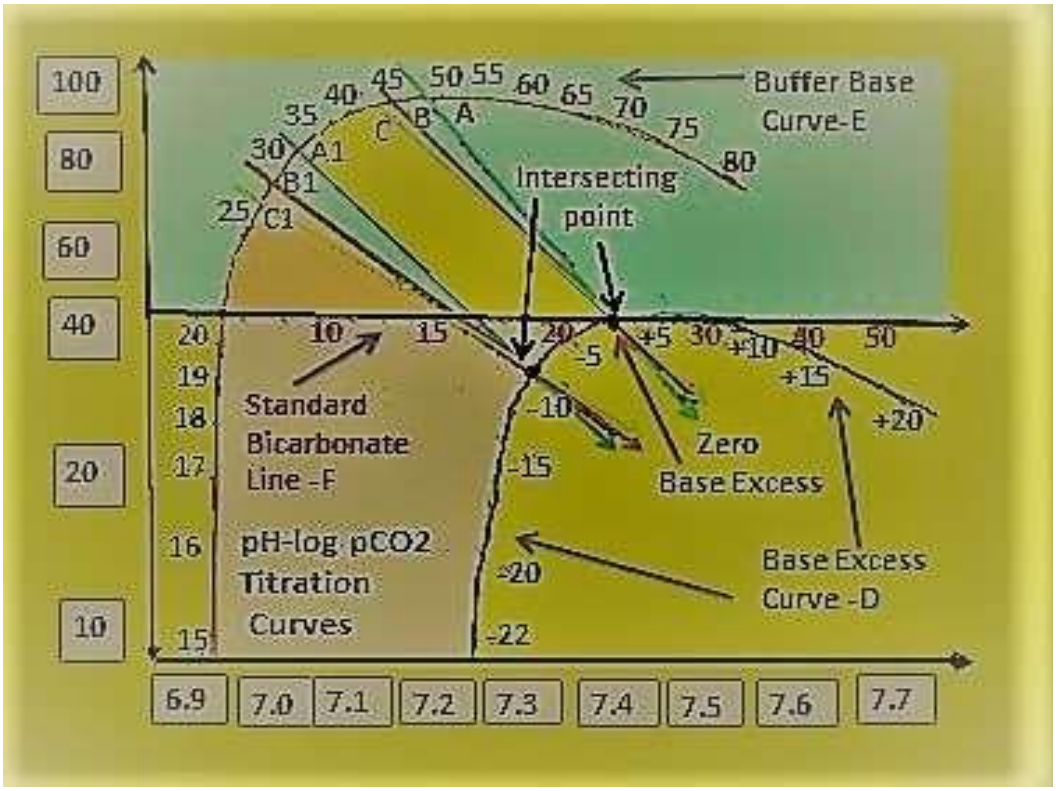
When the haemoglobin content of the blood is low, there is significant loss of buffering capacity, and the slope of the CO_2 titration line diminishes. However, blood of course contains buffers in addition to haemoglobin, so that even the line drawn from the zero point on the haemoglobin scale through the normal pCO_2–pH intercept is steeper than the curve for a solution containing no buffers. (depicted in the **figure 5**)

CONSTRUCTION OF THE BASE EXCESS AND BUFFER BASE CURVE:

Let **A** denote the **whole blood** , **B** denote the blood with **haemoglobin** concentration of **5g/100ml** and **C** denote the plasma(**blood without haemoglobin**).Then the equilibrium titration was done and the results were plotted in the axis in log pCO_2/pH coordinates.The **pCO_2-pH plot** can be constructed by **plotting several pH's** while **altering pCO_2**. Two points are usually enough because the graph is approximately a straight line

on log pCO_2-pH coordinates. The titration curves (being lines in the semi-logarithmic coordinates) of the **blood samples with various haematocrit** and the **same BE** always **crossed** in the **same points**.(shown in the **figure 6**) Similarly, the titration curves of the samples with various haematocrit concentrations (and various BE), but with the **same BB** crossed in the **same points**, too. The **intersection of the points** were the base for the **experimental determination** of the base excess and buffer base curve.[21]

FIGURE 6: Intersecting Points - Determination of Base Excess



The **base excess** values are **changed** by adding defined amounts of **strong acids** or **bases** to blood samples with various haematocrit concentrations.BB and BE change after addition of a strong acid (or strong base) or bicarbonates to the blood sample. Addition of one millimole of a strong acid to one litre of blood results in BE fall by one millimol; addition of one millimol of bicarbonates (or withdrawal of one millimol of hydrogen ions by a reaction with a strong base) results in BB and BE increased by one millimol. The experiment is repeated.

The titration curves of the samples with the **same BE** crossed in the **same points**. By connecting all the intersecting points(of the different base excess values) the base excess curve is constructed(depicted in the **figure 6**). Similarly, the **sample** with the **same BB crossed** in the **same points**. The buffer base curve is also constructed by changing the concentration of buffer base and connecting all the intersecting points.[21]

Increased partial pressure of carbon dioxide(pCO_2) causes it to freely diffuse into the erythrocyte where it reacts with **water molecules** to form **carbonic acid.** The **bicarbonate** and **hydrogen ions** are dissociated from carbonic acid catalysed by the enzyme **carbonic anhydrase** present inside the red blood cells(RBC). Haemoglobin is an important intracellular protein buffer present inside the red blood cells. Bicarbonates are transported into plasma by the concentration gradient (by exchange for chloride ions). Thus, the increase in CO_2 concentrations is associated with the **decrease** in the value of **Buffer Base** concentrations **inside erythrocytes**

(BBe) or **increase** in the value of **Buffer Base** in the **plasma** (BBp). The **equilibration titration** with carbon dioxide helps to achieve pCO_2 level at which the Buffer Base (BB) concentrations in erythrocytes and plasma equilibrate (**BBe = BBp**). With this level of pCO_2 at BBp=BBe, this value determines the point where the titration curves with the same total BB and various haematocrit (Hct) of the blood samples will cut each other in the same point on Siggaard-Andersen nomogram.[21]

As: $BB = BBp (1 - Hct) + BBe\ Hct$

$= BBp + Hct (BBe – BBp)$

The term Hct (BBe – BBp) is zero if BBe = BBp, so the **whole blood BB** does **not depend on haematocrit.** Thus, the Buffer Base(BB) curve on Siggaard-Andersen nomogram is a **geometric site** of the points where **plasma** and **erythrocytes** have the **same buffer base concentrations**, as at BBe is equal to BBp the whole blood BB does not depend on haematocrit (Hct).

Similarly this can be applied for the Base Excess(BE) curve, too.

As: $BE=BEp (1 – Hct) + BEe\ Hct$

$= BEp + Hct (BEe – BEp)$

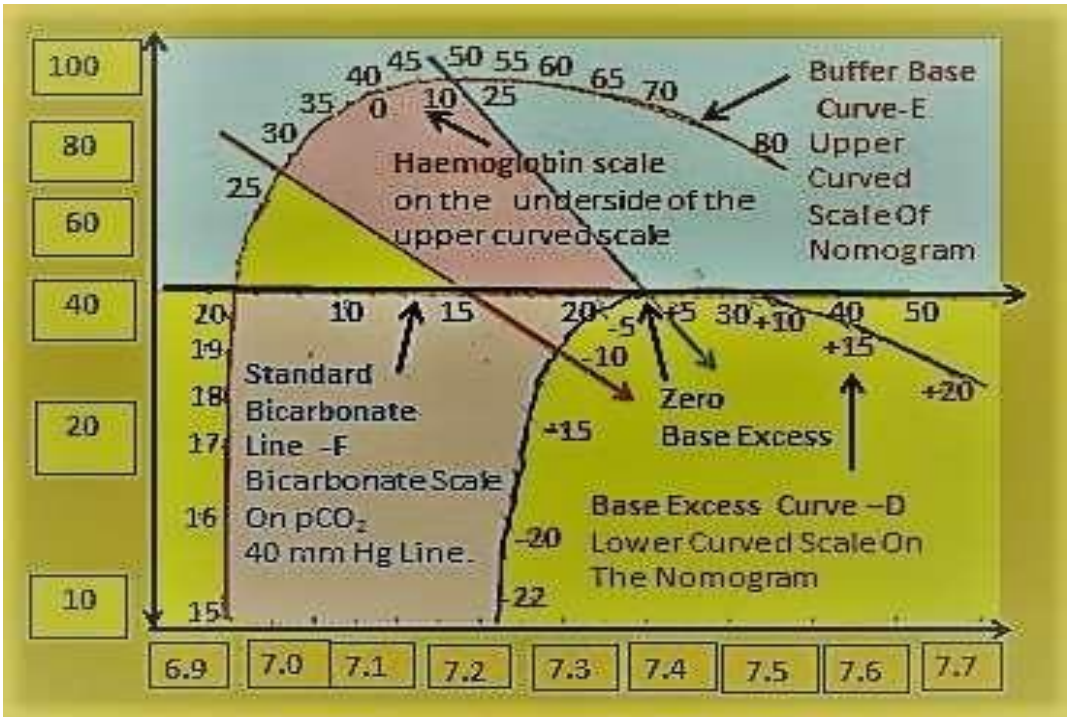
The term Hct (BEe – BEp) is zero at BEe=BEp then and the whole blood BE does not depend on haematocrit (Hct) or haemoglobin concentration. Thus, the **BE curve** on Siggaard-Andersen nomogram is a **geometric site** of the points with the **same BE** in the whole blood and plasma, as the whole blood BE does not depend on haematocrit at proper pCO_2 and pH, when BEe is equal to BEp.[21]

Thus, a nomogram with BE and BB curves with semi-logarithmic coordinates was obtained and these curves enable the determination of BE and BB in the samples to be tested. Siggaard-Andersen used the **mixture of O_2 - CO_2** for blood titration with fully oxygen-saturated blood , so the BE curves are those for fully oxygenated blood

$BE = BEox + 0.2\ cHB\ (1\text{-}sO_2)$

cHb denotes the haemoglobin concentration [g/100ml] and sO_2 denotes the haemoglobin oxygen saturation.[21]

FIGURE 7: DIFFERENT CURVES OF THE NOMOGRAM



Buffer Base Curve (E)-

upper curved scale of nomogram shows the mEq/L of buffer base in the sample

Base Excess Curve(D)-

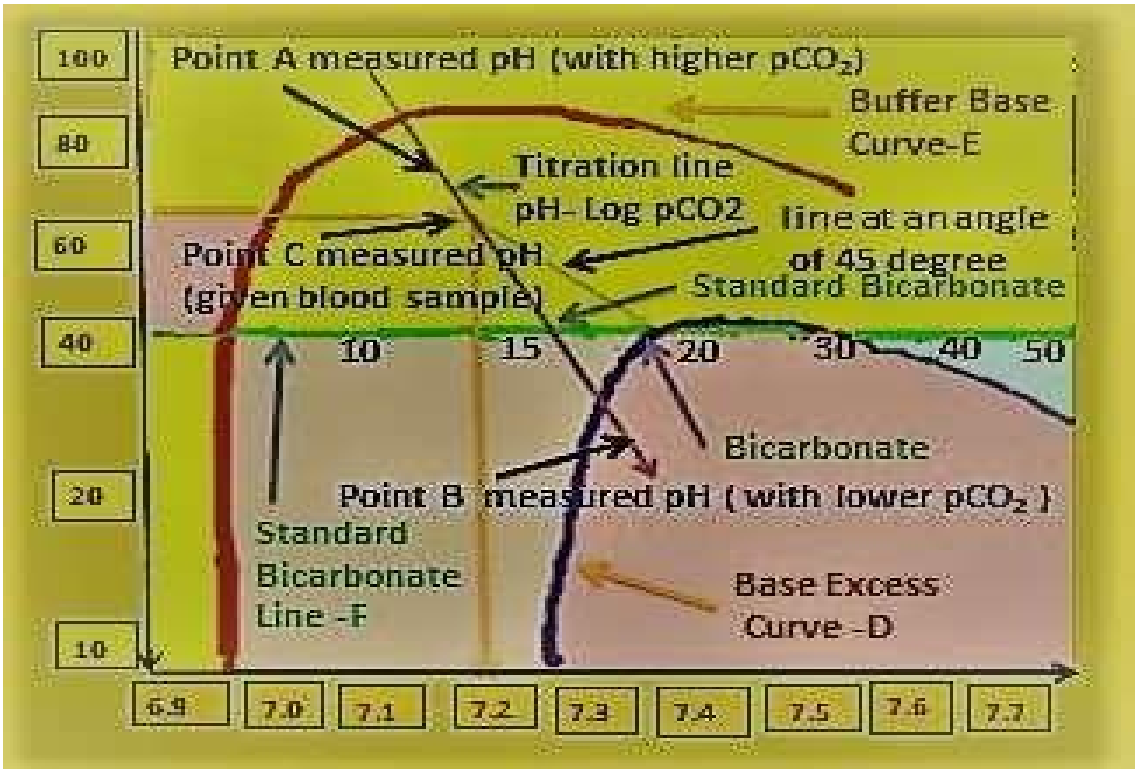
a lower curved scale on the nomogram indicates the base excess.

Standard Bicarbonate(F) Line Scale-

bicarbonate scale on the pCO_2 equal to 40 mm Hg line.

The **point of intersection** of the CO_2 calibration line of the arterial blood sample with the base excess curve(D), buffer base curve(E) and the standard bicarbonate(F) line scale **indicate the values** of base excess, buffer base and standard bicarbonate values respectively.(depicted in **figure 7 and 8**) The **actual bicarbonate** is determined in the graph by drawing a line at an **angle of 45 degree** from the point which denotes the **measured pH** of the blood sample (**without in-vitro equilibration**) to the **standard bicarbonate scale**. This intersecting point denotes the value of the actual bicarbonate.[21,22]

FIGURE 8: Siggaard-Andersen nomogram



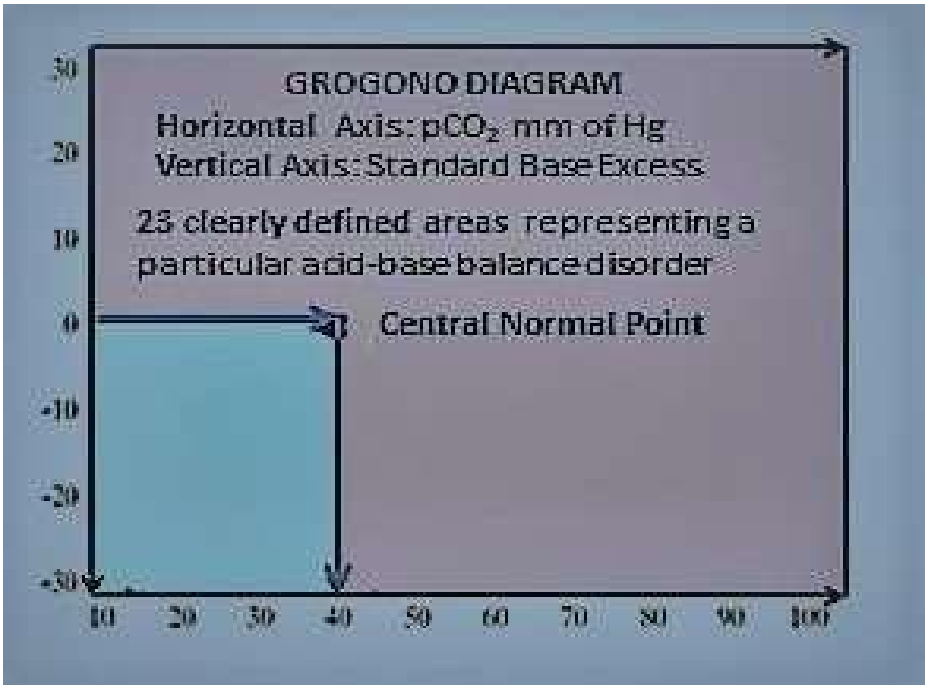
THE OXYGEN-STATUS-ALGORITHM (OSA) SOFTWARE:

This software was developed by the Radiometer-Copenhagen Company and installed in all blood gas analyzers manufactured by Radiometer-Copenhagen. This is based on the diagram and workdone by Siggaard-Andersen.[23]

THE GROGONO DIAGRAM: The initial concept for the construction of the Grogono diagram was based on the previously proposed Siggaard- Andersen and J. Severinghaus diagrams. It has two axes where the **pCO_2**

representing the **respiratory** component of the acid base disorder is plotted in the **horizontal** axis and the **standard base excess** denoting the **metabolic** component of the acid base disorder is plotted in the **vertical** axis.This diagrammatic representation tool consists of **23 clearly defined areas**, each one representing a particular acid-base disorder.(depicted in **figure 9**) This new diagrammatic approach proposed by A. Grogono is superior to the S- A chart, but it cannot be utilized for the interpretation of acid-base disorders , because it has not provided accurate interpretation in at least 25% of the ABG data. Despite its higher diagnostic agreement compared to the classic Siggaard-Andersen diagram, Grogono diagram is not superior to the Oxygen Status Algorithm software program.[23,24]

FIGURE 9: Grogono Diagram



A NOVEL FOUR QUADRANT GRAPHICAL METHOD:

The great technological advance in laboratory methods and instruments that has occurred in the last decades has not brought a respective development in the field of diagnosis of various acid base disturbances.[23] A new graphical tool was developed by the **current author** for ABG interpretation using **standard base excess** in the **x axis** and **ratios** derived using bicarbonate, standard bicarbonate and carbonic acid values in the **y axis**. The various acid-base disturbances are plotted and analysed in a four quadrant graph method.[25,26,27]

Modified Henderson equation is applied to calculate the concentration of bicarbonate. The concentration of bicarbonate varies with pCO_2 , so Standard bicarbonate is utilized which denotes the concentration of bicarbonate with a normal $PaCO_2$ (40 mmHg) and a normal pO_2 (over 100 mmHg) at a normal temperature (37°C).[28,29] Under normal ventilatory conditions both are similar but in abnormal ventilation (either hypo or hyperventilation) both the actual bicarbonate and the standard bicarbonate concentrations deviate from each other depending on the changes in magnitude and direction of variation in pCO_2.[13,14,30]

Ratio 1 = HCO_3/Std HCO_3

Ratio 2 = (HCO_3/H_2CO_3) - (Std HCO_3/H_2CO_3) **OR**

Ratio 2 = (HCO_3 - Std HCO_3) / H_2CO_3

Modified Ratio 2 = (Std HCO_3/1.2) - (HCO_3/H_2CO_3)

At **pCO_2 40 mmHg**, H_2CO_3 concentration is 1.2 mmol/L. So **ratio 2 is modified here** to correlate Std bicarbonate with H_2CO_3 concentration of 1.2 mmol/L.The **Compensation** bedside rules using the **Boston Method** (6 rules) involving bicarbonate can be applied for the assessment of compensation and to identify the presence of compensations or a mixed acid base disorder in clinical practice. [13,14,29,30] The graphical relationship between **pCO_2** and **ratio 1**, **ratio 2** and **modified ratio 2** were clearly depicted in the **figure 10**. A **4 quadrant graphical tool model constructed** using **standard base excess** and either (**pCO_2 - 40** mm of Hg) parameter or **modified ratio 2** is shown in **figure 11** which clearly shows the plot for various acid base disturbances.

FIGURE 10: NOVEL DERIVED RATIOS

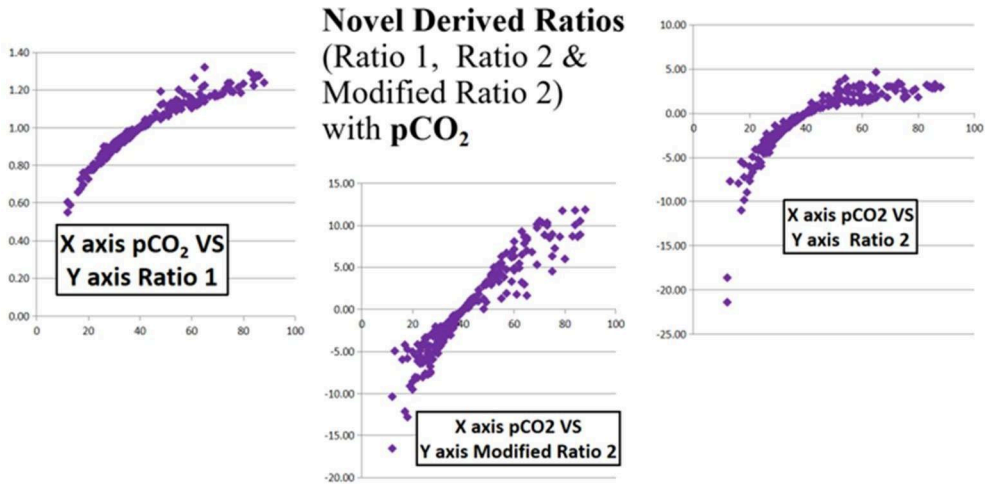
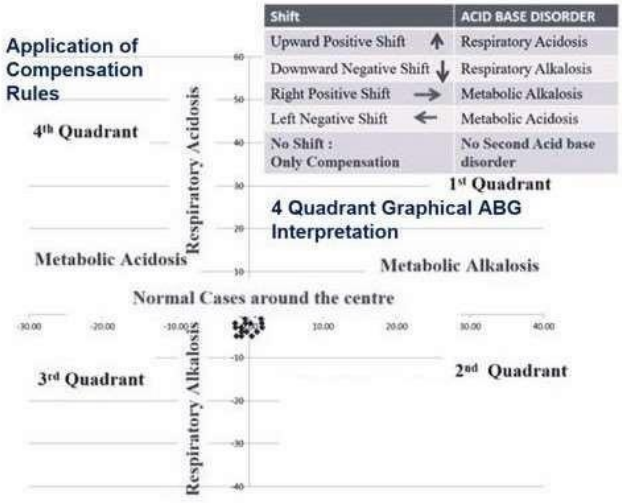


FIGURE 11: 4 Quadrant Graphical Tool Model for ABG Interpretation



If the value of **standard base excess** is **> +2 mmol/L** then it denotes **metabolic alkalosis** and if the value is **< - 2 mmol/L** then it denotes **metabolic acidosis**. The normal range for **pCO_2** is **35 to 45** mm Hg. **Higher pCO_2** values in **respiratory acidosis** and **lesser pCO_2** values in **respiratory alkalosis** are seen. [13,14,29,30]The **modified ratio 2** is **greater positive** for respiratory acidosis and **greater negative** for respiratory alkalosis. Using the **concept of shift** in the **plotted point position** after the application of compensation rules, the mixed acid base disorders can be identified in this graphical tool. [14,30] The scientific breakthroughs from Siggaard-Andersen nomogram to latest developed graphical tool representation for Arterial Blood Gas (ABG) Interpretation may help us to observe the transition, transformation and evolution in the field of diagnosis of acid base clinical chemistry through graphical representation methods.

CONCLUSION:

The detailed discussion of the construction, clinical interpretation and the limitations of the various proposed graphical tools may help us in teaching the students to gain in-depth knowledge in acid base chemistry.The latest proposed four quadrant graph method which involves the application of compensation rules looks very simple, easy and user friendly to overcome the arduous task of complex acid base disorders involving various compensations and mixed disorders.

REFERENCES:

1. Pramod Sood, Gunchan Paul, and Sandeep Puri Interpretation of arterial blood gas Indian J Crit Care Med. 2010; 14(2): 57–64.
2. Magder S, Emami A. Practical approach to physical-chemical acid-base management. Stewart at the bedside. Ann Am Thorac Soc. 2015;12:111–117
3. Adel Badr, Peter Nightingale Alternative approach to acid–base abnormalities in critically ill patients Continuing Education in Anaesthesia, Critical Care & Pain 2007;7(4):107-111
4. O. Siggaard-Andersen , N. Fogh-Andersen Base excess or buffer base (strong ion difference) as measure of a non-respiratory acid-base disturbance Acta Anaesthesiol Scand 1995; 107:123-128
5. Siggaard-Andersen O. Acid-base balance. In: Boston GEO, editor. Encyclopedia of respiratory medicine. Amsterdam: Academic Press; 2006. pages: 5-10
6. J. Kofstad: All about base excess – to BE or not to BE July 2003 Article downloaded from acutecaretesting.org Pages 1-5
7. V. M. Roemer The significance of base excess (BEB) and base excess in the extra cellular fluid compartment (BEEcf) July 2010 Article downloaded from acutecaretesting.org Pages 1-10
8. Rajini Samuel T Application of standard bicarbonate/carbonic acid ratio in arterial blood gas analysis International Journal of Clinical Biochemistry and Research, 2018;5(2):314 -320
9. Samuel R. A Graphical Tool for Arterial Blood Gas Interpretation using Standard Bicarbonate and Base Excess. Indian J Med Biochem, 2018; 22(1): 85-89
10. Rajini Samuel,Vyshnavi, Vanaja, Ragashree, Balaji Rajagopalan Graphical Analysis of Arterial Blood Gas Analysis Using Standard Base Excess, Int. J. Pharm. Sci. Rev. Res., 2017; 46(1) : 223-228
11. Barnette, L. & Kautz, D.D. Creative ways to teach arterial blood gas interpretation. Dimensions of Critical Care Nursing (DCCN), 2013; 32(2): 84-87.
12. Doig AK, Albert RW, Syroid ND, Moon S, Agutter JA. Graphical arterial blood gas visualization tool supports rapid and accurate data interpretation Comput Inform Nurs, 2011; 29(4): 53-60
13. Samuel, R. Revised Graphical Tool for ABG Interpretation using Modified Bicarbonate/Standard Bicarbonate Ratio. International Journal of Clinical Chemistry and Laboratory Medicine (IJCCLM), 2019; 5(3): 19-29.

14. Rajini Samuel Application of Compensation Rules in the Four-quadrant Graphical Tool for Arterial Blood Gas Interpretation: A Cross-sectional Study.National Journal of Laboratory Medicine. 2024;13(1): B018-BO21
15. Rajini Samuel A Graphical Representation For Aiding Arterial Blood Gas Interpretation Using Non-Respiratory And Respiratory pH WJPMR, 2018;4(12):192-202
16. L.Ebihara MD-PhD Acid-Base Balance A Respiratory Approach Assigned reading: Chapter 6, pages 83-87 from West, Respiratory Physiology-The Essentials, 7th ed.
17. Kopel J, Berdine G. Winters's formula revisited. The Southwest Respiratory and Critical Care Chronicles 2019;7(27):43-49
18. E. Wrenn Wooten Calculation of physiological acid-base parameters in multicompartment systems with application to human blood J Appl Physiol 2003; 95: 2333–2344
19. E. Wrenn Wooten Analytic calculation of physiological acid-base parameters in plasma.Journal of applied physiology1999; 326 – 334.
20. Davenport, Horace W. (1974). The ABC of Acid-Base Chemistry: The Elements of Physiological Blood-Gas Chemistry for Medical Students and Physicians (Sixth ed.). Chicago: The University of Chicago Press.
21. Jiří Kofránek Complex model of blood acid-base balance Chapter pages 1-16InMEDSOFT 2009, ISBN978-80-904326-0-4, http://www.physiome. cz/references/AcidBaseMedsoft2009.
22. H. G. Morgan Acid-Base Balance In Blood British Journal Of Anaesthesia.1969;41:196- 212
23. Panagiotis Behrakis, Miltos Vasileiou, Loukos A., Theakos N. Latest developments in graphic diagnostic approach of arterial blood gases disturbances Pneumon 2004, 17(2):150-158.
24. N. Theakos, A. Loukos*, M. Vassiliou**, M. Moukas*, M. Zaka* And P.K. Behrakis pH-logPaCO2 diagram for the diagnostic approach of simple acid-base disorders 2nd Mediterranean Congress on Thoracic Diseases Athens, Greece 4-7 March 1998
25. Rajini Samuel T. Book: Novel Models of Acid Base Balance – ABG Interpretation 2019 LAP LAMBERT Academic Publishing. ISBN: 978 -620-0-30040-9
26. Rajini Samuel T. Book: Novel Graphical Tools – ABG Interpretation 2019 Mahi Publication, ISBN NUMBER : 978-93-89339-22-2
27. Rajini Samuel T. Chapter Who Is Balancing: Is It RBC or Acid-Base Status? Intech Open Publisher; pages 1-15 DOI: http://dx.doi.org/10.5772/intechopen.84768
28. Jørgensen, K., and P. Astrup. "Standard bicarbonate, its clinical significance, and a new method for its determination." Scandinavian Journal of Clinical & Laboratory Investigation.1957;9(2): 122-132.
29. Samuel R. Application of Boston Compensation Rules in the Development of a Stepwise Approach for Novel Diagnostic Arterial Blood Gas Interpretation Method. Indian J Crit Care Med.2023;27(10):717– 723
30. Rajini Samuel. Stepwise Approach In Arterial Blood Gas (ABG) Interpretation Using Novel Acid Base Balance Theory Afr. J. Biomed. Res. 2024; 27(3S): 3398-3404