Acid - Base Disturbances[i]

Bio390

1. The reported data are normal values for a healthy individual breathing room air at sea level.

arterial *p*H: 7.40

$[HCO_3^-]_{plasma}$: 24 mM

P_{aCO2}: 40 mm Hg

α for CO_2: $\frac{0.03\ mmols}{L\ mmHg}$

apparent *pKa* for carbonic acid-bicarbonate buffer = 6.10

The plasma of an individual exhibiting **hyperpnea** (hyperventilation) was analyzed and the following data obtained:

$[HCO_3^-]$: 8 mM **pH: 7.22** P_{aCO2}: 20 mm Hg

Are these results consistent with the interpretation that the hyperpnea is the result of respiratory acidosis or metabolic acidosis? What is the basis for your answer?

The condition is overall acidotic.

Now, let's look for the cause. The easiest thing is to look for respiratory causes first. This acidosis is obviously not respiratory -- the P_{aCO2} is LOWER than normal, exactly as would be expected from the hyperventilation (hyperpnea). Low P_{aCO2} is associated with alkalosis, not acidosis.

Thus, the acidosis has a non-respiratory cause.

But there is still more to this. The hyperventilation is either a compensation or an additional disturbance whose magnitude is not as large as the magnitude of the "metabolic" disturbance. Thus, the person also has respiratory alkalosis. We would describe the overall condition as acidotic with its main cause being metabolic (non-respiratory) acidosis by modified by respiratory alkalosis. (Again, the overall effect however, is acidosis in this case.)

[i] thanks to Dr. J.F. Anderson, Dept Zoology, Univ of Florida, Gainesville for the original idea for this problem

We have said that what we have here is <u>uncompensated acidosis with a major non-respiratory acidosis as its cause; it is complicated by respiratory alkalosis.</u> Such a condition <u>could be perfectly normal</u> -- it would be present in any sprinter at the end of a race! In this case, the acidosis would be caused principally by lactic acid and the hyperventilation would be an attempt (incomplete) at compensation.

2. The data in the table refer to plasma measurements of individuals exhibiting various acid-base disturbances.

INDIVIDUAL	$P_{a}CO2$ (mm Hg)	$[HCO_3^-]$ mM	*p*H	$[H^+]$ $\frac{nanomol}{L}$
A	29	22.0	7.50	31.6
B	33	32.0	7.61	24.8
C	35	17.5	7.32	48.0
D	60	37.5	7.42	38.0

Identify the specific type of acid-base disturbance for each individual.

The best procedure is to first decide if the overall condition is normal, acidotic or alkalotic. This is done simply by comparing the observed *p*H with the normal value for arterial blood, 7.4. *Note however, that even if the $_{pH}$ is normal, there may still be a compensated disturbance although you will probably not be able to tell what caused the original disturbance without additional data.*

Next, if there is a disturbance, it is easiest to decide if there is a respiratory cause. As we learned in class, any P_{aCO2} that is above 40 torr is respiratory acidosis; P_{aCO2} below 40 torr is respiratory alkalosis.

Now, without a Davenport diagram, the going gets tougher but you may still be able to go! The most obvious will be when the *p*H predicts a condition different from the P_{aCO2}. As in the last problem, let's assume that the arterial *p*H predicts acidosis but the P_{aCO2} predicts respiratory alkalosis. Then, the only way you can get an overall acidosis is if there is a larger magnitude non-respiratory acidosis and so the "diagnosis" is non-respiratory acidosis accompanied by respiratory alkalosis" or partial compensation or whatever. Likewise, a normal *p*H accompanied by a P_{aCO2} that suggests, for example, respiratory alkalosis, means that compensation has occurred (but you won't know if the original cause was respiratory or non-respiratory -- Why?).

Where this approach fails (without a Davenport) is in detecting situations where there are respiratory and metabolic disturbances of the same type (which will obviously agree with the overall *p*H disturbance).

However, even here you can approximate a bit because you know that the normal arterial value has [bicarbonate] = 24 mM. You know that the blood buffer line's bicarb values increase as the *p*H goes down and decreases from 24 as *p*H goes up.

So, if you see an overall acidosis with resp. acidosis where the bicarb value below 24 you know that it must also be complicated by non-respiratory acidosis. The opposite is true with alkalosis -- I leave it to you to work out the rules. However, remember that this method is imprecise because the blood buffer line has a negative, not a zero slope. Go back and look at the line -- the greater the acidosis or alkalosis, the more this approximation will fail to detect some conditions. But, lacking a diagram, it is better than nothing!

OK, here we go:

A: Overall alkalotic (see *p*H)
P_{aCO2} = 29 argues for resp. alkalosis
Is there a non-respiratory cause? Can't tell in this case for sure but the best bet is that there is none since the [bicarb] is slightly lower than normal, as expected in resp. alk. where the *p*H disturbance is small. So, without a Davenport, this is ANS (A) uncompensated respiratory alkalosis

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B. Overall alkalotic.
The low P_{aCO2} argues for respiratory alkalosis.

Is there a non-respiratory cause? The [bicarb] is 33 mM which is way above 24 mM. In overall alkalosis, if the [bicarb] can is greater than 24 then there must also be non-respiratory alkalosis. This is because at any *p*H > 7.4 the blood buffer line intersects [bicarb] at values less than 24 (see Davenport -- remember the neg. slope and Normal pt). Thus:

ANS (B) respiratory and non-respiratory alkalosis

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C. Overall acidotic.
However, the P_{aCO2} is lower than 40 torr which indicates respiratory alkalosis. This, coupled with the fact that the overall condition is acidotic means that there must also be non-respiratory acidosis. We can back this up with the fact that the [bicarb] is 17.5 which is far

below its "minimum" of 24 for overall acidosis with only a respiratory cause (note I have already said that the respiratory effect was alkalosis in any case).

ANS (C) non- respiratory acidosis and respiratory alkalosis

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D. Overall alkalosis but very close to normal (7.4). First notice the severe respiratory acidosis -- the P_{aCO2} is 60 torr! This means that the non-respiratory the [bicarb] is very high compared to normal and the total amount of buffer present is greater (since we are coming closer to the *pKa*). This immediately tells us that there must also be non-respiratory alkalosis or else the *p*H would be acidic. Furthermore, notice that for a slightly alkalotic condition, the [bicarb] was well over 24 it was 37.5 mM! This also indicates metabolic alkalosis.

Now, the *p*H is very close to normal. This could be viewed as either non-respiratory alkalosis with respiratory acidosis or *probably more correctly as compensated something but you don't know what the original cause was!*