Normal HCO_3^- values [2]	
Arterial blood	**Mixed venous blood**
22–26 mEq/L (mmol/L)	24–28 mEq/L (mmol/L)

(pH > 7.45) + (↑ HCO_3^-) ± (↑$PaCO_2$) [1,4]

Primary Metabolic Alkalosis [1,4]

Urinary Cl^- ≤ 25 mEq/L [4] = Chloride responsive & pH <7.6 [2]

↑ intravascular volume Or Na intolerent [2] (ex: CHF) [1]

PO acetazolamide 250-375 mg QD-BID (max effect occurs after 15 hrs) [2,8] (relative lack of effect in renal dysfunction) [5] + KCl if K^+ <3.5 mEq/L [2,8]

↓intravascular volume [2] (orthostatic hypotension, tachycardia, poor skin turgor) [5]

NS **2** at 100-125 ml/hr above total hourly fluid losses

Vol (L) = $\frac{0.2 * wt\ (kg) * (100 - plasma\ Cl^-)}{154}$ [6]

Additional ttt acc to cause:

Vomiting → metoclopramide 10mg IMI/IVI [9]

NG suction →PPI (better) or H2RA [9]

cystic fibrosis [5]***, diarrheoa ex villous adenoma*** [9]***, laxative abuse*** [6]***, post hypercapnia, prior loop/thiazide diuretic use***

- If on NS infusion → Reassess ABG Q6h & stop infusion at pH 7.45 to avoid overcorrection [8]
- Response to ttt monitored via urine pH & urine Cl^- conc [7]
- In hepatic/renal failure, ↑ vol infusion of Na & KCl may cause fluid overload or hyperkalemia. So proceed cautiously & monitor closely for complications [5]

Persistant alkalosis or initial pH > 7.6 **2**
Adm NH_4Cl [2] 5g tds PO [3]

Urinary Cl^- > 25 mEq/L [4] = Chloride resistant & pH <7.6 [2]

If patient is on ***Exogenous CS*** [2,5] — YES → ↓dose or change to CS with ↓mineralocorticoid activity **2**

NO

- ***Glucose ingestion after starvation*** [2]
- ***↑dose penicillin therapy*** [2,8]
- ***Xss alkali intake*** [2] (HCO_3^-, acetate, citrate ex in huge blood transfusion [8,9], lactate)
- ***Milk alkali syndrome:*** chronic ingestion of ↑doses of CaCO3 ± vit D for osteoporosis prevention or as antacids (accompanied by hypercalcemia) (common in renal impairment) [1,8]

YES → Adjust therapeutic regimen [2]

NO

K^+ < 3mEq/L [2,5] — YES → oral KCl [2] 40 mEq Q 4-6 hrs [7].
if impossible → KCl IV [2] infusion (10 mEq/hr) & close monitoring of serum K [7]
***Adm Mg in case of deficiency to avoid resistance to k supplementation** [6]

***check Mg levels** [6]

NO

HTN **1** — YES

NO

In case of advanced RF & resistance to acetazolamide:
HD with ↓HCO_3^- dialysate (as ↓ as 18 mmol) or buffer-free dialysate in which HCO_3^- is absent but is infused separately as needed Or PD using NS as dialysate. 1

Barter's / Gietleman syndrome (xss endogenous mineralocorticoid) 2	***OR Active Diuretic therapy*** 1
• Spironolactone/amiloride/ triamterene 1,2 • If no response →surgery 2	↓ dose Or stop diuretic if possible Or add spironolactone

- ***Current diuretic use*** 1:↓dose/stop diuretic if possible/add spironolactone
- Renal Doppler US/captopril renogram/MRI/ renal angiography to Dx ***renal artery stenosis*** 1
- ***Renin secreting tumor*** 1
- ***Accelerated HTN*** 1

HIGH ← Plasma renin activity 1

LOW ↓

Plasma aldosterone 1

LOW ↓

- dexamethasone suppression test + Obesity, Moon face, Buffalo hump, Hirsutism, Violaceous skin striae, Acne →***Cushing syndrome*** → K^+ sparing diuretic to correct alkalosis till surgery (transsphenoidal microresection of ACTH–producing pituitary adenomas & adrenalectomy for adrenal tumors.)1
- ↑tetrahydrocortisol & 5-alpha-tetrahydrocortisol : tetrahydrocortisone →***11B-HSD deficiency*** → K^+ sparing diuretic ± dexamethasone 1
- ↑plasma & urine levels of DOC & 11-deoxycortisol + Growth retardation, male (premature sexual development), female (virilaztion) →***11-hydroxylase deficiency*** 1
- ↑ DOC & ↓11-deoxycortisol + male (sexual ambiguity), female (sexual infantilism)→ ***17-hydroxylase deficiency*** 1
- ***Liddle syndrome*** →amiloride / triamterene not spironolactone 1,2
- ***Licorice ingestion*** 6 → Discontinuation corrects alkalosis; but full recovery of 11B-HSD may take 2 weeks following long-term licorice use, K^+sparing diuretics can be used during this interval. 1

HIGH ↓

- Measure aldosterone level in 24-hr urine collection after salt loading for Dx → ***1ry hyperaldosteronism*** →Spironolactone/ amiloride / triamterene 1
- Perform adrenal imaging (CT, MRI) to find its etiology 1

a) ***Adrenal adenoma/carcinoma***→ surgical removal of tumor to correct alkalosis. 1
b) ***glucocorticoid-remediable hyperaldosteronism***→ alkalosis & HTN are responsive to dexamethasone. 1
c) ***Bilateral adrenal hyperplasia*** 1

- Ps 1: In critically ill surgical patients → respiratory alkalosis due to MV, hypoxia, sepsis, hypotension, neurologic damage, pain, drugs + metabolic alkalosis due to vomiting or NG suctioning & large blood transfusions → Correct metabolic component by administering Na & KCl. Readjust ventilator or treat the underlying disorder causing hyperventilation to treat the respiratory component. 2
- Ps 2: In COPD →Chronic respiratory acidosis associated with compensatory metabolic alkalosis→ Sudden normalization of chronically ↑ PCO2 via MV can result in acute, potentially lethal ↑ in pH. So, PCO2 shouldn't be decreased rapidly in the setting of a well-compensated chronic respiratory acidosis. 7
  Aim treatment at decreasing plasma HCO_3^- with Na & KCl 2 or Acetazolamide 250 - 500mg daily 9
- Ps 3: drugs that may cause metabolic alkalosis→ barbituarates 10, carbenoloxone 9

1 https://emedicine.medscape.com/article/243160-overview
2 Dipiro Pharmacotherapy Handbook 9th Edition
3 Oxford Handook of Critical Care 3rd Ed
4 critical care work book (ACCP updates in therapeutics 2018)
5 Applied Therapeutics 2013 - The Clinical Use Of Drugs
6 the icu book paul marino 2014 (4th ed.)
7 the_washington_manual_of_critical_care [CRITICAL CARE SOURCES]
8 Accp updates in Therapeutics 2015: Fluids, Electrolytes, Acid-Base Disorders, and Nutrition Support
9 Oxford Handook of Nephrology and Hypertension 2nd Ed
10 Labtestsonline