1. A. Give TWO reasons why ATP is considered to be a high energy molecule. (10 points)

ATP is considered to be a high energy molecule because of the amount of free-energy change associated with cleavage of the phosphoanhydride bond. Several factors contribute to the large amount of energy released. First, electrostatic repulsion among the negatively charged oxygen atoms of the phosphoanhydride groups of ATP is less after hydrolysis. Second, the products of hydrolysis are better solvated that ATP itself decreasing the repulsion between phosphate groups. Third, the products of hydrolysis are more stable than ATP.

B. Why is ATP used as the major energy currency of the cell? (5 points)

ATP provides energy by group transfers, not by simple hydrolysis. ATP holds an intermediate position on the phosphoryl group transfer potential scale. Therefore, ATP can carry energy from high-energy phosphate compounds produced by catabolism to compounds such as glucose, converting them into more reactive species. In return, ADP can serve as an acceptor of phosphoryl groups from higher energy phosphate compounds yielding ATP.

Also, in aqueous solution, in addition to being thermodynamically unstable and therefore a good phosphoryl group donor, ATP is kinetically stable. ATP does not spontaneously donate phosphoryl groups to water or to any other potential cellular substrate. Only when specific enzymes are present to lower the energy of activation can phosphoryl group transfer from ATP occur.
2. Many metabolic reactions are coupled reactions. A common coupled reaction is substrate phosphorylation by ATP. For example, the oxidation of glucose begins with its phosphorylation to glucose-6-phosphate and is coupled to ATP hydrolysis. (15 points)

\[
\begin{align*}
\text{Glucose} + \text{Pi} & \leftrightarrow \text{glucose-6-phosphate} + \text{H}_2\text{O} \quad \Delta G^0' = 14 \text{ kJ/mol} \\
\text{ATP} + \text{H}_2\text{O} & \leftrightarrow \text{ADP} + \text{Pi} \quad \Delta G^0' = -30.5 \text{ kJ/mol}
\end{align*}
\]

A. In muscle cells at 37°C, the steady state ratio of [ATP]/[ADP] is 12. Assuming that glucose and glucose-6-phosphate (G6P) achieve equilibrium values in muscle, what is the ratio of [G6P] to [glucose]?

The phosphorylation of glucose coupled to ATP hydrolysis is the sum of the two reactions shown above.

\[
\begin{align*}
\text{ATP} + \text{glucose} & \leftrightarrow \text{glucose-6-phosphate} + \text{ADP} \quad \Delta G^0' = -16.5 \text{ kJ/mol}
\end{align*}
\]

At equilibrium, \( \Delta G = 0 \) and \( \Delta G^0' = -RT \ln \text{Keq} \)

\[
\text{Keq} = e^{-\Delta G^0'/RT} = \frac{[\text{ADP}][\text{G6P}]}{[\text{ATP}][\text{glucose}]}
\]

Since [ATP]/[ADP] = 12

\[
\begin{align*}
\frac{[\text{G6P}]}{[\text{glucose}]} &= 12 e^{-\Delta G^0'/RT} \\
&= 12 e^{(-16500J/mol)/(8.314JK/mol)(310K)} \\
&= 12 e^{6.40} \\
&= 7233
\end{align*}
\]

B. Each of the nine glycolytic intermediates between glucose and pyruvate is phosphorylated. Give ONE function of the phosphoryl groups.

The phosphoryl groups appear to have three different functions:

1. They are ionized at pH 7, giving each glycolytic intermediate a negative charge. Because the plasma membrane is impermeable to charged molecules, the phosphorylated intermediates cannot diffuse out of the cell. After the initial phosphorylation, no further energy is necessary to retain phosphorylated intermediates in the cell, despite the large difference in their intracellular and extracellular concentrations.
2. Phosphoryl groups are essential components in the enzymatic conservation of metabolic energy. Energy released in the breakage of phosphoanhydride bonds is partially conserved in the formation of phosphate esters such as glucose-6-phosphate. High-energy phosphate compounds formed in glycolysis can donate phosphoryl groups to ADP to form ATP.

3. Binding energy resulting from the binding of phosphate groups to the active sites of enzymes lowers the activation energy and increases the specificity of the enzymatic reactions.

3. Patients in shock experience decreased delivery of O₂ to tissues, decreased activity of the pyruvate dehydrogenase complex, and increased anaerobic metabolism. Excess pyruvate is converted to lactate, which accumulates in tissues and in the blood, causing lactic acidosis. (20 points)

A. What is the function of the pyruvate dehydrogenase complex?

The function of the pyruvate dehydrogenase complex is to oxidize pyruvate to CO₂ and acetyl-CoA.

B. Since O₂ is not a reactant or product of the citric acid cycle, why do low levels of O₂ decrease the activity of the pyruvate dehydrogenase complex?

The NADH produced by oxidative reactions of the TCA cycle must be recycled back to NAD⁺, a required co-factor for the pyruvate dehydrogenase complex. When O₂ levels are low, fewer NADH molecules are reoxidized by O₂ via electron transport and oxidative phosphorylation. Therefore, the activity of the pyruvate dehydrogenase complex decreases.

C. To alleviate lactic acidosis, shock patients are sometimes given dichloroacetate, which inhibits pyruvate dehydrogenase kinase. How does this treatment affect the activity of the pyruvate dehydrogenase complex?

Pyruvate dehydrogenase kinase catalyzes the phosphorylation of the pyruvate dehydrogenase complex, thereby inactivating it. Inhibition of the kinase shifts the pyruvate dehydrogenase complex to its more active form.
4. The disease beriberi, which results from a dietary deficiency of vitamin B1 (thiamine), is characterized by neuroloic and cardiac symptoms, as well as increased levels of pyruvate and α-ketoglutarate in the blood. (15 points)

How does a deficiency of thiamine account for the increased levels of pyruvate and α-ketoglutarate?

Thiamine (Vitamin B1) is required for the synthesis of thiamine pyrophosphate (TPP), a coenzyme in the pyruvate dehydrogenase and α-ketoglutarate dehydrogenase complexes. TPP plays an important role in the cleavage of bonds adjacent to a carbonyl group, such as the decarboxylation of α-keto acids. A thiamine deficiency reduces the activity of these enzyme complexes and causes an observed increase in levels of pyruvate and α-ketoglutarate.

5. You have isolated mitochondria and are measuring their respiratory function in a medium that initially contains only buffer and the substrate succinate. You measure their relative rate of oxygen consumption using an oxygen electrode.

The following graph illustrates the consumption of oxygen as a function of time. Reproduce the graph in your blue book and by extending the graphed line, show how the sequential addition of the substances indicated would alter the amount of oxygen consumed in mitochondria. (20 points)
For each addition, briefly explain the biochemical reason for the change in oxygen consumed that you have indicated:

1. ADP + Pi

   Consumption of ADP and Pi to make ATP have no effect on oxygen consumption, so it continues at the normal rate (i.e. oxygen is consumed, so concentration measured by electrode decreases with time).

2. Oligomycin (inhibits the F_0F_1 ATP synthase)

   Electron transfer is normally coupled to ATP synthesis, so that continued electron transfer and oxygen consumption requires ATP synthesis. Inhibiting the synthase will therefore stop oxygen consumption.

3. An uncoupling agent

   ATP synthesis remains inactivated. Electron transport resumes and continues, and oxygen consumption resumes because uncouplers allow electron transport in the absence of ATP synthesis.

4. Rotenone (inhibits electron transport between NADH and FMN in Complex I)

   Rotenone inhibits electron transport at Complex I. In this case the substrate is succinate so electrons are donated to Complex II, thus bypassing Complex I. Therefore, the drug has no effect on oxygen consumption.

5. Cyanide (prevents cytochrome a/a₃ from participating in electron transfer in Complex IV)

   Inhibiting cytochrome a/a₃ oxidase in Complex IV also blocks electron transfer so oxygen consumption stops.
6. Preparation of $[\gamma^{32}\text{P}]\text{ATP}$ (15 points)

Highly radioactive ATP labeled with $^{32}\text{P}$ in the $\gamma$ position is used extensively in metabolic studies. In one procedure to produce $[\gamma^{32}\text{P}]\text{ATP}$, investigators incubated the following components:

- 1 L 50 mM pH 8 buffer
- 10 mM magnesium chloride
- 2 mM reducing agent
- 0.4 mM glyceraldehyde-3-phosphate
- 0.05 mM NAD$^+$
- 0.2 mM ADP (not radioactive)
- 0.4 mg glyceraldehyde-3-phosphate dehydrogenase
- 0.2 mg phosphoglycerate kinase
- Small amount of $^{32}\text{P}$-labeled sodium phosphate

After the mixture was incubated for one hour, the ATP was recovered. Almost all of the $^{32}\text{P}$ was found in the $\gamma$ position of ATP.

A. How does this procedure work? Briefly describe the mechanisms.

The two enzymes catalyze a portion of the glycolytic pathway. First, glyceraldehyde-3-phosphate dehydrogenase converts glyceraldehyde-3-phosphate by oxidation and phosphorylation to 1,3-bisphosphoglycerate. This step uses inorganic phosphate to phosphorylate NOT ATP. Subsequently, phosphoglycerate kinase converts 1,3-bisphosphoglycerate to 3-phosphoglycerate. The phosphoglycerate kinase step produces ATP by transferring the high energy phosphoryl group from the carboxyl group of 1,3-bisphosphoglycerate to ADP. The $^{32}\text{P}$ incorporated as $32\text{Pi}$ in the first step is transferred in the second step, forming the radioactive $[\gamma^{32}\text{P}]\text{ATP}$.

B. What would you predict the outcome to be if an excess of arsenate ($\text{AsO}_4^{3-}$), was included in the reaction mixture. Explain your reasoning.

Arsenate, like phosphorus, is in Group V of the periodic table and is an analog of inorganic phosphate (Pi). Many enzymes that require phosphate will also use arsenate. Arsenate would compete for Pi in the active site of glyceraldehyde 3-phosphate dehydrogenase reaction to yield an unstable analog of 1,3-bisphosphoglycerate called 1-arseno-3-phosphoglycerate. This compound rapidly hydrolyzes in water non-enzymatically to yield 3-phosphoglycerate. So in the presence of arsenate, glycolysis can proceed from 3-phosphoglycerate but the ATP production reaction involving 1,3-bisphosphoglycerate is bypassed. In our example, no ATP would be formed.
Inorganic Cumulative Exam

March 30, 2002

(a) Overall stoichiometry

\[ 3 \text{V(IV)} + \text{Cr(VI)} \rightarrow 3 \text{V(V)} + \text{Cr(III)} \]

(b) \[
\frac{d[\text{Cr(III)}]}{dt} = k_2[\text{V(IV)}][\text{Cr(VI)}]
\]

because the next step is fast.

Steady-state in [Cr(VI)]

\[
\frac{d[\text{Cr(VI)}]}{dt} = k_1[\text{V(IV)}][\text{Cr(VI)}] - k_1[\text{V(V)}][\text{Cr(V)}] - k_2[\text{V(IV)}][\text{Cr(V)}] = 0
\]

\[
[\text{Cr(VI)}] = \frac{k_1[\text{V(IV)}][\text{Cr(VI)}]}{k_1[\text{V(V)}] + k_2[\text{V(IV)}]}
\]

So

\[
\frac{d[\text{Cr(III)}]}{dt} = \frac{k_1 k_2 [\text{V(IV)}]^2 [\text{Cr(VI)}]}{k_1 [\text{V(V)}] + k_2 [\text{V(IV)}]}
\]

From the stoichiometry

\[
-\frac{d[\text{V(IV)}]}{dt} = 3 \frac{d[\text{Cr(III)}]}{dt} = \frac{3 k_1 k_2 [\text{V(IV)}]^2 [\text{Cr(VI)}]}{k_1[\text{V(V)}] + k_2[\text{V(IV)}]}
\]

(c) \text{Cr(VI)} is chromate ion, \text{CrO}_4^{2-}, in base, but in acid it can add a proton to give \text{CrO}_4^{3-} and this species can dimerize to give the dichromate ion, \text{Cr}_2\text{O}_7^{2-} (which is not taken into account in the above mechanism).
\[ \text{Cr}^{(III)} = \text{Cr(H}_2\text{O)}_{6}^{3+} = \]
\[ \text{Cr(OH)(H}_2\text{O)}_{5}^{2+} \]

\[ \text{V}^{(IV)} = \text{VO(H}_2\text{O)}_{4}^{2+} = \]

\[ \text{V}^{(V)} = (\text{VO}^{+})_{2}^{2-} = \]

\[
\begin{align*}
\text{Cr}^{(III)} & = \text{Cr(H}_2\text{O)}_{6}^{3+} = \text{Cr(OH)(H}_2\text{O)}_{5}^{2+} \\
\text{V}^{(IV)} & = \text{VO(H}_2\text{O)}_{4}^{2+} = \\
\text{V}^{(V)} & = (\text{VO}^{+})_{2}^{2-} =
\end{align*}
\]

\[
\begin{align*}
\text{Cr}^{(III)} & = \text{Cr(H}_2\text{O)}_{6}^{3+} = \text{Cr(OH)(H}_2\text{O)}_{5}^{2+} \\
\text{V}^{(IV)} & = \text{VO(H}_2\text{O)}_{4}^{2+} = \\
\text{V}^{(V)} & = (\text{VO}^{+})_{2}^{2-} =
\end{align*}
\]
(50). ZnC(a) with excess $\text{[Fe(H}_2\text{O)}_6^{2+}]$, the forward reaction first-order rate constant is $k_{\text{on}} [\text{Fe(H}_2\text{O)}_6^{2+}]$ and the reverse rate constant is $k_{\text{off}}$ so for 1st order reversible

$$k_{\text{obs}} = k_{\text{on}} + k_{\text{off}}$$

To obtain $k_{\text{obs}}$, the variation of $[\text{NO}]$ and $[\text{Fe(H}_2\text{O)}_5\text{NO}]^2^+$ with time is measured. (b) Variation of the level of excess $[\text{Fe(H}_2\text{O)}_6^{2+}]$ will permit the evaluation of $k_{\text{on}}$ and $k_{\text{off}}$ from $k_{\text{obs}}$ change.

$$k_{\text{obs}} \rightarrow k_{\text{off}} \left[\text{Fe(H}_2\text{O)}_6^{2+}\right]$$

slope = $k_{\text{on}}$

(c) $\Delta H^{\ddagger}_{\text{on}} = 37.1 \text{ kJ mol}^{-1}$, $\Delta S^{\ddagger}_{\text{on}} = -3 \text{ JK}^{-1}\text{mol}^{-1}$

$$k_{\text{on}} = \left(\frac{h}{2\pi}\right)^{\frac{1}{2}} e^\frac{\Delta S^{\ddagger}}{R} e^{-\frac{\Delta H^{\ddagger}}{RT}}$$

$$\frac{h}{2\pi} = \frac{1.38 \times 10^{-23} \text{J K}^{-1}}{6.63 \times 10^{-34} \text{J s K}^{-1}} = 6.21 \times 10^{12} \text{ s}^{-1}$$

$$\frac{\Delta S^{\ddagger}}{R} = -3 \text{ JK}^{-1}\text{mol}^{-1}$$

$$\frac{\Delta H^{\ddagger}}{RT} = 37.1 \times 10^3 \text{ J mol}^{-1} \times \frac{8.314 \text{ J K}^{-1} \text{mol}^{-1} \text{K}^{-1}}{298 \text{ K}} = 15.0 \text{ s}$$

$$e^{-0.361} = 0.697$$

$$e^{15.0} = 3.06 \times 10^6$$

$$k_{\text{on}} = 6.21 \times 10^{12} \times 0.697 \times 3.06 \times 10^6 \text{ M}^{-1} \text{s}^{-1} = 1.32 \times 10^6 \text{ M}^{-1} \text{s}^{-1}$$
Stereoselectivities:

1. \( \text{MgBr} + \text{CH}_x \rightarrow \text{OH} \)

Cram's "Chelation Control" Model:

(prior coordination to Mg^+)

Note that Felkin-Ahn model doesn't apply to chelate

2. \( \text{OPMB} \)

\[ \text{MOOM} \text{OSiMe}_3 \]
Physical Chemistry
Cumulative Exam
Saturday March 30, 2002

1. Since the gases in both the L and R compartments are ideal, we have in both states i and f

\[ P_L V_L = N_L R T_L \quad \text{and} \quad P_R V_R = N_R R T_R \]

where

\[ N_L = N_{\text{HCl}} \quad \text{and} \quad N_R = N_{\text{CO}_2}. \]

2. Specializing to the final state f, we have for, respectively, the L and R compartments

\[ P_f V_{L_f} = N_{\text{HCl}} R T_f \quad \text{and} \quad P_f V_{R_f} = N_{\text{CO}_2} R T_f. \]

Eqs. (3) give \( \frac{P_f}{RT_f} = \left( \frac{N_{\text{HCl}}}{V_{L_f}} \right) = \left( \frac{N_{\text{CO}_2}}{V_{R_f}} \right) \) or

\[
\frac{N_{\text{HCl}}}{V_{L_f}} = \frac{N_{\text{CO}_2}}{V_{R_f}}.
\]

4. Since the system as a whole is isolated

(i) \( \Delta U = U_f - U_i = 0 \)
But \( \Delta U = \Delta U_L + \Delta U_R \) and thus Eq. (5) gives
\[
\Delta U_L = -\Delta U_R
\]

where
\[
\Delta U_L = U_{L_f} - U_{L_i} \quad \text{and} \quad \Delta U_R = U_{R_f} - U_{R_i}.
\]

However for a nonreactive one-phase system
(we are both the L and R subsystems)
\[
dU = \left[ \frac{\partial U}{\partial T} \right]_V \ dT + \left[ \frac{\partial U}{\partial V} \right]_T \ dV.
\]

For ideal gases however \( \left[ \frac{\partial U}{\partial V} \right]_T = 0 \) and
thus Eq. (8) becomes
\[
dU = \left[ \frac{\partial U}{\partial T} \right]_V \ dT = n C_v(T) \ dT.
\]

Applying Eq. (9) for both the L and R systems
assuming the molar heat capacities \( C_v \) are \( T \)-
independent gives for the L (HCl) system
\[
\Delta U_L = U_{L_f} - U_{L_i} = n_{HCl} C_{v,HCl} (T_f - T_i), \quad (10a)
\]

Similarly for the R (CO\(_2\)) system
\[
\Delta U_R = U_{R_f} - U_{R_i} = n_{CO_2} C_{v,CO_2} (T_f - T_i). \quad (10b)
\]
Combining Eqs. (16) and (10) gives

\[ \Delta H_{\text{HCl}} C_{V_{\text{HCl}}} (T_f - T_{li}) = -\Delta H_{\text{CO}_2} C_{V_{\text{CO}_2}} (T_f - T_{ri}) \]

Solving Eq. (11) for \( T_f \) yields

\[
T_f = \frac{\Delta H_{\text{HCl}} C_{V_{\text{HCl}}} T_{li} + \Delta H_{\text{CO}_2} C_{V_{\text{CO}_2}} T_{ri}}{\Delta H_{\text{HCl}} C_{V_{\text{HCl}}} + \Delta H_{\text{CO}_2} C_{V_{\text{CO}_2}}} \]

From Eq. (13), we have that

\[
T_{li} = \frac{P_{li} V_{li}}{\Delta H_{\text{HCl}}} \quad \text{and} \quad T_{ri} = \frac{P_{ri} V_{ri}}{\Delta H_{\text{CO}_2}} \]

and from the first of Eqs. (3)

\[
T_f = \frac{P_f V_{li}}{\Delta H_{\text{HCl}}} \]

Inserting Eqs. (13) and (14) into Eq. (12) gives

\[
\frac{P_f V_{li}}{\Delta H_{\text{HCl}}} = \frac{C_{V_{\text{HCl}}} \left( \frac{P_{li} V_{li}}{R} \right) + C_{V_{\text{CO}_2}} \left( \frac{P_{ri} V_{ri}}{R} \right)}{\Delta H_{\text{HCl}} C_{V_{\text{HCl}}} + \Delta H_{\text{CO}_2} C_{V_{\text{CO}_2}}} \]

The previous equation can be rewritten as

\[
P_f = \frac{\Delta H_{\text{HCl}} P_{li} C_{V_{\text{HCl}}} (V_{li}) + \Delta H_{\text{HCl}} P_{ri} C_{V_{\text{CO}_2}} V_{ri}}{\Delta H_{\text{HCl}} C_{V_{\text{HCl}}} + \Delta H_{\text{CO}_2} C_{V_{\text{CO}_2}}} \]
4. However, from Eq. (4) \( \frac{\Delta V_{L_1}}{V_{L_5}} = \frac{\Delta V_{c_2}}{V_{R_3}} \) and thus the previous equation becomes

\[
P_f = \frac{\Delta V_{L_1} P_{L_1} C_v, \text{HCl} \left( \frac{V_{L_1}/V_{L_5}}{V_{R_1}/V_{R_5}} \right) + \Delta V_{c_2} P_{R_3} C_v, \text{CO}_2 \left( \frac{V_{R_1}/V_{R_5}}{V_{R_1}/V_{R_5}} \right)}{\Delta V_{L_1} C_v, \text{HCl} + \Delta V_{c_2} C_v, \text{CO}_2}
\]

C. From the data table on page 1 of the exam, near room temperature \( \approx 300 \) K, the vibrational modes do not contribute to either \( C_v, \text{HCl} \) or \( C_v, \text{CO}_2 \) (since all \( \Theta_{vib} \) 's \( \approx \) 300 K). Thus: for HCl, the best cavity is that of a linear rigid rotor, namely

\[
C_v, \text{HCl} = \frac{5}{2} R
\]

For CO\(_2\), the data table shows four normal modes. Thus by the \( 3N-6 \) rule for \( N = 3 \), CO\(_2\) has two rotational degrees of freedom and therefore is also linear. Thus

\[
C_v, \text{CO}_2 = \frac{5}{2} R = C_v, \text{HCl}
\]

1. Inserting Eqs. (17) into Eq. (12) and cancelling the \( C_v \) terms
\[ T_f = \frac{\Omega_{\text{HCl}}}{\Omega_{\text{HCl}} + \Omega_{\text{CO}_2}} T_{\text{Li}} + \frac{\Omega_{\text{CO}_2}}{\Omega_{\text{HCl}} + \Omega_{\text{CO}_2}} T_{\text{Ri}} \]

Using Eq. (5) of the exam, however, permits us to rewrite Eq. (18) as

\[ T_f = x_{\text{HCl}} T_{\text{Li}} + x_{\text{CO}_2} T_{\text{Ri}} \]

(ii) Similarly inserting Eqs. (17) into Eq. (15) gives

\[ P_f = x_{\text{HCl}} P_{\text{Li}} \left( \frac{V_{\text{Li}}}{V_f} \right) + x_{\text{CO}_2} P_{\text{Ri}} \left( \frac{V_{\text{Ri}}}{V_f} \right) \]

\[ a. \] From the data table on page 4, near \( T = 1300 \text{ K} \), the symmetric band modes contribute to \( C_v, \text{CO}_2 \) since \( 1300 \text{K} \) \( \Theta_{\text{v18}} (3\beta) = 960 \text{ K} \). The \( \text{HCl} \) mode and the two stretching modes of \( \text{CO}_2 \) do not contribute appreciably to the \( C_v \)'s since their \( \Theta_{\text{v18}}'s \) are apparently greater than 1300 K. We will assume that each of the \( \text{CO}_2 \) bending modes contribute their full equiatomic value \( R \) to \( C_v, \text{CO}_2 \). Then near \( T = 1300 \text{ K} \)

\[ C_v, \text{HCl} = \frac{5}{2} R \]
\[ C_{v,CO_2} = \frac{5}{2} R + 2R = \frac{9}{2} R. \]

Inserting Eqs. (21) into Eq. (12) and cancelling on

\[ T_f = \frac{5 \text{N}_\text{HCl} T_{Li} + 9 \text{N}_\text{CO}_2 T_{Ri}}{5 \text{N}_\text{HCl} + 9 \text{N}_\text{CO}_2} \]

Dividing the numerator and denominator in the previous equation by \( \text{N}_\text{HCl} + \text{N}_\text{CO}_2 \) and using Eq. (5) of the exam gives

\[ T_f = \frac{5 \times \text{HCl} T_{Li} + 9 \times \text{CO}_2 T_{Ri}}{5 \times \text{HCl} + 9 \times \text{CO}_2} \]

Using Eqs. (21) into Eq. (15) and proceeding similarly yields

\[ P_f = \frac{5 \times \text{HCl} P_{Li} \left( \frac{V_{Li}}{V_{L_f}} \right) + 9 \times \text{CO}_2 P_{Ri} \left( \frac{V_{Ri}}{V_{R_f}} \right)}{5 \times \text{HCl} + 9 \times \text{CO}_2} \]

\[ \text{E.} \]

\[ \text{(i) Compute } \times_\text{HCl} \text{ and } \times_\text{CO}_2 \]

\[ \text{Since } V_{L_f} = 213 V, \quad V_{R_f} = 213 V \]
Thus Eq. (4) becomes

\[ \frac{\Omega_{\text{HCl}}}{213V} = \frac{\Omega_{\text{CO}_2}}{213V} \quad \text{or} \quad \Omega_{\text{HCl}} = 2 \Omega_{\text{CO}_2} \]

implies

\[ \Omega_{\text{HCl}} = 2 \Omega_{\text{CO}_2} \]

But since \( \Omega_{\text{HCl}} + \Omega_{\text{CO}_2} = 1 \), it follows from Eq. (25) that

\[ \Omega_{\text{HCl}} = \frac{2}{3} \quad \text{and} \quad \Omega_{\text{CO}_2} = \frac{1}{3} \]

(ii) Using Eq. (26) in Eq. (22) gives

\[ T_f = \frac{10 T_{L_i} + q T_{R_i}}{10 + q} = \frac{10(1250K) + q(1350K)}{10 + q} \]

or

\[ T_f = 1297.4K \]

(iii) Since

\[ V_{L_i} = 213V, \quad V_{R_i} = 213V \]

Using Eqs. (24) and (28) in Eq. (23) gives
\[ P_f = \frac{5 \times HCl \cdot P_{Li}^{4/2} + 9 \times CO_2 \cdot P_{Ri}^2}{5 \times HCl + 9 \times CO_2} \]

Using Eq. (26) in the previous equation gives:

\[ P_f = \frac{5 P_{Li} + 18 P_{Ri}}{19} \]

Now \( P_{Li} = 2.0 \) atm. To find \( P_{Ri} \) we use Eq. (1), which gives for state:

\[ \frac{P_{Ri} V_{Ri}}{P_{Li} V_{Li}} = \frac{n_{CO_2} \cdot T_{Ri}}{n_{HCl} \cdot T_{Li}} \]

Hence, using Eq. (25a) and Eq. (28) the previous equation becomes:

\[ 2 \frac{P_{Ri}}{P_{Li}} = \frac{1}{2} \frac{T_{Ri}}{T_{Li}} \]

or

\[ P_{Ri} = \frac{1}{4} \left( \frac{T_{Ri}}{T_{Li}} \right) P_{Li} \]
Using Eq. (30) in Eq. (29) gives

\[
P_f = \frac{\left[ 5 + 4.5 \left( \frac{T_R}{T_L} \right) \right] P_L}{20} = \frac{\left[ 5 + 4.5 \left( \frac{1350}{1250} \right) \right] 2.0 \text{ atm}}{20}
\]

\[
P_f = 1.038 \text{ atm}
\]