u = superficial gas velocity, m/s
v = interstitial gas velocity in bed, m/s
V = pore volume; V_a and V_i are macro- and micropore values, respectively, m^3/g

Greek Symbols

β = total porosity, sum of macro (β_a)- and micro (β_i)-porosities
$\delta_o, \delta_1, \delta_i, \delta_a, \delta_f$ = moment contributions defined by Eqs. 3 to 7
ϵ = void fraction in the bed
η = diffusibility, Eq. 12
ρ = gas density, kg/m^3
ρ_p = density of carbon particles, kg/m^3
μ_1 = first absolute moment, s
$(\mu_1)_i$ = first moment for nonadsorbable gas, s
μ_2' = second central moment, s^2
τ_a = macropore tortuosity factor

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Manuscript received September 4, 1979; revision received April 11, and accepted May 5, 1980.

Substrate-Inhibited Kinetics With Catalyst Deactivation in an Isothermal CSTR

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Analytical expressions are developed for the time-dependent reactant concentration and catalyst activity in an isothermal CSTR with Langmuir-Hinshelwood kinetics of deactivation and reaction. Several parallel and series posioning mechanisms are considered for a reactor which, without poisoning, would operate at a unique steady state. The use of matched asymptotic expansions and abandonment of the usual initial-steady-state assumption give results, valid from startup to final loss of activity, whose accuracy can be improved systematically.

I. Unique Pseudosteady State

SCOPE

One of the weaknesses of all catalysts is that they become degraded or deactivated with use. In previous papers (Do and Weiland, 1979a, 1979b), we have presented analyses of the effect of catalyst poisoning by reactants and products of reaction on the performance of a CSTR. Both n-th order and Michaelis-Menten kinetics were examined. This paper presents the same sort of problem, but for a Langmuir-Hinshelwood rate form. Previous work, which specifically tackles catalyst deactivation with substrate inhibition, includes that of Lin (1977) whose analysis was restricted to poisoning at a rate independent from the main reaction; thus, deactivation could be examined quite separately. See also Chu (1968), and Kam and Hughes (1979).

When the reactant or product is responsible for the poisoning, we have shown previously (Do and Weiland, 1980) that the rate forms for the main reaction and for deactivation must bear certain minimum relationships for consistency. Such self-consistent rate expressions are used here to analyze the effect of catalyst poisoning on reactor performance when the Langmuir-Hinshelwood parameters admit of a single steady state in the nonpoisoned case. The method of matched symptotic expansions is used to develop solutions valid over the entire period of reactor operation from initial startup to final loss of activity.

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0001-1541-80-4188-0948-$00.95.

Part II of this paper appears on page 1020 of this issue.

CONCLUSIONS AND SIGNIFICANCE

The method of matched asymptotic expansions is used to obtain a complete description of the performance of a CSTR for substrate-inhibited kinetics, when the catalyst is being slowly poisoned by either the reactant or the reaction product. Self-consistent rate expressions are used for the reaction and poisoning rates. The rate of deactivation is taken to be slower than the main reaction rate; this gives rise to the appearance of a small parameter, ϵ, which is basically the ratio of these rates. Solutions are obtained which are correct to $0(\epsilon)$ and higher order corrections can be obtained in a completely systematic way.

The usual approach taken in this type of problem is to assume that the reactor is already operating at an initial steady state before catalyst deactivation begins to occur. Here, we show that if one is to obtain a description of reactor performance correct to any order in ϵ, the initial steady state assumption must be abandoned.

It is found that for parallel (reactant) poisoning, operation at high space velocity leads to earlier loss of activity; the reverse is true for series deactivation. For systems which have large values of the Langmuir-Hinshelwood kinetic parameters, deactivation is always delayed for both series and parallel mechanisms. As the complexity of the poisoning mechanism increases, catalyst life becomes greatly extended. The weakness of the initial steady state assumption is underlined by considering reactor performance for moderately fast deactivation (but with $\epsilon<1$). In this case, the reactor never reaches steady conditions before the effects of deactivation are felt.

INTRODUCTION

The effect of substrate inhibition on the performance of chemical reactors has been the subject of many studies, mainly in the context of multiple steady-state phenomena. See for example Ho (1976), Pereira and Varma (1978), and De Vera and Varma (1979). Multiplicity is possible for this type of kinetics even under isothermal conditions. Pereira and Varma (1978) have given several examples from conventional catalysis by metals, including CO oxidation over platinum in excess oxygen; certainly substrate inhibition is common in catalysis by enzymes (Laidley and Bunting, 1973).

One of the weaknesses of all catalysts is that they become degraded or deactivated with use. Previous work, which specifically addresses the reactor problems associated with deactivation in substrate-inhibited kinetics, includes that of Lin (1977). His analysis was based on the usual Langmuir-Hinshelwood rate form:

$$R = kC_A a/(C_A^2 + K_s C_A + K) \tag{1a}$$

for the main reaction, but, it was assumed that the catalyst was denatured according to

$$R = k_d a \tag{1b}$$

Thus, inactivation took place quite independent of the main (in this case enzymatic) reaction rate. Single catalyst pellets have been studied by Chu (1968), and Kam and Hughes (1979).

As previously reported (Do and Weiland, 1980c), when poisoning is caused by the reactant or the product of reaction (parallel and series poisoning, respectively), the deactivation rate expression can only have certain basic forms if it is to be consistent with the rate expression for the main reaction. In particular, it must have the same denominator as the main kinetics. Thus, for kinetics given by Eq. 1a, the deactivation rate expression might take on one of the forms:

$$R = \begin{cases} k_d C_A^n a/(C_A^2 + K_s C_A + K), & \text{(2a)} \\ k_d C_A^{n-1} C_B a/(C_A^2 + K_s C_A + K) & \text{(2b)} \end{cases}$$

Eqs. 2a and 2b correspond to parallel and series poisoning mechanisms, respectively. The exponent n may take on the values $\{1, 2, 3\}$ and these correspond to deactivation directly by the reactant, by reactant bonding with an existing reactant-catalyst complex, and by reactant bonding with a catalyst-(two-reactant-molecule)-complex, respectively, for parallel poisoning. For series mechanisms, these values of n correspond to the analogous cases of *product* bonding with catalyst or catalyst-*reactant* complexes. Further details for this and other types of kinetics may be found in Do and Weiland (1980c).

This paper will use the self-consistent forms of Eqs. 2a and 2b to analyze the effect of parallel and series poisoning on the performance of an isothermal CSTR for the case of a *unique* pseudo-steady state. (The fact that activity is always decreasing implies that a true steady state can never be achieved). The three separate mechanisms given by $n = \{1, 2, 3\}$ will be considered, but only the analysis for a parallel mechanism and $n = 1$ will be shown in detail; in the interest of space, results for the other cases will be given in tabular form.

FORMULATION

Consider a substrate-inhibited reaction in a well-mixed, isothermal CSTR without mass transfer resistances. The nondimensional transient mass balance equations for reactant and product, respectively, are

$$A = H(1 - A) - Aa/(A^2 + \gamma A + \beta), \tag{3a}$$

$$\dot{B} = -HB + Aa/(A^2 + \gamma A + \beta) \tag{3b}$$

where the feed to the reactor has been assumed product-free. The rate expressions for catalyst activity are

$$\dot{a} = \begin{cases} -\epsilon A^n a/(A^2 + \gamma A + \beta), & \text{(3c)} \\ -\epsilon A^{n-1} B a/(A^2 + \gamma A + \beta) & \text{(3d)} \end{cases}$$

for parallel and series poisoning, respectively. The parameter H is the nondimensional space velocity, and ϵ is the ratio of the deactivation rate to that of the main reaction. Time has been scaled on the reaction rate constant and we will assume $\epsilon << 1$.

Prior to reactor startup, the catalyst has unit activity and only inert material is present in the fluid:

$$A(0) = B(0) = 0, \tag{3e}$$

$$a(0) = 1 \tag{3f}$$

At reactor startup, a product-free feed having unit reactant concentration is introduced at a fixed flow rate. Our objective is a long-time description of reactor performance.

For series deactivation mechanisms, Eqs. 3a and 3b can be combined and integrated to give

$$A + B = 1 - \exp(-Ht) \tag{4}$$

which enables the equations for series poisoning to be written solely in terms of A and a. Eq. 3d becomes:

$$\dot{a} = \frac{-\epsilon[1 - A - \exp(-Ht)]A^{n-1}a}{A^2 + \gamma A + \beta} \tag{5}$$

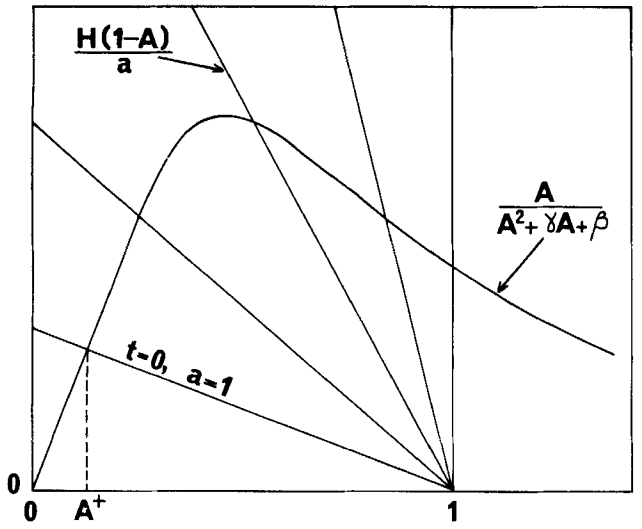

Figure 1. Operating diagram.

and the product poisoning case presents no additional difficulties over poisoning by the reactant.

If we now restrict our attention to parallel deactivation in which the reactant is directly responsible for the poisoning process ($n = 1$), the relevant equations describing the reactor become:

$$\frac{dA}{dt} = H(1 - A) - \frac{Aa}{A^2 + \gamma A + \beta} \overset{\text{def}}{=} g(A, a), \quad (6a)$$

$$\frac{da}{dt} = -\epsilon \frac{Aa}{A^2 + \gamma A + \beta} \quad (6b)$$

with initial conditions,

$$A(0) = 0, \quad (6c)$$

$$a(0) = 1 \quad (6d)$$

Note that the kinetic expressions are self-consistent.

When there is a unique quasisteady state, one can take the usual singular perturbations approach (Cole, 1968; Nayfeh, 1973) and obtain an inner solution, valid during reactor startup, and an outer solution applicable to the period of significant catalyst decay. These solutions can then be matched and a single composite solution constructed which is valid over the *entire* time of reactor operation. There is no need to assume an initial steady state, and indeed to obtain higher order corrections to the solutions, such an assumption has to be abandoned altogether. The case of a unique quasisteady state is shown schematically in Figure 1. Figure 1 shows that as t increases and a decreases, the reactor moves smoothly through a continuum of pseudosteady states given by the intersection of the line $f_1(A) = H(1 - A)/a$ with the curve $f_2(A) = A/(A^2 + \gamma A + \beta)$.

STARTUP PERIOD

Eqs. 6a, 6b, 6c, and 6d describe the behavior of the reactor on the time scale given by the chemical reaction rate. This time is appropriate to the period of reactor startup. We take advantage of the slowness of deactivation ($\epsilon << 1$) and expand the dependent variables in the following asymptotic series:

$$A(t; \epsilon) \sim A_o(t) + \epsilon A_1(t) + \ldots \quad (7a)$$

$$a(t; \epsilon) \sim a_o(t) + \epsilon a_1(t) + \ldots \quad (7b)$$

When these are used in Eqs. 6a, 6b, 6c, and 6d a sequence of subproblems results. The order unity and $0(\epsilon)$ equations are:

$$\frac{dA_o}{dt} = g(A_o, a_o), \quad (8a)$$

$$\frac{da_o}{dt} = 0, \quad (8b)$$

$$A_o(0) = 0, \quad (8c)$$

$$a_o(0) = 1 \quad (8d)$$

and

$$\frac{dA_1}{dt} = -\left[H + \frac{a_o(\beta - A_o^2)}{(\beta + \gamma A_o + A_o^2)}\right] A_1 - \frac{A_o}{(\beta + \gamma A_o + A_o^2)} a_1, \quad (9a)$$

$$\frac{da_1}{dt} = -\frac{A_o a_o}{\beta + \gamma A_o + A_o^2}, \quad (9b)$$

$$A_1(0) = 0, \quad (9c)$$

$$a_1(0) = 0 \quad (9d)$$

respectively.

The solution to Eq. 8b with Eq. 8d as initial condition is just

$$a_o(t) \equiv 1 \quad (10)$$

Combining this result with Eqs. 8a and 8c, and integrating gives:

$$Ht = \frac{A^{+2} + \gamma A^+ + \beta}{A^{+2} + \alpha_1 A^+ + \alpha_o} \ln\left(\frac{A^+}{A^+ - A_o}\right) + \frac{1}{2}\frac{(\alpha_1 - \gamma)A^+ + \alpha_o - \beta}{A^{+2} + \alpha_1 A^+ + \alpha_o} \ln\left(\frac{\alpha_o}{A_o^2 + \alpha_1 A_o + \alpha_o}\right) + \frac{\alpha_o(\alpha_1 - 2\gamma) + \alpha_1\beta + [\alpha_1(\alpha_1 - \gamma) - 2(\alpha_o - \beta)]A^+}{(A^{+2} + \alpha_1 A^+ + \alpha_o)(4\alpha_o - \alpha_1^2)^{\frac{1}{2}}} \tan^{-1}\frac{A_o(4\alpha_o - \alpha_1^2)^{\frac{1}{2}}}{2\alpha_o + \alpha_1 A_o} \quad (11)$$

in which A^+ is the only real root of

$$A^3 + (\gamma - 1)A^2 + (\beta + H^{-1} - \gamma)A - \beta = 0 \quad (12)$$

and

$$\alpha_o = \beta/A^+, \quad (13)$$

$$\alpha_1 = A^+ + \gamma - 1 \quad (14)$$

To determine the $0(\epsilon)$ solutions, we substitute the result (Eq. 10) into Eq. 9b and eliminate time between this and Eq. 8a:

$$\frac{da_1}{dA_o} = \frac{A_o}{(A_o^2 + \gamma A_o + \beta)\, g(A_o, 1)} \quad (15)$$

The solution is

$$a_1 = \frac{1}{H(A^{+2} + \alpha_1 A^+ + \alpha_o)} \left\{ A^+ \ln\left(\frac{A^+ - A_o}{A^+}\right) - \frac{A^+}{2} \ln\left(\frac{A_o^2 + \alpha_1 A_o + \alpha_o}{\alpha_o}\right) + \frac{(\alpha_1 A^+ + 2\alpha_o)}{(4\alpha_o - \alpha_1^2)^{\frac{1}{2}}} \tan^{-1}\frac{A_o(4\alpha_o - \alpha_1^2)^{\frac{1}{2}}}{2\alpha_o + A_o\alpha_1} \right\} \quad (16)$$

where we have used the initial conditions of Eqs. 9c and 9d. Eqs. 8a, 9a, and 16 can be combined to give:

$$\frac{dA_1}{dA_o} = -\frac{H + \frac{(\beta - A_o^2)}{(\beta + \gamma A_o + A_o^2)}}{g(A_o; 1)} A_1 - \frac{A_o a_1(A_o)}{g(A_o, 1)(\beta + \gamma A_o + A_o^2)} \quad (17)$$

The solution is given in terms of the quadrature:

$$A_1 = \int_o^{A_o} \eta(s) \exp\left[-\int_o^s \xi(m)dm\right] ds, \quad (18a)$$

where

$$\xi(m) = \left[H + \frac{(\beta - m^2)}{(m^2 + \gamma m + \beta)}\right] \Big/ \left[H(1 - m) - \frac{m}{m^2 + \gamma m + \beta}\right], \quad (18b)$$

$$\eta(s) = -\frac{\dfrac{s}{(s^2+\gamma s+\beta)}\,a_1(s)}{H(1-s) - \dfrac{s}{(s^2+\gamma s+\beta)}} \int_0^s \frac{x\,dx}{(x^2+\gamma x+\beta)\,g(x;1)} \tag{18c}$$

The asymptotic behavior of these solutions as $t \to \infty$ will be required for matching to the outer or long-time solution to be determined in the next section. At leading order

$$a_o \to 1, \quad A_o \to A^+ \quad \text{for} \quad t \to \infty \tag{19}$$

To find the form of a_1, we start with Eq. 9b and use the results given by Eq. 19. Thus,

$$a_1(t) \to -\frac{A^+ t}{A^{+2}+\gamma A^+ + \beta} + C \text{ for } t \to \infty \tag{20}$$

The constant of integration may be evaluated as follows: eliminate the nonlinear term between Eqs. 9b and 20. Then,

$$C = \lim_{t\to\infty}\left[a_1(t) - t\frac{da_1(t)}{dt}\right], \tag{21}$$

and combining this with Eqs. 9b and 16, and taking the limit gives

$$C = \frac{1}{H(\beta+\gamma A^+ + A^{+2})}\int_0^{A^+}\frac{(\beta - xA^+)dx}{(x^2+\alpha_1 x+\alpha_o)} \tag{22}$$

Thus,

$$C = \frac{\dfrac{A^+}{2}\ln\left(\dfrac{\alpha_o}{A^{+2}+\alpha_1 A^+ + \alpha_o}\right) + \dfrac{(2\beta+\alpha_1 A^+)}{(4\alpha_o-\alpha_1^2)^{\frac{1}{2}}}\tan^{-1}\dfrac{A^+(4\alpha_o-\alpha_1^2)^{\frac{1}{2}}}{2\alpha_o+\alpha_1 A^+}}{H(\beta+\gamma A^+ + A^{+2})} \tag{23}$$

The asymptotic behavior of $A_1(t)$ as $t \to \infty$ can be easily found from Eqs. 9a and 23 with the result

$$\lim_{t\to\infty} A_1 = -\frac{C\,A^+}{[H(A^{+2}+\gamma A^+ + \beta)+\beta - A^{+2}]} - \frac{A^{+2}}{[H(A^{+2}+\gamma A^+ + \beta)+\beta - A^{+2}]^2} + \frac{A^{+2}\,t}{(A^{+2}+\gamma A^+ + \beta)\,[H(A^{+2}+\gamma A^+ - \beta)+\beta - A^{+2}]} \tag{24}$$

Note that catalyst deactivation *does* take place at $0(\epsilon)$, even during the startup period. To obtain anything more than the first approximation to the performance of the reactor, it is absolutely essential that a quasisteady-state assumption *not* be made.

PERIOD OF SEVERE DEACTIVATION

The time scale appropriate to an examination of reactor performance during serious catalyst poisoning is based on the rate constant for deactivation. With time stretched according to

$$\tilde{t} = \epsilon t \tag{25}$$

Eqs. 6a, 6b, 6c, and 6d become

$$\epsilon\frac{d\tilde{A}}{d\tilde{t}} = g(\tilde{A}, \tilde{a}), \tag{26a}$$

$$\frac{d\tilde{a}}{d\tilde{t}} = -\frac{\tilde{A}\tilde{a}}{\tilde{A}^2+\gamma A+\beta} \tag{26b}$$

We seek solutions of the form

$$\tilde{A}(\tilde{t};\epsilon) = \tilde{A}_o(\tilde{t}) + \epsilon\tilde{A}_1(\tilde{t}) + \ldots, \tag{27a}$$

$$\tilde{a}(\tilde{t};\epsilon) = \tilde{a}_o(\tilde{t}) + \epsilon\tilde{a}_1(\tilde{t}) + \ldots \tag{27b}$$

Initial conditions for these outer solutions are obtained as a result of matching with the large time limit of the inner solutions found earlier. Rewrite t as ϵt and expand the asymptotic forms of Eqs. 27a and 27b in a Taylor series about $\tilde{t} = 0$. Then,

$$\tilde{A}(\tilde{t};\epsilon) = \tilde{A}_o(0) + \epsilon\left[\frac{d\tilde{A}_o(0)}{d\tilde{t}}\,t + \tilde{A}_1(0)\right] + \ldots, \tag{28a}$$

$$\tilde{a}(\tilde{t};\epsilon) = \tilde{a}_o(0) + \epsilon\left[\frac{d\tilde{a}_o}{d\tilde{t}}\,t + \tilde{a}_1(0)\right] + \ldots \tag{28b}$$

Now, by insisting that these forms agree with the limiting forms of the inner solutions as $t \to \infty$, we find that

$$\tilde{A}_o(0) = A^+, \tag{29a}$$

$$\tilde{a}_o(0) = 1, \tag{29b}$$

$$\tilde{a}_1(0) = C, \tag{29c}$$

$$\tilde{A}_1(0) = -\frac{C\,A^+}{[H(A^{+2}+\gamma A^+ + \beta)+\beta - A^{+2}]} - \frac{A^{+2}}{[H(A^{+2}+\gamma A^+ + \beta)+\beta - A^{+2}]^2} \tag{29d}$$

By substituting Eqs. 27a and 27b into Eqs. 26a and 26b, and equating coefficients of like powers of ϵ to zero we find that the $0(1)$ and $0(\epsilon)$ equations are, respectively

$$H(1-\tilde{A}_o) - \frac{\tilde{A}_o\tilde{a}_o}{\tilde{A}_o^2+\gamma\tilde{A}_o+\beta} = 0, \tag{30a}$$

$$\frac{d\tilde{a}_o}{d\tilde{t}} = -\frac{\tilde{A}_o\tilde{a}_o}{\tilde{A}_o^2+\gamma\tilde{A}_o+\beta} \tag{30b}$$

with Eqs. 29a and 29b as initial conditions, and

$$\frac{d\tilde{A}_1}{d\tilde{t}} = -\left[H + \frac{\tilde{a}_o(\beta-\tilde{A}_o^2)}{\tilde{A}_o^2+\gamma\tilde{A}_o+\beta}\right]\tilde{A}_1 - \frac{\tilde{A}_o\tilde{a}_1}{\tilde{A}_o^2+\gamma\tilde{A}_o+\beta} \tag{31a}$$

$$\frac{d\tilde{a}_1}{d\tilde{t}} = -\frac{\tilde{A}_o\tilde{a}_1}{\tilde{A}_o^2+\gamma\tilde{A}_o+\beta} - \frac{\tilde{a}_o(\beta-\tilde{A}_o^2)\tilde{A}_1}{\tilde{A}_o^2+\gamma\tilde{A}_o+\beta} \tag{31b}$$

with Eqs. 29c and 29d as initial conditions.

Solutions to Eqs. 30a and 30b are readily found to be

$$\tilde{a}_o = H(1-\tilde{A}_o)(\tilde{A}_o^2+\gamma\tilde{A}_o+\beta)/\tilde{A}_o, \tag{32a}$$

$$2(A^+ - \tilde{A}_o) + \beta\ln(\tilde{A}_o/A^+) + (1+\beta+\gamma)\ln(1-A^+)/(1-\tilde{A}_o) + \beta(1/A^+ - 1/\tilde{A}_o) = \tilde{t} \tag{32b}$$

These solutions allow us to integrate Eqs. 31a and 31b as:

$$\tilde{A}_1 = -\frac{\dfrac{\tilde{A}_o^2(1-\tilde{A}_o)}{2\tilde{A}_o^3+(\gamma-1)\tilde{A}_o+\beta} + \dfrac{\tilde{A}_o}{\tilde{A}_o^2+\gamma\tilde{A}_o+\beta}\,\tilde{a}_1}{H\left[1+\dfrac{(1-\tilde{A}_o)(\beta-\tilde{A}_o^2)}{A_o}\right]}, \tag{33a}$$

and

$$\tilde{a}_1(t) = \tilde{a}_1(0)\exp\left[\int_{A^+}^{\tilde{A}_o} M(x)dx\right] + \int_{A^+}^{\tilde{A}_o} N(s)\exp\left[-\int_{\tilde{A}_o}^{s} M(x)dx\right]ds, \tag{33b}$$

where

$M(x) =$

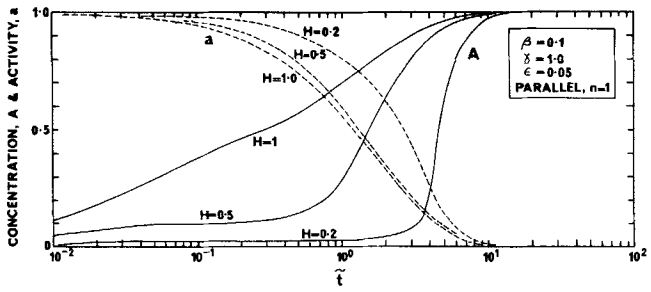

Figure 2. Effect of space velocity on concentration and activity.

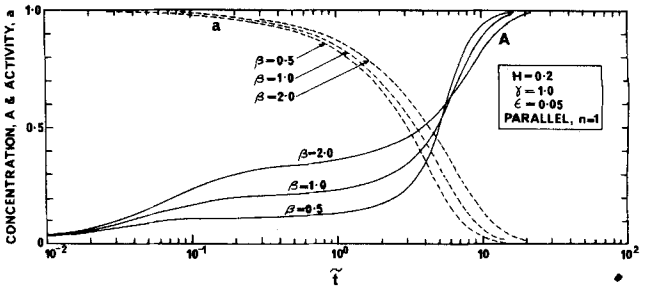

Figure 3. Dependence on kinetic parameter β.

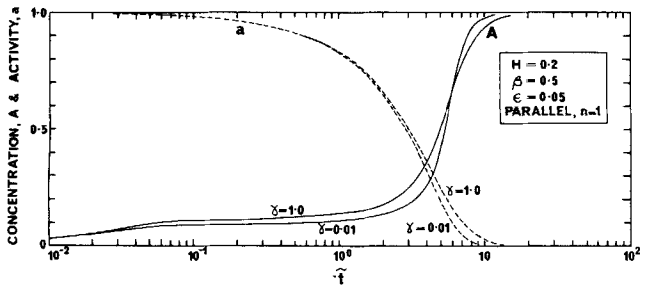

Figure 4. Dependence on kinetic parameter γ.

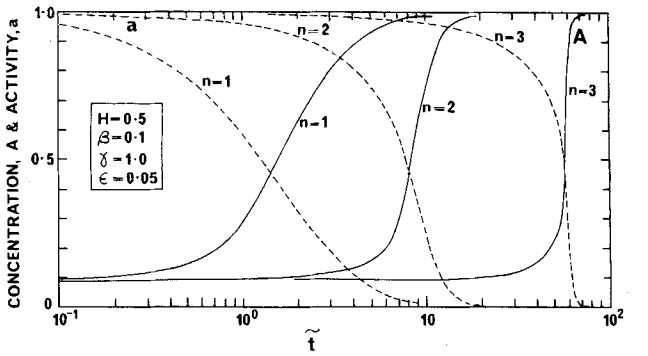

Figure 5. Influence of mechanism of parallel deactivation.

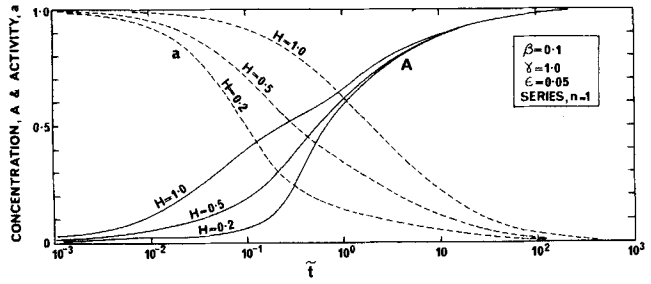

Figure 6. Effect of space velocity on concentration and activity.

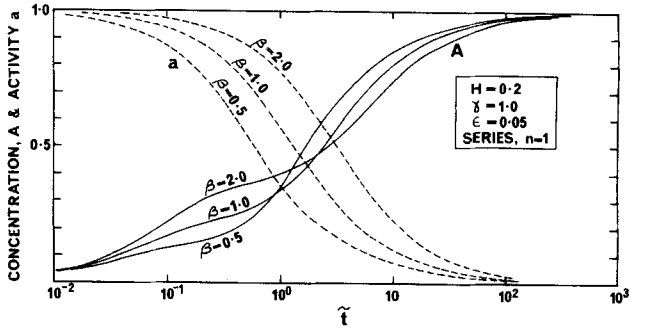

Figure 7. Dependence on kinetic parameter β.

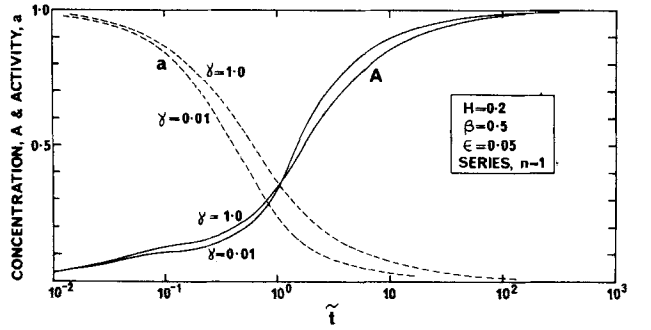

Figure 8. Dependence on kinetic parameter γ.

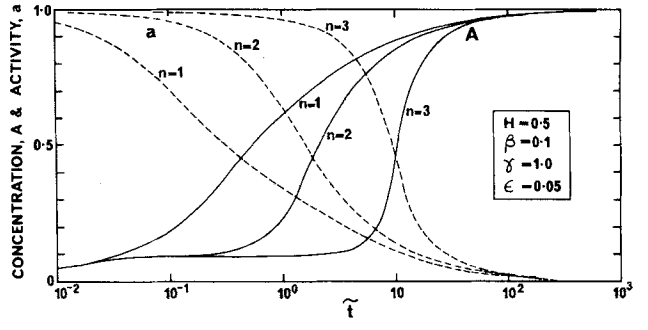

Figure 9. Influence of mechanism of series deactivation.

$$- \frac{[2x^3 + (\gamma - 1)x^2 + \beta]}{(x^2 + \gamma x + \beta)\,[x + (1 - x)\,(\beta - x^2)]\,(1 - x)}, \tag{33c}$$

and

$$N(s) = \frac{(\beta - s^2)\,(1 - s)}{[s + (1 - s)\,(\beta - s^2)]} \tag{33d}$$

Composite solutions, uniformly valid in time, can be found by adding together the inner and outer expansions and subtracting the common parts, which are:

$$A_{cp} = A^+ + \epsilon\Big[\lim_{t\to\infty} A_1 t)\Big] + 0(\epsilon^2), \tag{34a}$$

$$a_{cp} = 1 + \epsilon\Big[\lim_{t\to\infty} a_1(t)\Big] + 0(\epsilon^2) \tag{34b}$$

The limits which appear in Eqs. 34a and 34b are given by Eqs. 20 and 24.

This completes the solution to the reactor problem to $0(\epsilon)$ for parallel deactivation with $n = 1$. These results are valid during the entire period of operation from initial startup to final loss of activity. We again draw attention to the fact that at no stage has a quasisteady-state assumption been invoked; if it had been, the $0(\epsilon)$ corrections could have not been obtained. Higher-order corrections can be found in an obvious way, although the algebraic tedium becomes prohibitive.

The key results for other mechanisms of deactivation given by

TABLE 1. THE FUNCTIONS $F(A)$ USED IN EQ. 35b CORRESPONDING TO VARIOUS DEACTIVATION MECHANISMS.

n	$F(A)$
	Parallel Poisoning
1	$-2A + \beta \ln A - (1 + \beta + \gamma)\ln(1 - A) - \beta/A$
2	$(\beta + \gamma - 1)\ln A - (1 + \beta + \gamma)\ln(1 - A) - \beta/A - \beta/2A^2$
3	$(1 + \beta + \gamma)\ln[A/(1 - A)] - (\gamma + \beta - 1)/A - \beta/2A^2 - \beta/3A^3$
	Series Poisoning
1	$2A + \beta \ln A + (3 - \beta + \gamma)\ln(1-A) + (1 + \beta + \gamma)/(1 - A)$
2	$2\beta \ln A + 2(1 - \beta)\ln(1 - A) - \beta/A + (1 + \beta + \gamma)/(1 - A)$
3	$(\gamma + 3\beta - 1)\ln[A/(1 - A)] - 2\beta/A - \beta/2A^2 + (1 + \beta + \gamma)/(1 - A)$

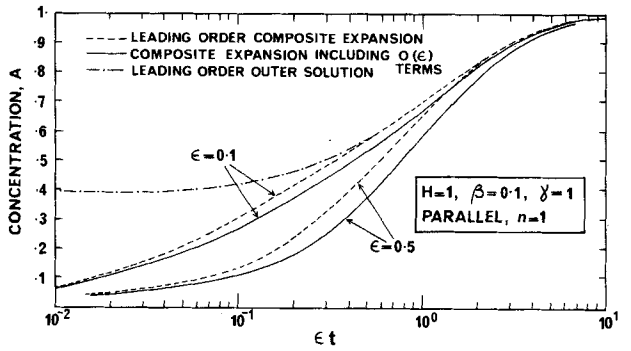

Figure 10. Reactor behavior for moderately fast catalyst deactivation.

Eqs. 3c and 3d with $n = \{1, 2, 3\}$ can be had by considering only the outer solutions. For a unique quasisteady state, we find that

$$a = H(1 - A)(A^2 + \gamma A + \beta)/A, \tag{35a}$$

with A given implicitly by

$$F(A) - F(A^+) = \tilde{t} \tag{35b}$$

where $F(A)$ is given in Table 1 for the various mechanisms and A^+ is the only real root of Eq. 12. These results are only valid to leading order, but $0(\epsilon)$ corrections can be obtained in the manner outlined.

DISCUSSION

The effects of the space velocity, H, the kinetic parameters, β and γ, and the mechanism of deactivation (specified by n) on the long-time dependence of reactant concentration and catalyst activity are shown in Figures 2 to 5, respectively, for parallel poisoning. Corresponding results for series poisoning are presented in Figures 6 to 9. The abscissa in these plots is the time made nondimensional on the time-scale for *deactivation*. All results shown in these figures are based on matched inner and outer solutions at leading order. It can be generally observed that on startup, the reactant concentration rapidly rises to a level which is essentially independent from ϵ. The time for approach to this level is very short in terms of $\tilde{t}$.

Ultimately, at times $\tilde{t} = 0(1)$, a significant amount of deactivation has occurred and simultaneously the bulk concentration of reactant rises. It is noteworthy that for small values of ϵ, the catalyst activity at any time $\tilde{t}$ is independent from ϵ; most of the ϵ-dependence is already contained in the slow-time variable. The case of moderately large ϵ (but still significantly less than unity) will be discussed separately.

The effect of increasing space velocity under parallel poisoning (Figure 2) is to maintain the reactor contents at higher levels of the reactant. Since this is the poison, loss of activity occurs sooner. Figure 6 shows that the reverse is true for series deactivation; a high space velocity keeps the product (poison) concentration at low levels and deactivation is correspondingly delayed. In this case, loss of activity implies still lower product concentrations, with the result that the catalyst decays as a much slower rate for series than for parallel poisoning. For the parameter values given in Figures 2 and 6, for example, it takes a time of roughly 100 units to completely destroy a catalyst by product poisoning as against only 10 units for deactivation by the reactant.

The reaction rate expressions we have been dealing with are in terms of the concentrations of reactant and product in the *bulk fluid*. If we think of the Langmuir-Hinshelwood rate form as containing information about the adsorption isotherm, it is apparent that the term $A/(A^2 + \gamma A + \beta)$ is just the concentration of A in the *sorbed* phase. Increasing values of β and γ imply less favorable isotherms. Thus, in the case of parallel poisoning (Figures 3 and 4) larger β and γ *delay* deactivation because there is *less* sorbed poison (reactant), despite the fact that its bulk fluid level has risen. For the series poisoning case this same less favorable isotherm results in lower product concentrations and a similar delay in loss of activity. The effect of β and γ is somewhat stronger in the case of series deactivation than for reactant poisoning. For the simple mechanisms shown in the figures, this can be readily explained by the modifying influence of the multiplicative factor A, present in the parallel poisoning case, which is absent for series deactivation.

Figures 5 and 9 show the effect of varying degrees of complexity of parallel and series poisoning mechanisms, respectively. The important observation is that final loss of activity occurs at a time which is essentially independent from the details of the mechanism for series deactivation; whereas, for parallel poisoning, this time is a strong function of mechanism. For reasons already given, the ultimate failure of the catalyst also occurs much later for a series mechanism than for a parallel one.

A typical example of reactor behavior for systems in which the catalyst is inactivated at a rate which is a significant proportion of the main reaction rate is shown in Figure 10. It should be noted that the time axis is ϵt and larger values of ϵ give *earlier* loss in performance. As ϵ increases, it becomes evident that the assumption of an initial steady state (shown by the curve — · —) would be quite inappropriate. For sufficiently large ϵ, no steady state is ever reached. It can be seen further that neglecting the $0(\epsilon)$ terms leads to a conservative estimate of reactor conversion.

NOTATION

a = catalyst activity
a_i = defined in Eq. 7b
$\tilde{a}_i$ = defined in Eq. 27b
A = nondimensional reactant concentration (C_A/C_{Ao})
A^+ = only real root of Eq. 12
A_i = defined in Eq. 7a
$\tilde{A}_i$ = defined in Eq. 27a
B = nondimensional product concentration (C_B/C_{Ao})
c = constant in Eq. 20
C_A = dimensional reactant bulk concentration
C_{Ao} = dimensional reactant feed concentration
C_B = dimensional product bulk concentration
F = feed rate to reactor
$F(A)$ = function given in Table 1
$g(A, a)$ = function defined in Eq. 6a
H = nondimensional space velocity (FC_{Ao}^2/kV)
k = rate constant for main reaction
k_d = deactivation rate constant
K = constant in Eqs. 1 and 2
K_s = constant in Eqs. 1 and 2
n = exponent in Eqs. 2a and 2b
R = reaction rate or deactivation rate
t = nondimensional time (kt'/C_{Ao}^2)
t' = dimensional time
$\tilde{t}$ = long-time variable (ϵt)
V = reactor volume

Greek Letters

α_0 = defined in Eq. 13
α_1 = defined in Eq. 14
β = kinetic constant (K/C_{Ao}^2)
γ = kinetic constant (K/C_{Ao})
ϵ = nondimensional deactivation rate constant $(k_d C_{Ao}^n/k)$

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Manuscript received October 1, 1979; revision received April 28, and accepted May 1, 1980.

Determination of Interaction Second Virial Coefficients; He-CO_2 System

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We have obtained values for interaction second virial coefficients of the helium-carbon dioxide system in the range $230 \leq T/K \leq 300$. Our experimental technique is essentially the Burnett mixing method described by Hall and Eubank (1973, 1974). We have modified the analysis to account for higher-order effects and to detect significant systematic errors. We also report virial coefficients for the pure components: helium in the range $100 \leq T/K \leq 300$ and carbon dioxide at 300 K.

SCOPE

Interaction second virial coefficients, B_{ij}, contain information about mixtures which is essential for both theoretical and practical applications. Within statistical mechanics, B_{ij} reflects molecular interactions between unlike molecules and provides insight for theoretical mixture models. On the practical side, B_{ij} is necessary for thermodynamic calculations at low pressure when the application dictates use of the (truncated) virial equation.

The normal method for obtaining B_{ij} is reduction of mixture second virial coefficients, B_m. This is the most obvious but least accurate method. The sources of inaccuracies are experimental errors in the B_{ii} and B_m as well as any errors in composition determination. Edwards and Roseveare (1942) appear to have pioneered this method. Knobler et al. (1959) developed a significantly better technique: the differential pressure method. Their technique eliminated composition and B_m as experimental parameters but retained the B_{ii} explicitly and ignored corrections for the effects of higher-order terms in the virial equation. Although other procedures are possible for obtaining B_{ij} (reduction of vapor-liquid equilibrium data; analysis of chromatographic data), these two techniques have produced the bulk of the B_{ij} data and essentially all of the precise values.

In this project, we extend a technique for isothermal determination of B_{ij} originally proposed by Hall and Eubank (1973). Their method was "direct" in that the pure-component virial coefficients, B_{ii}, did not appear explicitly. Because B_{ii} was a principal source of error in previous methods for obtaining B_{ij}, the direct method appeared to have potentially greater accuracy. We chose to evaluate the method with mixtures of simple molecules, because they are more tractable for theoretical studies. This particular test involves helium (a spherical, nonpolar molecule) and carbon dioxide (a linear, quadrupolar molecule).

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0001-1541-80-3374-0954-$01.25.