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ON THE POISONING OF CATALYSTS

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That surface non-uniformity plays an important rôle in the poisoning of contacts had been known for more than 20 years, yet not until recently was a comprehensive theory of poisoning built up on this basis. This is easily done for a highly non-uniform surface by employing the leading (control) band method (1). The notion of contact poisoning is generally understood to embrace all cases where the decrease in catalytic activity is due to sorption of foreign matter. The mechanism of this phenomenon vary widely. In fact, the activity of the catalyst can diminish: 1) as a result of unfavourable changes in the adsorption and kinetic surface constant caused by the penetration of the poison into the surface layer of the catalyst lattice, or 2) the reason may lie in the poison being adsorbed by some isolated microscopic portions of the surface, which are thereby prevented to influence the reaction.

Such poisoning by microblocking of the surface is essentially different from 3) macroscopic blocking which is due either to the filling of the pores and capillaries by easily condensing liquids, or to the formation of a polymolecular barrier from the solid reaction products, which hampers access to the active surface.

The classical theory of the poisoning of catalysts with a uniform surface put forward by Langmuir is applicable only to the case of microblocking on uniform surfaces.

Below is developed a theory of microblocking for highly non-uniform surfaces (see also (2)).

Catalyst poisoning at constant temperature is conveniently characterized by the dimensionless quantity λ, which is the ratio of the activity of the poisoned catalyst A_Γ to its activity in the absence of contact poisons A_0:

$$\lambda = A_\Gamma / A_0 \tag{1}$$

λ is directly dependent upon the amount Γ of poison adsorbed by the contact or upon a proportional quantity Φ_Γ which is the portion of the total surface area occupied by the poison:

$$\Phi_\Gamma = \Gamma / \Gamma_\infty \tag{1a}$$

where Γ is the amount of adsorbed poison, and Γ_∞ is the value of Γ when the entire surface is covered by one layer of the poison. The function describing the dependence of λ upon Φ_Γ

$$\lambda = \lambda(\Phi_\Gamma) \tag{2}$$

may be called isotherm of poisoning with reference to the ratio of the poisoned area to total area or, simply, Φ_Γ [Γ]-isotherm. This function is applicable to all types of poisoning, reversible as well as irreversible. In the latter case use is frequently made of the dependence of the activity upon the concentration, or upon the partial pressure of the contact poison in the volume.

Of such concentration isotherms, the following will be used hereafter:

$$\lambda = \lambda(\pi) \tag{3}$$

where π is the partial pressure of the contact poison in the gas volume.

A. Linear Isotherms. $\Phi_\Gamma[\Gamma]$-isotherms and concentration isotherms are often found to have a linear course (see[3] and the earlier researches by Maxted). Usually they are regarded as a characteristic indication of surface uniformity, since, according to Langmuir's theory, only one equation is possible for these isotherms:

$$\lambda = \Psi_\Gamma \tag{4}$$

where Ψ_Γ is the poison free portion of the total surface area

$$\Psi_\Gamma = 1 - \Phi_\Gamma \tag{4a}$$

But in actual fact the surface must not necessarily be uniform in this case. Equation (4) will hold, indeed, for any degree of non-uniformity of the surface (Fig. 1) and for any position of the control band, provided that the arrangement of the molecules of the poison over the surface is in no way connected with its activity. There will be no such connexion, for instance, if the Q of the adsorption of the poison is not correlated with the E of the catalytic reaction, or again if there is no correlation between the E of the poison and the E of the catalytic reaction.

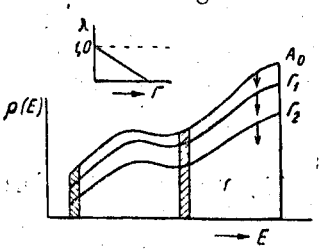

Fig. 1.

In the first case the final picture is represented in Fig. 1; in the second case a redistribution of the poison over the surface is possible with the result that the activity of the latter may either be reduced considerably (Fig. 2, *a*) or not reduced at all (Fig. 2, *b*).

Taking into account the possibility of a linear dependence between the residual activity and the quantity of adsorbed poison, owing to the adsorption of successive layers of the poison by porous and powder contacts, as was shown by Russell and Ghering (4), and the possibility of replacing most of the complex functions by linear ones when the interval in which the argument varies is limited, one has to admit that the linearity of $\Phi_\Gamma[\Gamma]$-isotherms is of itself no evidence of the uniformity or non-uniformity of the surface.

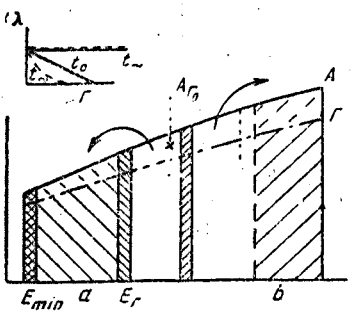

Fig. 2

B. Poisoned Area Isotherms when the Most Active Spots are Mainly Affected by the Poison. The situation is entirely different in the case of a preferential poisoning of the more active sites. This may occur because the value of the heat of adsorption is higher at these spots (E and Q vary in the opposite sense), or the adsorption of the poison is more rapid (both E's vary in the same sense), or, finally, because of the fixation of the poison at the spot where it is formed during auto-poisoning. In all these cases a simple relation should exist between the quantity of adsorbed poison and the change in the energy of activation of the catalytic process

$$\Phi_\Gamma \simeq \int_{E_{min}}^{E_\Gamma} \rho(E)\, dE \tag{5}$$

with the equation

$$\rho(E) \simeq \rho(E_\Gamma) = d\Phi_\Gamma / dE_\Gamma \tag{6}$$

that follows from it (Fig. 3).

To derive the Φ_Γ [Γ]-isotherm, we make use of the equation for the fractional activity λ' (5):

$$\lambda = \frac{A_\Gamma}{A_0} = \frac{M_\Gamma \exp(-E_\Gamma/R\Gamma)}{M_1 \exp(-E_1/R\Gamma)} \approx e^{-\gamma E_\Gamma/RT} \tag{1b}$$

By making use of equation (5) it is not difficult to express E_Γ through E_1 and Φ_Γ, which immediately gives the result sought for:

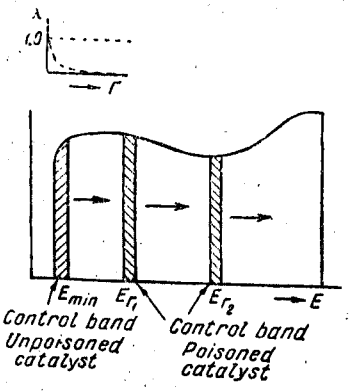

Fig. 3.

a) for exponential distribution:

$$E_\Gamma \simeq \frac{1}{\alpha} \ln \frac{\alpha \Phi_\Gamma}{H} \text{ when } \alpha > 0$$

b) for power distribution:

$$E_\Gamma \simeq \beta \Phi_\Gamma^{1/(n+1)}, \quad \beta = \sqrt[n+1]{\frac{n+1}{H}}$$

c) for uniform distribution:

$$E_\Gamma = E_1 + \frac{\Phi_\Gamma}{H}$$

(7)

This leads to the following equations for λ (in the case of exponential distribution allowance is made for the change of M_Γ with increasing E):

a) for exponential distribution:

$$\lambda \simeq \lambda_0 / \Phi_\Gamma^m, \quad m = \frac{1 - \alpha RT}{\alpha RT}$$

b) for power distribution:

$$\lambda \simeq \lambda_0' \exp[-\alpha \Phi_\Gamma^{1/(n+1)}], \quad \alpha = \beta / RT$$

c) for uniform distribution:

$$\lambda = \lambda_0'' \exp[-\alpha \Phi_\Gamma], \quad \alpha - 1/HRT$$

(8)

Of these equations c) was discovered by us (6) during a study of the catalytic oxidation of carbon dioxide (CO_2 *in statu nascendi* is the contact poison); equation a) seems to have been obtained by Kubokawa (7). Equation b) appears for the first time. These equations, as well as equation (6), make it possible to determine the form of the function $\rho(E)$. Taking (1b) into account, we may write equation (6) in a somewhat different form:

$$\gamma \rho(E) \simeq - \frac{d\Phi_\Gamma}{RT d \ln \lambda} \tag{9}$$

It may be remarked that when the poisoning is very slight, all of the isotherms conform to the linear equation $\lambda = \lambda_0(1 - c\Gamma)$. It is only when the degree of poisoning attains medium or high values that the differences between the isotherms, accounted for by differences in the type of the function $\rho(E)$, become significant. To study the nature of non-uniformity from the poisoning the interval of variation of λ must be sufficiently wide, in any case not narrower than two orders.

The equations that have been derived are applicable to many systems with a stationary control band lying at the distribution boundary. Here poisoning fails to alter the kinetics of the process. The same van't Hoff equations are retained in one limiting case, and the zero orders in the other. For systems with a mobile (suspended) band there is little probability that the blocking will start from the sites controlling the process. In this case poisoning should be better described by Φ_Γ[Γ]-isotherms obtained for a poisoning beginning from slightly active sites.

C. Poisoning Beginning with Active Sites. In this case for systems in which the control band is fixed at the edge blocking by the contact, poison begins to involve all but the controlling sites which are the last to be blocked (Fig. 4). Therefore up to a certain value of Φ_r close to unity the surface shows no sign of poisoning at all, and not until the limits of $\Delta\Phi_r$ are such as to change E by as much as $(1\div2)\ RT$, does a complete poisoning take place (Fig. 4). In other words, a critical stage in the process of surface poisoning should exist, at which the activity changes abruptly from A_0 to 0.

In the case of a mobile band the picture is more complicated. If covering by the poison proceeds from small values of E, then up to a certain value of Φ_r we have:

$$A=\text{const}=A_0 \qquad (10)$$

and, from this value of Φ_r onwards, a rapid decline takes place. However, inasmuch as the band is not pressed to the edge, it begins to move towards large values of E, and poisoning obeys the law characteristic for the blocking of the most active sites, with different curves for different ρ. When the band reaches the edge, the activity falls rapidly to zero. Characteristic is the sharp change in the kinetics (Fig. 5) upon transition through Φ_r critical.

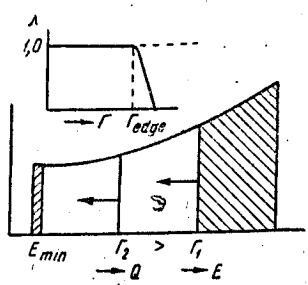

Fig. 4

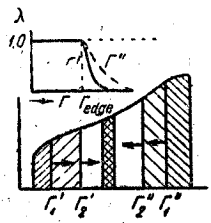

Fig. 5

Within definite limits the change in the concentration ceases to affect the rate. The fractional orders described in our preceding paper [5] are replaced by zero orders. However, upon a sufficiently marked lowering of the partial pressures, such as tears the control band away from the blocked zone, the fractional orders should again be re-established.

The picture of the poisoning process is different when the sites with maximum values of E are the first to be covered by the poison. The instant the poison reaches the leading band, the latter begins to move. At the same time the fractional orders are transformed not into zero orders, but into simple van't Hoff orders.

The true energy of activation does not increase with poisoning but rather declines. But since to the left of the leading band covering of the surface by the exothermic complex also decreases exponentially, and for Q faster than for E, the temperature coefficient at a given Φ_r is determined not by E but by $E-Q$, and the apparent energy of observation $E_{obs}=E-Q$ increases as the poisoning goes on.

Thus, during poisoning the equality $E_r=E$ is possible; here E is the energy of activation at the given sites, and also $E_r=E-Q$. The activation energies determined from the temperature coefficient during constant fractional covering always increase with the poisonig.

Such are the basic peculiarities of the isotherms of poisoning with respect to covering on broad non-uniform surfaces. Their study in a large interval of activities may give highly valuable information both on the statistics of the catalytic process and on the mechanism of the latter.

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