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Recovery of Electron-Irradiated Copper. II. Interstitial Migration

J. W. Corbett, R. B. Smith,* and R. M. Walker
General Electric Research Laboratory, Schenectady, New York

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Several experiments are presented dealing with substages I_D and I_E of Stage I (14°K–65°K) recovery in electron-irradiated pure copper. It is concluded that both I_D and I_E represent the recovery due to the free diffusion of a defect, presumably an interstitial atom, with an activation energy for motion $E_m=0.12\pm0.005$ ev. I_D is the result of correlated recovery (the interstitial returns to the vacancy from which it came) while I_E is the result of uncorrelated recovery (the interstitial travels to a distant sink). The migrating interstitial is shown to interact with vacancies, traps, and other interstitials leading respectively to annihilation, trapping, and cluster formation. The interaction between two interstitials leading to cluster formation is found to be comparable with the annihilation interaction between an interstitial and a vacancy. Clusters containing more than two interstitials are also formed. Previous work in copper is discussed, and it is concluded that this previous work is consistent with the above description of Stage I recovery. The relationship of the present work to the general problem of assigning defect processes to the higher temperature recovery states in copper is also discussed.

INTRODUCTION

THE general introduction to the subject matter and experimental apparatus has been given in the preceding paper.[1] In that paper it was shown that the recovery of electron-irradiated copper between 14° and 65°K consisted of five distinct substages. Only the first three (I_A, I_B, and I_C) were discussed. In this paper we describe several experiments which deal with the remaining two recovery sub-stages (I_D and I_E). Consideration of these experiments leads to the following model for the recovery processes:

1. Low-temperature radiation damage produces interstitials and vacancies, in pairs.
2. Upon heating, recovery proceeds by the following stages: I_A, I_B, I_C: three stages of close-pair recombination, each with a characteristic activation energy, each corresponding to a different close-pair configuration. I_D, I_E: two stages of recombination associated with the free diffusion of interstitials, one the result of correlated recovery (the interstitial returns to the vacancy from which it came), and the other the result of uncorrelated recovery (the interstitial travels to a distant sink), both with the same activation energy.
3. Free diffusion of an interstitial ceases when it meets a vacancy (annihilation), when it meets an impurity (trapping), or when it meets another interstitial (cluster formation). In pure copper, as many as half of the interstitials participating in stage I_E form stable clusters that persist to at least 80°K.

The evidence for this description of the recovery processes is as follows:

1. The evidence for close-pair recombination associated with stages I_A, I_B, I_C has been given in the preceding paper.
2. The evidence for free diffusion in stage I_E is as follows:

a. Concentration-dependence. Stage I_E proceeds more rapidly when the damage is greater, as there are more sinks for interstitials. The shift agrees with that predicted for a bimolecular process using the activation energy determined from isothermal experiments.

b. Radiation doping. Prior radiation damage at 80°K (producing a large density of radiation sinks) enhances the rate of stage I_E recovery from subsequent low-temperature damage.

3. The evidence for free diffusion in stage I_D is as follows:

a. Stage I_D has the same activation energy as stage I_E, which has been shown to be free diffusion.

b. A diffusion analysis of the recombination of interstitials and vacancies, along the lines proposed by Waite,[2] shows that a correlated recombination stage and an uncorrelated recombination stage are to be expected. The analysis gives good agreement with the I_D and I_E recovery stages (in spite of the fact that it considers only annihilation and neglects cluster formation).

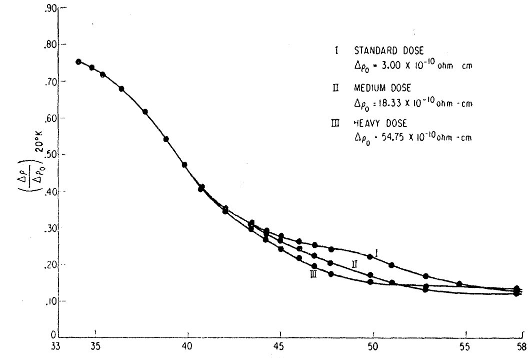

FIG. 1. Concentration dependence of the I_D and I_E isochronal recovery. The standard dose (*I*) corresponds to ~1 ppm initial defect concentration. The subscript 20°K refers to the irradiation temperature.

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* Now at Hanford Laboratory Operations, Hanford Atomic Products Operations, Richland, Washington.

[1] Corbett, Smith, and Walker, preceding paper [Phys. Rev. **114**, 1452 (1959)].

[2] T. R. Waite, Phys. Rev. **107**, 463, 471 (1957).

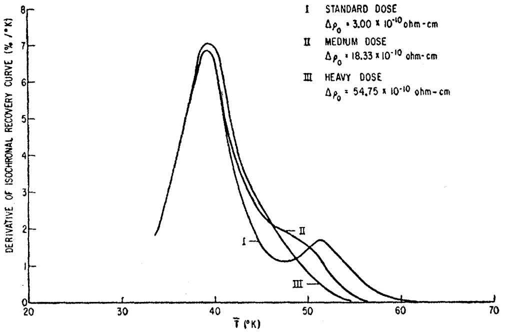

FIG. 2. Numerical derivative of the concentration dependence I_D and I_E isochronal recovery curves shown in Fig. 1.

4. The evidence for clustering of interstitials is as follows:

a. Persistent damage. Irrespective of the radiation dose given to clean copper at low temperatures, a constant fraction (about half) of the damage remaining after stage I_D persists through stage I_E. Thus when free diffusion is over, some interstitials and vacancies remain uncombined.

b. Radiation doping. Not only does prior doping at 80°K enhance the *rate* of stage I_E recovery from subsequent low-temperature damage, but it markedly enhances the *amount* of recovery. This evidence suggests that newly introduced interstitials are largely eliminated through recombination with residual vacancies in radiation-doped copper, in contrast to their behavior in pure copper where about half of them form clusters and remain unrecombined.

In the following section we present a qualitative but fairly detailed discussion of the experimental evidence which leads to the previously outlined recovery model. In the section which follows this qualitative discussion, the data are analyzed in more detail in terms of a specific analysis which incorporates the general features of the recovery model. In the final section of this paper the relationship of these results to previous work and to the general problem of assigning defect processes to higher temperature recovery states is discussed.

GENERAL FEATURES OF THE RECOVERY PROCESSES

In this section we present a qualitative discussion of some of the experimental results. We wish to show that these experiments lead to some general conclusions concerning the nature of the recovery processes.

Demonstration of Free Migration of Defects

As indicated in the previous paper, the fractional rate of recovery for local recombination processes is independent of total concentration. This is not in general true of recovery processes which involve free migration of defects. If the interstitial is performing a random walk through the lattice, the probability that it encounters a defect with which it can interact, e.g., a vacancy, will depend on the concentration of these defects. Hence, the fractional rate of recovery will increase with increasing defect concentration. In the case where the initial distribution of defects is random and only annihilation of wandering interstitials at fixed vacancy sites is occurring, the process is described by the simple bimolecular equation

$$dC/dt = -C^2 A e^{-E_m/kT}, \tag{1}$$

where C is the instantaneous concentration of interstitials, A is the frequency factor, and E_m is the activation energy of motion.

Figure 1 shows the results of several different isochronal recovery experiments on I_D and I_E recovery with different initial defect concentrations. Figure 2 gives the same data plotted as a numerical derivative curve. In Fig. 3 the isothermal recovery at 52.7°K (I_E) is shown for initial concentrations differing by a factor of six. It can be seen from both the isochronal and isothermal experiments that substage I_E is markedly affected by the change in initial defect concentration. In the isochronal experiments, the maximum rate of recovery is shifted to lower temperatures. The measured shift agrees with that predicted for a bimolecular process using the activation energy determined from isothermal experiments. The fact that the production curve is accurately linear at the bombarding temperature up to the highest concentration of defects suggests that the same type of damage is introduced initially, independent of concentration. The observed concentration effect, therefore, is due to a difference in the recovery processes during annealing and is not due to differences in the character of the induced damage at the bombarding temperature. This conclusion is substantiated by the fact that the recovery curve is quite independent of concentration for the lower temperature substages. The very low concentration of defects makes it difficult to ascribe the observed effect to any static long range interaction be-

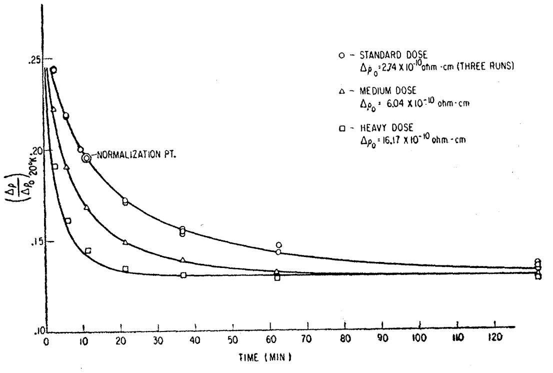

FIG. 3. Concentration dependence of the 52.7°K (I_E) isothermal recovery. The solid lines are dimer formation theory, normalized at one point as discussed later in the text. The subscript 20°K refers to the irradiation temperature.

tween defects. The simplest interpretation of the observed concentration dependence is to ascribe it to the free migration of interstitial atoms.

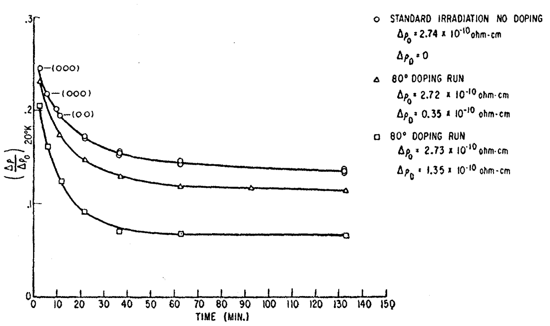

FIG. 4. The effect of 80°K radiation doping on the 52.7°K (I_E) isothermal recovery. The subscript 20°K refers to the irradiation temperature for the isothermal experiments.

A perhaps more striking demonstration of the existence of a migrating defect is the existence of the "radiation doping effect." We find that the isothermal recovery in substage I_E of a standard irradiation is markedly affected by radiation defects which have been introduced by prior bombardment at 80°K. Consider the results shown in Fig. 4. The top curve shows the isothermal recovery at 52.7° (I_E) for three different runs with identical initial defect concentrations of $\sim 1\times10^{-6}$. The sample was annealed at $\sim$410°K between each of these runs. The middle curve shows what happens when the fully annealed sample is first bombarded at 80°K, allowed to remain at this temperature until no recovery is occurring (this happens almost immediately), and is then subjected to the same low-temperature bombardment and annealing as in the three runs of the top curve. The presence of the defects introduced by the prior 80°K bombardment enhances both the rate and final amounts of recovery for the standard irradiation. The bottom curve in Fig. 4 shows the same type of experiment with a larger amount of prior irradiation at 80°K. That this effect is due to a difference in the recovery processes after warmup and is not due to a difference in the nature of the initial damage introduced at the bombarding temperature can be seen in two ways. In the first place, the production rate at the bombarding temperature is the same whether or not the sample has been previously doped with radiation defects. Secondly, the low-temperature recovery states are relatively unaffected by the radiation doping and reproduce the standard recovery curves. This is shown in Fig. 5. The triangle points are for the 1 ppm standard isothermal at 40.7°K (I_D) taken with no prior irradiation. The circle points show an identical isothermal with the exception that the sample in this case had received a prior irradiation at 80°K to a defect concentration of $\sim 7\times10^{-7}$. There is only a slight difference in the two curves. However, when the experiments are carried through the I_E recovery, a marked difference shows up. As can be seen in Fig. 5, referring to the final recovery values after annealing at 80°K, the radiation doping was fully effective in enhancing the I_E recovery even though it left the I_D recovery virtually unaffected.

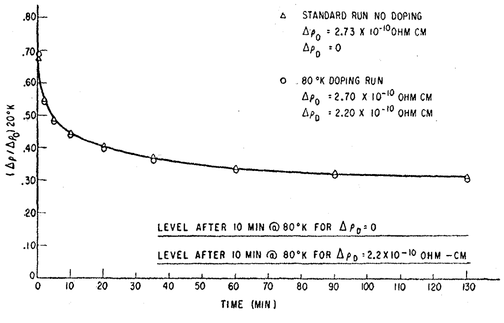

FIG. 5. The effect of 80°K radiation doping on the 40.7°K (I_D) isothermal recovery. The horizontal lines indicate the extent of the recovery after the annealing 10 minutes at 80°K of the two experiments shown in the figure. The subscript 20°K refers to the irradiation temperature. Rise-time correction is not included.

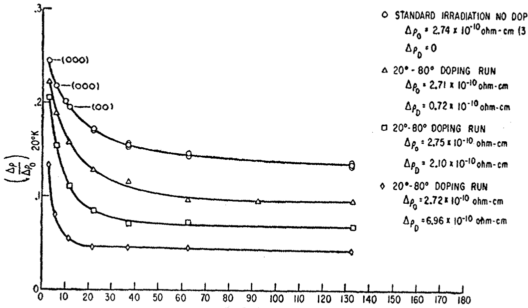

FIG. 6. The effect of 20°K irradiation–80°K annealing doping on the 52.7°K (I_E) isothermal recovery. The subscript 20°K refers to the irradiation temperature.

The simplest interpretation of this radiation-doping effect is that interstitial atoms can move long distances in substage I_E and annihilate at vacancy sites which have been introduced by the prior irradiation. If we make the assumption that a defect can affect at most 100 atoms, the existence of this effect at defect concentrations of $\sim 1\times10^{-6}$ in both the standard irradiation and the doping radiation implies a migrating defect which makes at least 10^4 jumps in order to recover.

The radiation doping can also be introduced by first irradiating at 20°K and then annealing at 80°K. The results of isothermal experiments at 52.7° (I_E) with various amounts of doping added in this manner are shown in Fig. 6. It is found in this case that a given amount of doping resistivity is not quite as effective in modifying the recovery of the standard irradiation as is the doping resistivity added by bombardment at 80°K. It is therefore clear that these two methods of introducing doping result in different microscopic configurations of defects. Clearly these experiments also have a great deal to say on the nature of the radiation-induced resistivity which does not recover at 80°K. Both types of radiation doping are considered later in more detail.

Interaction of the Moving Defects

The foregoing experiments indicate that in the substage I_E the predominant process is the free migration of an interstitial. In spite of this, complete recovery does not occur in stage I. Figure 7 shows the recovery above 80°K of resistivity which was added partly by bombardment directly at 80°K and partly by bombarding at 20°K and then annealing to 80°K. Only about 1.8% of the equivalent total resistivity added at 20°K recovers between 80°K and 250°K (stage II). Most of the remaining 10.2% of the initial resistivity increment recovers between 250°K and 350°K (stage III). This is similar to the observations of Meechan and Brinkman[3] on the recovery of samples bombarded at 80°K, with the difference that stage II is less pronounced and stage III more pronounced in the present experiments. The residual 0.1% which remains after annealing at ~410°K has no apparent effect on the recovery of a subsequent low-temperature irradiation.

The question arises as to what defects are responsible for the fraction which does not recover until stages II and III. At least three possibilities exist:

(a) Moving interstitials are trapped at other than radiation induced lattice defects. The resistivity left after stage I is then due to these trapped interstitials and a corresponding number of isolated vacancies.

(b) Moving interstitials interact with each other to form clusters. The residual resistivity is then composed of interstitial clusters and isolated vacancies.

(c) The radiation produces another lattice defect (perhaps a different type of interstitial) which does not interact with the migrating defect. The residual resistivity in this case is due then to this different species of lattice defect.

Of these three possibilities, only the second, which postulates interstitial clustering, is consistent with our experimental results on the zone-refined copper. We shall demonstrate that the first possibility mentioned—the trapping of interstitials by impurities—can occur, but is not important for the purest samples.

The evidence for interstitial clustering derives mainly from the observation that the total fractional recovery in stage I is very nearly independent of initial defect concentration. The data are shown in the top curve of Fig. 8 which plots the amount of the radiation-induced resistivity which is left after annealing at 80°K as a function of the initial resistivity added at 20°K. Although the *rate* of recovery is strongly dependent on concentration, the *final fractional recovery* is the same within ±0.5% for defect concentrations ranging from $<1\times10^{-6}$ to $\sim2\times10^{-5}$. We shall show explicitly later in the paper that this is the behavior one would expect if the defects were both annihilating and forming clusters.

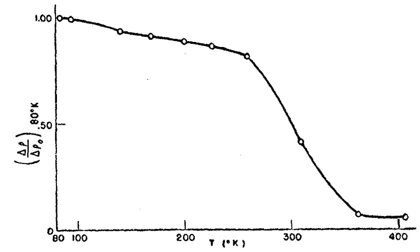

FIG. 7. Stage II and stage III recovery of defects introduced partly by irradiation at 80°K and partly by irradiating at 20°K. The recovery is normalized at the 80°K value.

Impurity trapping of interstitials could also lead to the observed constancy of the total fractional recovery but can be ruled out by the following argument. At a defect concentration of 1×10^{-6}, the radiation defects must be interacting frequently with each other and not exclusively with other lattice defects. This is shown both by the radiation-doping effect and by the marked concentration dependence of the recovery rate. We would therefore expect that when the radiation defect concentration was twenty times higher or $\sim2\times10^{-5}$, the effect of other than radiation-induced defects would be negligible. The fact that virtually the same fractional recovery is observed for this heavy concentration run shows that the fraction which remains cannot be due solely to interaction of the radiation defects with other lattice defects such as impurities.

The third alternative proposed to explain the resistivity which is left after stage I recovery was the possibility that two types of interstitials are formed in the initial damage. One of these was presumed to be mobile in stage I and the other immobile until a later recovery stage. This possibility would explain the observed constant fractional recovery since all the mobile interstitials would annihilate leaving behind a constant fraction of immobile interstitials and their vacancies.

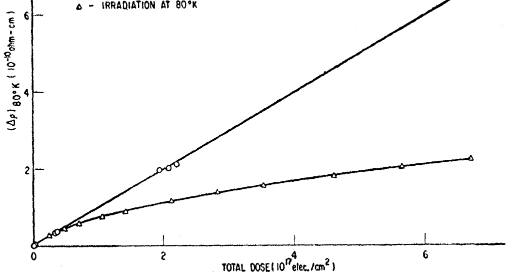

FIG. 8. Comparison of the resistivity change obtained by bombarding directly at 80°K with that obtained by irradiating at 20°K and then annealing at 80°K. Each point in this latter curve corresponds to an independent experiment.

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[3] C. J. Meechan and J. A. Brinkman, Phys. Rev. **103**, 1193 (1956).

However, consideration of another experiment shows that this cannot be the case. Also included in Fig. 8 is the result of an irradiation on the same high-purity copper sample, carried out directly at 80°K. The resistivity increment corresponding to a given electron dose is *considerably less* in this case than the resistivity that remains when the sample is bombarded at 20°K to the same total dose and then annealed at 80°K. The two-interstitial model would predict the same straight line for the two experiments. If anything, we would expect the curve for the direct bombardment at 80°K to lie *higher* than the other curve since in this case the probability of impurity trapping is enhanced by the low instantaneous concentration of interstitials.

The results shown in Fig. 8 can also be used independently to show the importance of cluster formation in the case of the standard irradiations performed at 20° and subsequently annealed at 80°K. The low instantaneous concentration of interstitials in the direct 80°K irradiation would inhibit cluster formation and favor the trapping of interstitials by impurities. We would therefore expect that if impurity trapping were dominant the resistivities for this irradiation would be the same as or higher than in the case of a 20°K irradiation with subsequent anneal at 80°K. Since the opposite is true, we may conclude that impurity trapping is not dominant for the standard 20°–80°K experiment. Also, since the relative concentration of vacancies to impurities is higher in this latter case, the resulting higher resistivities show the importance of interstitial cluster formation. The nonlinear shape of the 80°K exposure curve is caused by the increasing probability of interstitial annihilation at the ever increasing number of vacancies left behind by trapped interstitials.

Since the cluster hypothesis satisfactorily explains the above experiments while the alternative hypotheses do not, we conclude that cluster formation is an important process.

We have also performed an experiment on the stage I recovery of a 1×10^{-6} initial defect concentration irradiation on a sample of American Smelting and Refining copper which has a nominal impurity content of 1×10^{-5}. In this case essentially none of the I_E recovery took place. The radiation-induced defects were apparently retained in the sample by being trapped by the impurities. This is in agreement with the observations of Blewitt, Coltman, Klabunde, and Noggle,[4] who found that stage I recovery could be completely suppressed, by the addition of suitable chemical impurities.

Relationship of I_D to I_E

Although substage I_D is apparently only slightly affected by the changing concentration, it is not a simple monomolecular process. A cursory inspection of the isothermal annealing data shown in Fig. 9 shows that in I_D these isothermals are not simple exponentials in time. Using these isothermals we will show that I_D and I_E recovery are characterized by *one activation energy*. Since I_D and I_E recovery spans a wide temperature range, the recovery kinetics must be such that some recovery can take place after a very few interstitial jumps, and yet some recovery does not take place until $\sim10^5$ interstitial jumps. Substages I_D and I_E can be accounted for in the following way.[5] Initially the defects are produced as interstitial-vacancy pairs with a range of (i–v) separations. Those pairs within some critical distance interact sufficiently strongly to make recombination almost certain and are defined as "close pairs." These "close pairs" give rise to substages I_A, I_B, and I_C. Those pairs with separations larger than the critical distance interact sufficiently weakly so that the interstitial is essentially equally likely to jump in any direction as it begins to move. However, because of its proximity to its own vacancy it will, in a random walk process, much more frequently recombine with its own vacancy than with other vacancies. Fletcher and Brown,[6] and more recently Waite,[2] have considered this problem of diffusion-limited recovery in detail. Waite showed that as time goes on there is a transition from the initially correlated recovery which is independent of total concentration, to a random recovery describable by Eq. (1). In the next section we shall show that detailed application of Waite's analysis gives reasonable agreement with the I_D and I_E recovery data. Stages I_D and I_E are therefore interpreted as representing the same fundamental process—free interstitial migration—with the separation between the substages arising in the transition from the correlated to the random recovery of the interstitials.

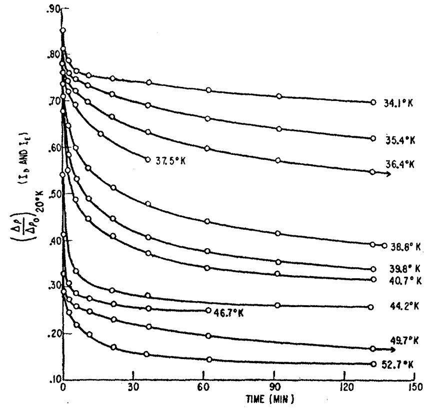

FIG. 9. Independent I_D and I_E isothermal recovery curves. The arrows indicate that some of the recovery curves were measured to times longer than could be conveniently shown. The subscript 20°K refers to the irradiation temperature.

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[4] Blewitt, Coltman, Klabunde, and Noggle, J. Appl. Phys. **28**, 639 (1957).

[5] Prior to our completion of the activation energy analysis, Professor J. S. Koehler of the University of Illinois suggested explicitly this possible explanation of I_D and I_E recovery. Using the activation energy previously determined for the fourth peak observed in the Illinois deuteron work, he showed that the calculated number of jumps for each of these peaks was consistent with this hypothesis.

[6] R. C. Fletcher and W. L. Brown, Phys. Rev. **92**, 585 (1953).

DETAILED ANALYSIS OF THE RECOVERY PROCESSES

In this section the recovery processes are considered in more detail. We wish to show here that the model previously outlined is capable of quantitatively accounting for the data.

Transition from Correlated to Random Interactions

Waite[2] has treated the question of diffusion-limited recovery in detail. We shall employ Waite's analysis to treat the recovery which remains after the close pair recovery has taken place. In this analysis the problem of the recombination of freely migrating interstitials with immobile vacancies, when the initial distribution of the interstitials with respect to the vacancies is not random, is treated as a continuum diffusion problem with the vacancies considered as spherical sinks with a fixed capture radius. In order to obtain an analytic expression for the recovery process, Waite assumed a specific expression for the initial distribution of interstitials with respect to vacancies. In particular, the explicit recovery relations were derived under the following assumptions:

(a) When a freely migrating interstitial comes within a critical radius, r_0, of a vacancy, annihilation takes place immediately. This radius will include the close pair sites.

(b) The probability that an interstitial-vacancy pair have an initial separation r is given by

$$P(r)dr = N \exp[-(r/\lambda r_0)^2] 4\pi r^2 dr, \tag{2}$$

where λ is the parameter which characterizes the radial distribution function, and N is a normalization constant defined by the condition

$$\int_{r_0}^{\infty} P(r)dr = 1. \tag{3}$$

The expression which describes the kinetics of the recovery is given by Waite[2,7]:

$$\begin{aligned}\frac{d\phi}{dZ} &= 2\pi r_0^3 C_0 (1-\phi)^2 \left[Z + \frac{2}{\pi^{\frac{1}{2}}}\right] \\ &\quad + 2\pi r_0^3 N \left\{ \frac{2\exp(-1/\lambda^2)}{\pi^{\frac{1}{2}}} \left[\frac{\lambda^2}{Z^2+\lambda^2}\right]^2 \right. \\ &\quad + \frac{Z}{2\lambda(Z^2+\lambda^2)^{\frac{1}{2}}} \exp\left(\frac{-1}{Z^2+\lambda^2}\right) \times \operatorname{erfc}\left(\frac{Z}{\lambda(Z^2+\lambda^2)^{\frac{1}{2}}}\right) \\ &\quad \left. \times \left[\frac{2\lambda^4}{(Z^2+\lambda^2)} - 4\left(\frac{\lambda^2}{Z^2+\lambda^2}\right)^2\right]\right\} \exp\left(\int_0^t f_{ii} dt\right), \end{aligned} \tag{4}$$

where C_0 is the initial concentration of defects, and C is the concentration at time t. The other symbols are defined below:

$$\phi = (C_0 - C)/C_0, \tag{5}$$

$$Z = \frac{2}{r_0}(Dt)^{\frac{1}{2}} = \frac{2D_0^{\frac{1}{2}}}{r_0}\left[t \exp\left(-\frac{E_m}{kT}\right)\right]^{\frac{1}{2}} \equiv \frac{2D_0^{\frac{1}{2}}(t')^{\frac{1}{2}}}{r_0}, \tag{6}$$

$$f_{ii} = -8\pi r_0^2 DC\left[\frac{1}{r_0} + \frac{1}{(\pi Dt)^{\frac{1}{2}}}\right]. \tag{7}$$

D is the diffusion coefficient for the interstitial, D_0 the frequency factor, and E_m the activation energy for motion. The exponential involving the integral of f_{ii} is equal to unity for the calculation of interest here.

Equation (4) has several important features. In the first place the recovery is a function only of the parameter Z which is proportional to an equivalent time t' defined in Eq. (6). This expresses the fact, recognized many times in the literature, that a given level of recovery corresponds to the same microscopic configuration of defects regardless of the time-temperature schedule used to achieve the recovery—provided only one activation energy is involved. The difference in arriving at the same recovery by annealing at two different temperatures is simply in the times involved, and these are related by the Boltzmann factor.

Physically the parameter Z is simply a measure of the number of jumps a defect makes. When the defects have made the same number of jumps, regardless of the rate of jumping, the same amount of recovery has taken place. A crucial test to see whether a set of isothermal recovery data represent a process with a single activation energy is to inquire whether the recovery can be expressed as a unique function of Z, or t'.

Figure 9 shows the results of a set of isothermal experiments which overlap the same recovery values. The sample was fully annealed at ~410°K between each of these runs, and the standard initial radiation dose (~1 ppm defect concentration) was given at the bombardment temperature of 20°K. Each curve, therefore, has the same initial configuration of defects, and the difference in the recovery curves arises only because of the different annealing temperatures. In Fig. 10 we plot the same recovery data as a function of t'. Also included are the results of the standard dose isochronal shown in Fig. 1. For the isochronal, t_n', is taken as

$$t_c \sum_{i=1}^{n} \exp\{-E_m/kT_i\}, \tag{8}$$

where t_c is the constant time 10 min. spent at each temperature. We find that an E_m value of 0.12 ev gives the best superposition of data from the different experiments onto a single universal curve. Assumed E_m values of 0.11 and 0.13 ev definitely do not superpose the data,

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[7] This expression differs slightly from Eq. (22) of Waite's paper but follows directly from his Eq. (14). [Note: Waite has used erf to indicate what is conventionally designated erfc (see Whittaker and Watson). We have used erfc and in the numerical calculations have appropriately conformed Waite's erf to the definition of the tabulated function.]

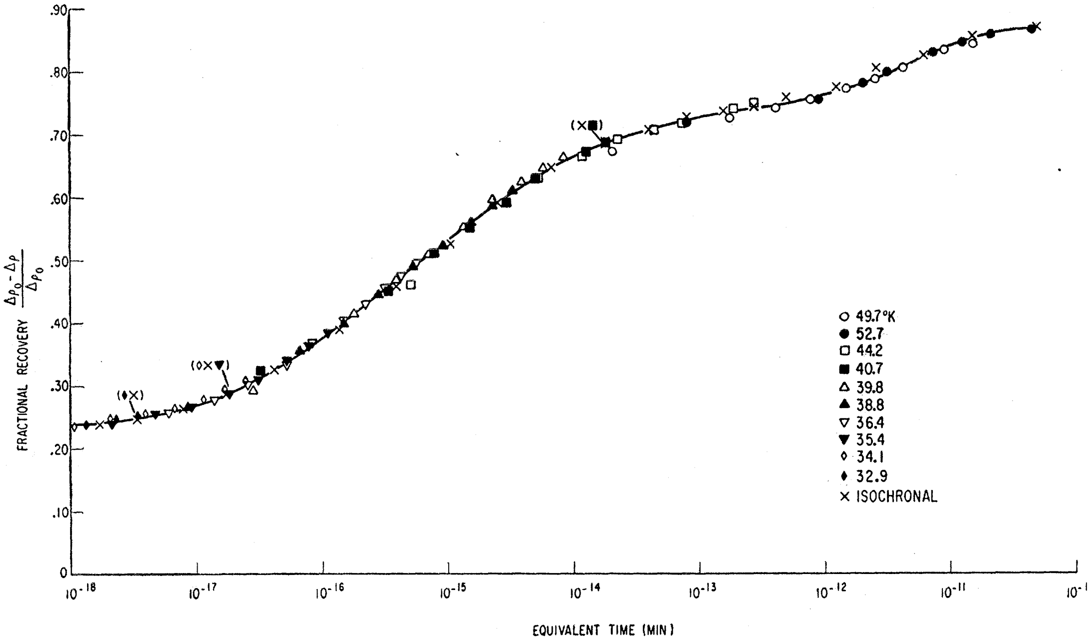

FIG. 10. I_D and I_E standard dose isothermal and isochronal recovery curves expressed as a function of equivalent time t' for $E_m=0.12$ ev. The equivalent time scale corresponds to measured minutes multiplied by the Boltzmann factor with the appropriate constants.

and we feel that E_m is probably determined in this manner to about 0.005 ev.

Another important feature of Eq. (4) is that it consists of two terms, one of which is not dependent on the initial concentration and the other which is. The concentration-independent term dominates at low Z (low number of jumps) while the concentration-dependent term becomes dominant at high Z (higher number of jumps). The concentration-independent term corresponds to the correlated recovery of an interstitial with its own vacancy. For high Z where the correlated recovery term is no longer important, the other term has the simple form of the bimolecular rate process of Eq. (1) and corresponds to the recovery of a random distribution of interstitials and vacancies. At sufficiently low concentrations the total recovery curve calculated from Eq. (4) is clearly divided into two regions, which correspond to the two types of recovery processes. The experimental recovery in Figs. 9 and 10 show this division into two distinct regions corresponding to the substages I_D and I_E.

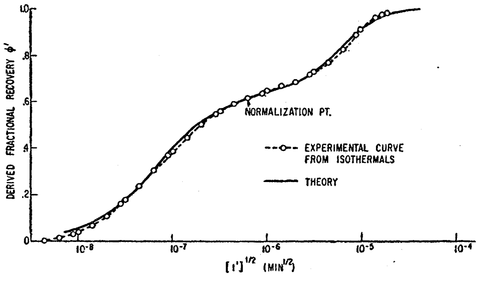

FIG. 11. The derived fractional recovery ϕ' corresponding to the isothermal recovery curve shown in Fig. 10, plotted as a function of the square root of the equivalent time t', and compared with theory.

Equation (4) takes into account only recombination processes and does not include the possibility of clustering of interstitials. The theory therefore predicts total recovery while the experiment indicates a constant fraction left after annealing. Rather than modify Eq. (4) to include clustering within the framework of Waite's theory, we have taken the following simplified approach. In region I_D we assume that the only process is the annihilation of interstitials at their own vacancies. In region I_E we assume that the interstitials have become completely randomized and undergo both annihilation and clustering. In region I_E, therefore, the measured recovery reflects a true decay of interstitials due to annihilation plus a buildup of interstitial clusters which are assumed to result in no net resistivity change. The measured recovery, ϕ, is corrected to a derived value, ϕ', which measures the true removal rate of free interstitials in both processes by assuming a linear relationship between the annihilation and clustering processes. The relationship between ϕ and ϕ' is given by the following expression:

$$\phi'=\phi_D+\phi_E/(1-A), \tag{9}$$

where the ϕ's are the measured fractional recoveries in the various substages (excluding the close pair peaks from consideration) and A is a normalization parameter which is adjusted to make $\phi'=1$ when ϕ has reached its saturation value.

We then assume that Waite's theory describes approximately the process of removal of interstitials in the two different ways and hence compare the ϕ' *vs* t' curves with the prediction of Eq. (4). This is a fairly crude method of comparing theory to experiment and cannot, of course, be taken as proof of clustering. The evidence for clustering has already been given. What we wish to show here is that the existence of the two substages I_D and I_E can be quite satisfactorily explained by the transition from correlated to random motion of a freely migrating interstitial.

The extent of agreement between theory and experiment is shown in Figs. 11 and 12. In Fig. 11, values of ϕ' calculated from values of the universal experimental curve of Fig. 10 are plotted against $(t')^{\frac{1}{2}}$ and compared with values of ϕ *vs* $Z^{\frac{1}{2}}$ calculated by numerical integration of Eq. (4). The values of the parameters obtained are $\lambda=1.25$, $r_0=12.5$ A, and $D_0=1.2\times10^{-2}$. Figure 12 shows the value of ϕ' *vs* $(t')^{\frac{1}{2}}$ computed for two of the three different concentration isochronal recovery curves shown in Fig. 1. In this case the best fit is obtained with $\lambda=1.25$, $r_0=13$ A, and $D_0=1\times10^{-2}$. In the highest concentration run, which is not plotted, it is not very meaningful to separate the recovery into two separate stages, since at this concentration the two processes merge to give a quite smooth recovery curve. Consequently, application of Eq. (9) using the value of ϕ_D used for the lower concentration curves does not give very good agreement. However, in this case if we arbitrarily increase somewhat the amount of I_E recovery relative to I_D recovery, excellent agreement can be obtained with experiment. The initial radial distribution function describing the separation of interstitials from vacancies is shown in Fig. 13 for the observed value $\lambda=1.25$.

The fact that one activation energy characterizes both I_D and I_E and that agreement is obtained with the Waite theory forms the basis for the conclusion that substages I_D and I_E represent respectively the correlated and random interactions of the freely diffusing interstitial. The specific values of the parameters λ, r_0, and D_0 depend upon the assumptions made in applying the analysis to the data. The values of these parameters are not required for the discussion in the succeeding sections of this paper.

Recovery Equations for Substage I_E

As is required qualitatively by the data and is seen quantitatively by the agreement with Waite's theory, I_E recovery represents the uncorrelated interaction of the migrating interstitial. In treating I_E recovery analytically, a considerable simplification is introduced if we forget the origins of this randomization and consider I_E recovery to represent the interactions of *initially* randomly distributed defects. While this procedure is lacking in rigor, it is not without some justification. The effect of time, or interstitial migration, on the initial radial distribution function shown in Fig. 13 is to make the distribution function uniform out to large distances. To be sure, in the course of this smoothing, some recovery is taking place as well—I_D recovery. As we have seen, for low defect concentrations at least, the division between I_D and I_E recovery is rather marked. Our procedure then is to make an arbitrary separation between I_D and I_E recovery, which separation we shall consider valid over a range of defect concentrations sufficiently broad that we can examine the concentration dependence of I_E recovery.

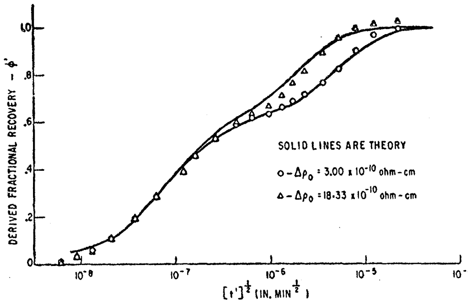

FIG. 12. The derived fractional recovery ϕ' corresponding to two of the concentration dependence experiments in Fig. 1, plotted as a function of the square root of the equivalent time t' and compared with theory.

With the distribution of migrating defects in substage I_E random, it is possible to write down the differential equations which govern the recovery, including the possibility of annihilation, clustering, and trapping. We assume that all interstitial clusters are immobile and stable and that the only migrating entity is the single interstitial. Only clusters up to a total of three interstitials are considered. We also assume that any traps which are present are of the "unsaturable" type which do not change either in cross section or concentration

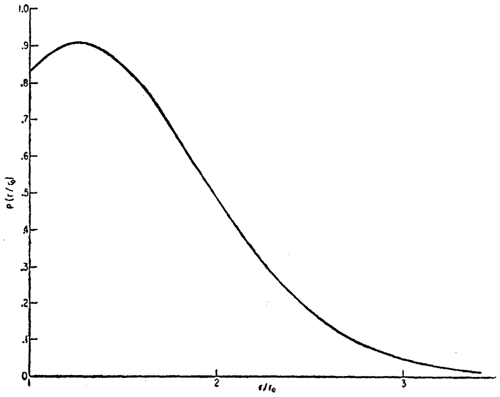

FIG. 13. Radial distribution function for the initial distribution of free interstitials around vacancies, corresponding to $\lambda=1.25$ in Eq. (2) of the text. r_0 is the vacancy capture radius for a freely migrating interstitial. The distribution of interstitials bound in close pairs is *not* included in this figure. These interstitials will occupy sites within r_0.

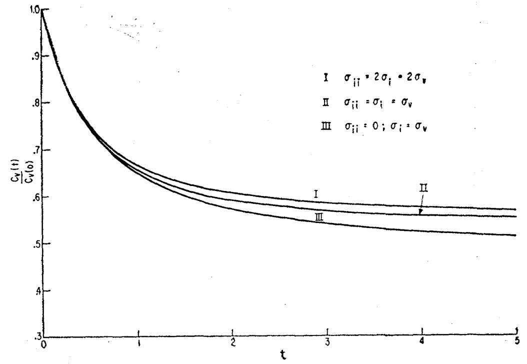

FIG. 14. Numerical solutions to Eqs. (10) for $\sigma_{iii}=0$ and various assumptions concerning the other interaction probabilities.

when an interstitial atom is added. With these restrictions the equations which govern the recovery become

$$\begin{aligned}
&\text{(a)}\quad dC_i/dt = -K\sigma_v C_i C_v - 2K\sigma_i C_i^2 \\
&\qquad\qquad -K\sigma_{ii} C_i C_{ii} - K\sigma_t C_{it} C_i, \\
&\text{(b)}\quad dC_v/dt = -K\sigma_v C_v C_i, \\
&\text{(c)}\quad dC_{ii}/dt = K\sigma_i C_i^2 - K\sigma_{ii} C_i C_{ii}, \\
&\text{(d)}\quad dC_{iii}/dt = K\sigma_{ii} C_i C_{ii}, \\
&\text{(e)}\quad dC_{it}/dt = K\sigma_t C_i C_{ii},
\end{aligned} \tag{10}$$

where the C's represent the instantaneous concentrations of the different entities, K is the average jump constant for a single interstitial, and the subscripts v, i, ii, iii, and t refer respectively to vacancies, single interstitials, dimers (clusters of two interstitials), trimers (clusters of three interstitials), and traps. The σ's are the corresponding cross sections for interaction with a single interstitial; e.g., σ_i is the cross section for the process where an interstitial combines with another interstitial to form a dimer. Since no aggregates higher than trimers are considered, σ_{iii} and higher cross sections are zero.

Experimentally we measure the resistivity given by

$$\begin{aligned}
\Delta\rho(t) = C_i\Delta\rho_i + C_v\Delta\rho_v + C_{ii}\Delta\rho_{ii} \\
+ C_{iii}\Delta\rho_{iii} + C_{it}\Delta\rho_{it}.
\end{aligned} \tag{11}$$

A considerable simplification is introduced if we assume

$$\begin{aligned}
&\text{(a)}\quad \tfrac{1}{3}\Delta\rho_{iii} = \tfrac{1}{2}\Delta\rho_{ii} = \Delta\rho_i, \\
&\text{(b)}\quad \Delta\rho_{it} = \Delta\rho_i.
\end{aligned} \tag{12}$$

Equation (11) then becomes

$$\Delta\rho(t) = C_v\Delta\rho_v + \Delta\rho_i[C_i + 2C_{ii} + 3C_{iii} + C_{it}]. \tag{13}$$

Since we initially start with the same number of interstitials and vacancies and since all the interstitials must be accounted for, we have

$$C_v = C_i + 2C_{ii} + 3C_{iii} + C_{it}. \tag{14}$$

Equation (13) therefore reduces to

$$\Delta\rho(t) = C_v(t)[\Delta\rho_v + \Delta\rho_i], \tag{15}$$

and since

$$\Delta\rho(0) = C_i(0)\Delta\rho_i + C_v(0)\Delta\rho_v = C_v(0)[\Delta\rho_v + \Delta\rho_i], \tag{16}$$

we have finally for the measured fractional recovery:

$$\Delta\rho(t)/\Delta\rho_0 = C_v(t)/C_v(0). \tag{17}$$

Equations (10) need therefore only be solved for C_v. The justification for assumption (12b) derives from the observation that all of stage I_E is suppressed in an experiment on a less pure sample of copper. W. Harrison[8] has calculated the effects of clustering and concludes that (12a) is approximately correct. Bross and Seeger[9] have reached a similar conclusion for the resistivity of a di-vacancy.

Equations (10) in general do not have simple analytic solutions and must be solved numerically. Figure 14 shows the results of such a solution for different assumed interaction probabilities for the clustering process with trapping neglected. In order to make the subsequent analysis tractable, we shall sometimes introduce assumptions which permit analytic solutions.

Radiation Doping Experiments

In attempting to analyze the results of experiments in substage I_E, it is important to assess the various cross sections appearing in Eqs. (10). In Fig. 14 we show that for $\sigma_i = \sigma_v$ (dimer formation cross section equal to that for annihilation) various assumptions for σ_{ii} (cross section for an interstitial interacting with a dimer to form a trimer) give $\Delta\rho(\infty)/\Delta\rho(0) \sim \frac{1}{2}$. From the isothermal recovery curves we estimate that region I_E starts when about 27% of the total resistivity remains. Since the saturation value in region I_E is about half of this value, we see immediately that the assumption that $\sigma_i = \sigma_v$ is going to give fair agreement with theory. However, the dividing line between I_D and I_E is sufficiently uncertain that it is not possible on this basis alone to decide on a reasonable value for σ_{ii}.

The experiment which bears most directly on the question of the relative importance of trimer formation is the radiation-doping experiment where the doping is done by first irradiating at 20°K and then annealing to 80°K. If the amount of doping radiation is large, then in the subsequent irradiation the migrating interstitial will interact predominantly with the products of the doping treatment. Since this doping treatment results primarily in vacancies and dimers, the annealing characteristics of the subsequent irradiation will reflect the extent of interaction of the migrating interstitials with the dimers.

We describe this situation with the following approximate model. The recovery of the doping radiation is analyzed using Eqs. (10), neglecting traps and assuming that $\sigma_i = \sigma_v$. For various assumed values of σ_{ii} we can then by numerical integration tabulate the number of vacancies, dimers, and trimers. In the re-

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[8] W. A. Harrison (private communication).

[9] H. Bross and A. Seegar, J. Phys. Chem. Solids **6**, 324 (1958).

covery of the second irradiation, however, we neglect the direct formation of trimers by the aggregation of three interstitials introduced in this second irradiation. The dominant cluster formation processes are (a) the interaction of single interstitials to form dimers and (b) the interaction of interstitials produced in the second irradiation with dimers and trimers produced in the doping radiation. This latter process is accounted for by considering the previously introduced dimers and trimers as trapping sites of the unsaturable type. The additional vacancies added by the doping radiation are taken into account by suitably modifying the initial conditions in the solution of Eqs. (10). This description of the second irradiation is adopted because Eqs. (10) have simple analytic solutions for the case including dimer formation, annihilation, trapping, but excluding trimer formation. The error involved in neglecting direct trimer formation in the second irradiation should be very small. Figure 14 shows that even if σ_{ii} were equal to $2\sigma_i$, the error is only at most $\sim 7\%$ of the total resistivity added at the bombarding temperature. In the presence of a large doping radiation, the direct trimer formation will, of course, be much reduced. Further, the final result indicates that $\sigma_{ii}=0.3\sigma_i$ and hence direct trimer formation is not too important. The error made in considering the dimers and trimers found in the doping irradiation as effective trapping sites of the unsaturable type is more serious, but the over-all conclusions should still be valid.

We shall only attempt to calculate the final reduction in the fraction which remains when stage I is complete. This corresponds to the saturation values of the curves in Figs. 4 and 6, and we assume here that the slight differences in these saturation values and the values at 80°K is due to the advent of some stage II recovery between 52.7°K and 80°K. The saturation value of the recovery of the second irradiation, assuming dimer formation and the presence of vacancies and traps (clusters) introduced by the prior bombardment, is given by the solution of the following equation:

$$\left[\frac{(2+k)+C_t(0)/2C_i(0)}{(1+k)^2}\right]\left(\frac{C_v(\infty)}{C_i(0)}\right)^2 - \left(\frac{C_v(\infty)}{C_i(0)}\right)=\frac{C_t(0)}{2C_i(0)}. \qquad (18)$$

In this equation $C_v(\infty)$ refers to the final value of the vacancy concentration *including* the doping vacancies, $C_i(0)$ to the initial concentration of interstitials due to the second irradiation, and $C_t(0)$ to the traps (clusters) which are present. The symbol k is defined as

$$k=\frac{C_{vD}}{C_i(0)}=\frac{\Delta\rho_D}{\Delta\rho_0}\equiv\frac{\Delta\rho_D}{f\Delta\rho_0'}, \qquad (19)$$

where C_{vD} is the concentration of vacancies introduced by the doping radiation, $\Delta\rho_D$ the measured doping resistivity, $\Delta\rho_0$ the resistivity increment corresponding to I_E recovery in the second irradiation, $\Delta\rho_0'$, is the total resistivity increment in the second irradiation, and f the fraction of the total recovery in substage I_E. The final saturation value after recovery for those vacancies added in the second irradiation, $C_v'(\infty)$, is given by

$$C_v'(\infty)=C_v(\infty)-C_{vD}. \qquad (20)$$

From Eq. (17), therefore, the saturation fractional recovery of resistivity for the second irradiation expressed as a fraction of the total recovery for all substages is

$$\frac{\Delta\rho(\infty)}{f\Delta\rho_0'}=\frac{C_v'(\infty)}{C_i(0)}=\left(\frac{C_v(\infty)}{C_i(0)}-k\right). \qquad (21)$$

The evaluation of an equivalent $C_t(0)$ for the dimers and trimers produced in the doping radiation proceeds as follows. From the numerical integration of Eqs. (10) including trimers but no traps, we can express the concentration of dimers and trimers in the doping resistivity in terms of C_{vD}. If we let $C_{iiD}/C_{vD}=a_1$ and $C_{iiiD}/C_{vD}=a_2$, then

$$C_{iiD}/C_i(0)=a_1C_{vD}/C_i(0)=a_1k, \qquad (22)$$

and

$$C_{iiiD}/C_i(0)=a_2C_{vD}/C_i(0)=a_2k. \qquad (23)$$

We now define $C_t(0)$ as

$$C_t(0)=k[a_1+a_2], \qquad (24)$$

where the term in brackets is a function of the various interaction cross sections.

For zero doping, the predicted fractional recovery for the second irradiation should be the same as the fractional recovery predicted for the doping radiation. However, since different models are used for the first and second irradiations, this will not be the case. We have corrected for this by using a slightly different f value in Eq. (21) (26%) than in Eq. (19) (25.5%) to normalize predictions for the case of zero doping. This is a small correction.

The above expressions were used to calculate the saturation fractional resistivity for the second irradiation as a function of doping resistivity for various assumed values of σ_{ii} and σ_{iii}. Certainly, $\sigma_{ni}<n\sigma_i$ for if $\sigma_{ni}=n\sigma_i$, no radiation-doping effect would exist since the relative probabilities of clustering and annihilation would be the same as in the absence of doping. The upper curve of Fig. 15 is drawn assuming $\sigma_{ii}=0.3\sigma_i$. The agreement with the (20°K irradiation–80°K annealing) doping experiments is satisfactory. The lower curve assumes $\sigma_{ii}=0$ and lies appreciably below these experimental points. The results therefore indicate that some clusters larger than dimers are formed. To be sure this conclusion is somewhat contingent upon the assumptions made in the analysis. However, a model in which only dimer formation and annihilation are allowed

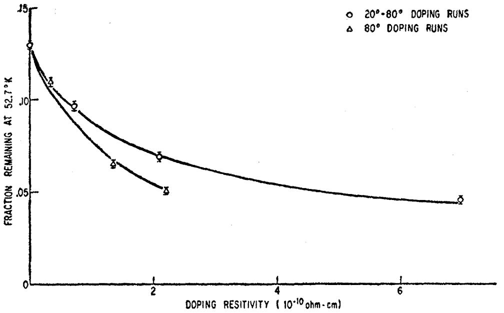

FIG. 15. The effect of the two kinds of doping resistivity on the saturation value of the 52.7°K isothermal recovery. The solid lines are theoretical.

(i.e., $\sigma_{ii}=\sigma_{it}=0$) does not adequately fit the upper curve in Fig. 15 for *any* values of $\Delta\rho_i/\Delta\rho_v$ and $\Delta\rho_{ii}/\Delta\rho_i$. Consequently, we believe the requirement for formation of some clusters larger than dimers to be quite general.

When the doping resistivity is added by direct bombardment at 80°K, the effect on the second irradiation is more pronounced. The instantaneous concentration of migrating interstitials during such a high-temperature bombardment is very low and as a consequence the probability of interstitial clustering is greatly reduced. If we assume in this case that the interstitials are trapped at a fixed concentration of trapping sites which change neither their cross section nor number as a result of the trapping process, then the previous equations apply with $\sigma_{ii}=0$. The bottom curves of Fig. 15 show the predictions based on this assumption. The agreement with the 80°K irradiation-doping experiments is seen to be satisfactory.

An estimate of the number of trapping sites necessary to suppress clustering can be formed as follows. If we approximate the recovery as a simple bimolecular process, then equilibrium between the decay and production of defects is reached at a concentration given by

$$C_E=(K_2/K_1)^{\frac{1}{2}}, \qquad (25)$$

where K_1 is the rate constant of Eq. (1) and K_2 is the constant production rate. Using a value of K_1 determined from the isothermal recovery curves we find at 80°K that $C_E\sim 1\times 10^{-9}$. A trap concentration of $\sim 5\times 10^{-9}$ would therefore serve to suppress cluster formation. Such a trap concentration is certainly reasonable and would not sensibly effect the recovery of the standard-dose irradiations performed at 20°K.

Concentration Dependence of Isothermal Recovery in Substage I_E

From Fig. 14 we can see that the difference between isothermal recovery curves for $\sigma_{ii}=0$ and for $\sigma_{ii}=0.3\sigma_i$ will be very slight. We have therefore analyzed the isothermal recovery using the simple analytic expression which results for $\sigma_{ii}=0$. When only dimer formation is included and no doping or traps are present, Eqs. (10) have the following solution:

$$[K\sigma C_v(0)]t=\left(1-\frac{C_v(0)}{C_v(t)}\right)-2\ln\left(2-\frac{C_v(0)}{C_v(t)}\right). \qquad (26)$$

In comparing with experiment, the constant $K\sigma$ is determined by normalizing at one point on the lowest dose isothermal. The predictions for the rest of this isothermal and for the higher dose isothermals are then calculated from Eq. (26). The agreement between theory and experiment is shown in Fig. 3. The value of f chosen here is 26%.

Equation (26) differs only in the logarithmic term from the equation for a bimolecular process. Equally good agreement between theory and experiment for these isothermals can be obtained with the assumption that the only process which occurs in I_E is vacancy-interstitial annihilation—the fraction left after recovery being ascribed to an inert defect which simply does not partipitate. The rejection of this possible process therefore must rest with other experiments as we have outlined in the initial qualitative discussion of the data.

DISCUSSION

In the previous sections of this paper we have discussed the qualitative and quantitative aspects of I_D and I_E recovery. We shall briefly summarize our conclusions here. Both I_D and I_E recovery represent the recovery due to the migration of interstitials with an activation energy for motion $E_m=0.12\pm 0.005$ ev. The separation into I_D and I_E substages results from a change in the character of the recovery from a correlated to a random recovery. The interstitial can interact with vacancies, chemical or physical trapping sites, and with other interstitials to form clusters. Using these recovery features, we are able to account quantitatively for all the I_D and I_E recovery data. As a result of the quantitative analysis, we conclude that the cross section for the interaction between two interstitials leading to cluster formation is comparable with that for the annihilation interaction between an interstitial and a vacancy. It is further necessary to conclude that some larger interstitial clusters are formed. In this connection, however, the cross section for the interaction between an interstitial and a cluster of two interstitials, is found to be less than that for the interaction between two single interstitials. In the course of the quantitative analysis we made certain simplifying assumptions in order to facilitate calculations. We do not believe that these assumptions, or the parameters which we deduced as a result of these assumptions, are necessarily unique. For example, if we relax the assumption that the resistivity of a cluster of n interstitials is n times the resistivity of a single interstitial, the relative interaction cross sections for the cluster and annihilation processes have to be changed accordingly. However, we feel that the assumptions we have made are sufficiently valid so that the conclusions concerning cross sections outlined above are essentially correct.

Although our results and analysis give a consistent model for stage I recovery, two questions arise concerning the validity of the interpretation. In the first place, it is necessary to inquire whether or not it is theoretically possible for interstitials to behave as we conclude they do, and secondly to inquire whether such a model for stage I recovery is consistent with the observations which have been made at higher temperatures—particularly those experiments which have been advanced as proof that interstitial migration does not occur until room temperature.

In connection with the first question, Magnuson, Palmer, and Koehler[10] have also suggested that interstitials could interact to form stable clusters. Their paper discusses the stage I recovery of deuteron-irradiated copper. They observed an activation energy spectrum for the recovery consisting of four peaks. Their experiment did not allow them to establish the kinetics of the peaks, but they suggested that the first three peaks corresponded to close-pair recovery and the last peak to free migration of the interstitial. As we discussed in the preceding paper, their first three peaks correspond to I_A, I_B, and I_C recovery. The activation energy for their fourth peak and the energy we have determined of I_D and I_E recovery agree within the limits of the analysis. They saw only one recovery peak corresponding to both I_D and I_E because their experiment was performed at such a high defect concentration that the dose-dependent I_E merges with I_D. We conclude therefore that their assignment of the stage I recovery processes is correct. Further, they suggested cluster formation as the explanation for the fact that only ~60% recovery is observed in the stage I recovery of deuteron-irradiated copper. In this connection they showed that the anisotropic elastic interaction between defects calculated following Eshelby[11] was capable of giving rise to stable interstitial pairs. It should be noted, however, that the magnitude of the binding energy calculated from Eshelby's formula is only ~0.1 ev, and if this were correct we would not expect the damage to persist with only minor change until stage III—as is experimentally observed. However, this calculation is probably a poor approximation to the interaction energy of two interstitials. As far as we know, no detailed calculations on such interactions have as yet been performed. Clearly, such calculations should be done.

We now consider the experiments which have been advanced to show that interstitial migration occurs in stage III. The proponents of this point of view have taken two different approaches. One approach has been to advance evidence that stage IV recovery was associated with vacancies and by elimination to assign stage III to interstitials. Since in our model the di-interstitial has been added as a defect which is present in stage III and could conceivably migrate in this temperature range, assignment of vacancies to stage IV does not preclude interstitial motion in stage I. Since we are also not persuaded that vacancies do not move in stage III, we shall not expand this approach further.

The second approach has been to show directly that stage III must be due to interstitial motion. An argument due to Seeger[12] suggests that since the ratio of changes in length,[13] lattice parameter,[14] and resistivity[10] remains constant through stages I, II, and III, the same basic process—namely, interstitial-vacancy annihilation—must be occurring in all three stages. He then argues that if interstitials had moved in stage I, there would be none left to participate in the observed interstitial-vacancy annihilation in stage III. If, however, as indicated by our experiments, interstitial clusters are formed in stage I, their subsequent annihilation in stage III would satisfactorily explain the observed results. There remains the question, however, of the lattice parameter to be associated with di-interstitials. Detailed examination of this question may shed light on the nature of the damage production mechanism in deuteron irradiations.

Brinkman and Meechan[3] also assign interstitial motion to stage III. The primary experimental observations which led them to this point of view are the shape of the damage production curve at ~80°K and the fact that the recovery kinetics in stage III are closely second order. Consider first the damage production curve. They observed that after an initial curved portion the production curve becomes quite linear at high electron doses. Since pure close-pair recovery in stage I would give rise to such a linear dependence, they attribute all of stage I recovery to close pairs. They pointed out that the magnitude of the initial curved portion varied from sample to sample. Hence the curved portion was attributed to the effect of impurities on the initial damage production process. We have performed an 80°K irradiation which has this same sort of curvature (see Fig. 8). However, the damage production curve for an irradiation on the same sample performed at 20°K is accurately linear up to the same total flux of electrons, and hence the curvature at 80°K is not due to the effect of impurities on the damage production rate. A natural explanation of the difference in the shape of production curves at 20°K and 80°K is to say that free migration of interstitials takes place in stage I, in which case the 80°K production rate will indeed reflect the impurity content of the sample.[15]

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[10] Magnuson, Palmer, and Koehler, Phys. Rev. **109**, 1990 (1958).

[11] J. D. Eshelby, Acta Met. **3**, 487 (1955).

[12] A. Seegar, *Proceedings of the Second United Nations International Conference on the Peaceful Uses of Atomic Energy, Geneva, 1958* (United Nations, Geneva, 1959), paper No. 998.

[13] R. W. Vook and C. A. Wert, Phys. Rev. **109**, 1529 (1958).

[14] R. O. Simmons and R. W. Balluffi, Phys. Rev. **109**, 335 (1958).

[15] In this regard, Mercereau and Simmons observed that the production rate for deuteron irradiation at 80°K was also impurity dependent. See F. Seitz and J. S. Koehler, *Solid State Physics* (Academic Press, Inc., New York, 1956), Vol. 2, p. 402.

The problem remains to explain the linear damage production rate at 80°K for high total fluxes. One explanation is to assume that as a result of the early part of the irradiation there are isolated vacancies and a fixed number of interstitial aggregates—perhaps nucleated at impurity sites. If now the cross section for the interaction of a migrating interstitial with one of these clusters increases linearly with the number of interstitials in a cluster, then the relative probability that a newly introduced interstitial go to a cluster or annihilate will be constant. The production rate will then become linear. Doubtless other models can be constructed to explain the linear behavior at high total fluxes.

The second piece of evidence that led Meechan and Brinkman to conclude that interstitial motion occurred in stage III was the observation that the recovery kinetics in stage III was second order. The process of interstitial migration in stage I leading to either trapped or clustered interstitials does not preclude second order kinetics in stage III. If either the di-interstitial or the vacancy became mobile in stage III, then the number of annihilation sites would be directly proportional to the number of migrating defects—thus satisfying the prime requisite for the bi-molecular kinetics. Under certain assumptions such a process could be precisely bi-molecular. We therefore feel that the observations of Meechan and Brinkman are not incompatible with interstitial migration in stage I.

The final conclusion that interstitial migration and clustering occur in stage I is therefore based on two lines of reasoning:

(A) The results and analysis of the electron experiments reported here are in quantitative agreement with this point of view.

(B) The results of other experiments which have been performed on this and other stages of recovery also seem to require this interpretation or at worst are not inconsistent with it.

Although we can say very little about the detailed nature of the interstitial, the data are more consistent with a defect which migrates fully in three dimensions than with one which is constrained to move in a line such as the crowdion.[4,16] Lomer and Cottrell[17] invoked the crowdion to explain what appeared to be an anomalous number of jumps to annihilation in what we call stage II recovery. They showed that the mean number of jumps to annihilation $\bar{n}$ will be of the order of $(\alpha ZC)^{-1}$ for a three-dimensional random walk, while in a one-dimensional random walk $\bar{n}\sim(\alpha ZC)^{-2}$ where C is the concentration of the defects and αZ is the number of new sites the defect can interact with each time it jumps. We assume $\bar{n}=\langle t\rangle\nu\exp(-E_m/kT)$, where $\langle t\rangle$ is the time for 50% of the recovery to occur. Using $\nu=10^{13}$, $E_m=0.12$ ev, $T=52.7$°K, and $\langle t\rangle=10$ min, we calculate $\alpha Z\sim250$ for the three-dimensional case and $\alpha Z\sim3\times10^4$ for the one-dimensional case. Clearly, the three-dimensional case is preferred. However, conclusions based on calculations of numbers of jumps are not particularly reliable, and we do not consider this argument as definite proof that the crowdion does not exist.

Having decided on the features of stage I recovery, there remains the problem of assigning defects and recovery processes to the higher recovery stages. The small amount of stage II recovery we observe could certainly be due to the release of interstitials from chemical traps. Some of the stage II recovery observed by other investigators can probably be similarly explained. In some types of irradiations, multiple defects such as divacancies may be formed. Such defects may explain some of the stage II recovery, since they are believed to have activation energies[18] such that they would move in this temperature range.

It is attractive to assign stage III recovery to vacancy migration since this could explain many of the observed features. However, this would leave stage IV unexplained. (Stage V is generally agreed to be associated with recrystallization.) At this point we feel that we cannot make any definite assignments and must consider the problem still open to further experiment.

In conclusion we would like to interject a word of caution concerning the convention of labeling recovery stages according to the temperature region in which it occurs. In the first place, it must be kept in mind that the appellations stage I, etc., do not refer directly to the microscopic recovery processes. The recovery occurring in a given temperature range following different methods of introducing defects into a sample may be due to quite different processes. Secondly, as we have seen, the same fundamental diffusion process may give rise to macroscopic recovery in quite different temperature regions. Thirdly, the complications inherent in intrinsic processes can be compounded considerably when trapping processes also become involved. And fourthly, as we have also seen, the results of a low-temperature irradiation-high-temperature anneal are not, in general, the same as a high-temperature irradiation. Clearly, care must be taken to ascertain what defect processes are operative in each experiment.

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[16] J. Frenkel and T. Kontorova, J. Phys. USSR **1**, 138 (1937); H. Paneth, Phys. Rev. **80**, 708 (1950).

[17] W. M. Lomer and A. H. Cottrell, Phil. Mag. **46**, 711 (1953).

[18] J. H. Bartlett and G. J. Dienes, Phys. Rev. **89**, 848 (1953).