Theory of Strain Percolation in Metals

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(Received 30 July 1998)

We demonstrate that a deforming metal is a self-organizing critical system, and that the stress-strain law can be expressed as an integration of certain derivatives of internal variables along the critical line of the system. [S0031-9007(98)07460-2]

PACS numbers: 62.20.Fe, 64.60.Lx, 81.40.Ef

The work hardening which occurs during the deformation of metals is one of the central long standing problems in materials science [1]. Because the large numbers of dislocations that form during deformation (distorted and tangled in three dimensions) interact strongly with both long- and short-range forces, work hardening is a many-body problem of vast complexity. It remains largely unsolved today. More specifically, the major theoretical challenge is to adequately account for the evolution of the dislocations into partially ordered structures during deformation and then show how the stress-strain law follows from the properties of these structures. After the early stages of deformation, which we do not address, the structures become three dimensional cells consisting of interior regions of nearly zero dislocation density, surrounded by walls composed of regions of high dislocation density, which are locked into the lattice by immobile or sessile dislocations. The walls are at first very diffuse, but as the deformation proceeds, they become narrower and better defined. See Argon [1] for further details of the phenomenology.

An adequate theory of work hardening will therefore consist of (1) a theory of the ordering process, and (2) a theory of the percolation or transport of mobile dislocations through the blocking walls of the partially ordered structure. Our purpose in this paper is to address the second problem, assuming that a partially ordered cell structure exists. This second problem is particularly important because the stress-strain law of the metal is a direct outcome of the transport model. We will parametrize the interactions of the mobile dislocations with the cell walls and expect to obtain these parameters either from experimental observations or from the results of dislocation simulations specifically designed for the purpose.

We assume first that a metal single crystal has been brought to a particular state of strain with generation of a definite 3D dislocation cellular structure. After the 3D cells have developed, Seeger [2] has shown from the slip traces appearing on the surface of the metal that the deformation is highly concentrated in linear slip bands randomly spaced on the surface, and that the length of these bands is much larger than the cell size.

In the next step, we subject the system to a small increment, $\delta\tau$, in the external stress and assume that a responding increment of strain, s_0, is nucleated at the weakest cell in a slip band. It is consistent with Seeger's observations to assume that this strain nucleus percolates through the sample from cell to neighboring cell, and the object of the model is to track the percolating strain through the slip band. We assume that throughout the process, the strain is restricted to its initial slip plane, so the evolution is two dimensional, at least to a good approximation.

After a strain burst is initiated and has begun to percolate through its neighboring cells, we assume that growth of the strain cluster takes place only on its periphery. The physical reason for this restriction is that after a given cell is strained, some of the dislocations participating in the strain will be incorporated into the walls of the cell, thus hardening the walls and making subsequent strain in that cell more difficult.

The law for the propagation of strain from a strained cell to a neighboring unstrained cell through the wall separating the two is assumed to be the linear relation,

$$s^* = sa\,. \tag{1}$$

Here, s^* is the strain induced in the unstrained cell, s is the strain in its strained neighbor, and a is a stochastic function, called the amplification factor, which depends on the properties of the wall separating the two cells. Since s is equivalent to a small pileup of dislocations expanding in the strained cell, the force on the front dislocation facing the wall barrier between the two cells is proportional to the number of dislocations in the pileup [3]. Since any physical process which allows strain to be propagated through the wall will depend on the force of the front dislocation against the wall, then it is reasonable to assume that s^* is also proportional to the number of pileup dislocations, and therefore to s.

An essential feature of the model is our physical picture for the two quite different mechanisms by which we propose strain is propagated through a wall separating a strained from an unstrained cell. In the first case, if the wall is relatively stable, then it can simply act as a source of new dislocations which expand into the unstrained cell. This mechanism can be quantified by writing $a_1 = P_1\zeta$, where $0 < \zeta < 1$ is a random number. P_1 is a parameter of the order of unity which is a measure of the strength and density of sources in the wall.

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But there can be a second, and more dramatic, behavior of a dislocation wall. Suppose the wall is pinned to the lattice, for example, by Lomer-Cottrell locks [3]. Further, assume some of these locks are not very stable and may be broken or "unzipped" by a nearby dislocation pileup, and the wall or a portion of it is thus swept aside. In such a case, the pileup dislocations flow through the broken wall, and the dislocations which were previously pinned in the wall become debris which are added to the pileup in the newly strained cell. In contrast to the first mechanism relating to the action of stable walls, we assume that weak walls are relatively rare, but when activated, yield a large value of a. Quantitatively, we can write such a contribution to a in the form $a_2 = P_2 \exp(-\zeta/\kappa)$, where $P_2 \gg 1$ is a parameter corresponding to the large amplification of such weak walls, and $\kappa \ll 1$ determines the (relatively small) probability for weak walls.

If the contributions of the two mechanisms are added, the form of a becomes

$$\begin{aligned} a &= P_1\zeta + P_2 e^{-\zeta/\kappa}, \\ \bar{a} &\approx P_1/2 + P_2\kappa . \end{aligned} \tag{2}$$

The second equation gives the average value of a in the distribution. The values of the parameters in the amplification function must be determined from the underlying cell physics, which we emphasize must be obtained either from experimental observations or from specially designed dislocation simulations. Such studies, of course, may also show that the functional form of the amplification function may require modification.

In this 2D percolation problem, since many unstrained cells on the periphery of the cluster will have more than one strained neighbor, we propose a rule that propagation occurs only through that cell wall with the largest value of s^*.

A final rule states that since strain is composed of dislocations, the minimum amount of strain which can be induced in an unstrained cell is unity. Thus, values of $s^* < 1$ from Eq. (1) are set to zero.

These equations and rules fully specify the strain percolation model. As noted, the physical problem is 2D. However, the analogous 1D problem has an approximate analytic solution, some aspects of which remain valid in 2D. Thus, the 1D solution becomes a very useful tool in understanding the overall problem. In 1D, if s_0 is the initial strain at the origin, the recursion relation, Eq. (1), becomes

$$s_{n+1} = a_n s_n , \tag{3}$$

where cell indices refer to cell sites on the positive X axis. If the amplification factor is expanded about its average value, the continuum form of the equation can be written

$$\frac{ds}{dn} = \bar{\alpha} s(n) + \phi , \tag{4}$$

where $\bar{\alpha} = \bar{a} - 1$, and ϕ (note that $\bar{\phi} = 0$) has the form of a stochastic "noise" term. This equation has the standard form of a Langevin equation. Its solution is (exponentially) localized near the origin for $\bar{\alpha} < 0$ and has an avalanche character for $\bar{\alpha} > 0$. A critical point exists at $\bar{\alpha} = 0$, where the correlation function is divergent like $1/|\bar{\alpha}|$. In terms of the parameters of Eq. (2), the critical point in 1D is given by

$$\bar{\alpha}_c \approx \bar{a}_c - 1 = P_1/2 + P_2\kappa - 1 = 0 . \tag{5}$$

The critical "point" determined by this equation becomes a critical line in the space of the parameters, P_1 and $P_2\kappa$. [Note that P_2 and κ appear in (5) only as the product.]

The bimodal separation into stable and weak cell walls is an essential aspect of the model. For example, the 1D solution for $P_2 = 0$ exponentially decays from the origin for a subcritical value of P_1, and in 2D a corresponding decay will take place around an initiation site at the origin. But suppose that weak cells are distributed near the edge of the decayed solution just before it drops to zero (remember the rule that s goes to zero when $s = 1$). Then, a percolating strain path from weak cell to weak cell can be found if the distance between weak cells is less than the critical distance where the strain goes to zero. In such a case, percolation will occur on a critical surface in the space of the parameters describing the properties of the stable and weak cells, analogous to that written for 1D in Eq. (5). This kind of behavior, suggested by the 1D solution, is indeed just what is found in 2D computer simulations.

Proceeding to the numerical solution of the 2D model with the propagation law, Eq. (1), the model is "run" by assuming an initiation strain, s_0, at the origin. A single cluster is then grown by a method similar to that of Leath [4] until the growth stops or the cluster spans the (finite) system. We find that the model exhibits what seems to be a fractal spanning cluster at the percolation threshold. See Fig. 2. We also have determined that the percolation exponent, ν, has the value $4/3$, which is the standard value for 2D percolation [5]. Further mathematical details of the model will be presented in a subsequent paper [6].

For the simple case of $P_2 = 0$, a single critical point exists for a critical value of $P_1 = P_{1c} \approx 1.3$, and the critical point is independent of s_0. Below the critical point, the strain decays near the origin in a way reminiscent of the 1D model. Above the critical point, the strain becomes an avalanche. The reason only one critical point exists is that for $P_1 < P_{1c}$, the solution will always decay and never reach the (infinitely) distant boundary, no matter what value of s_0 is chosen.

The presence of P_2 dramatically changes the character of the result. As in the 1D case, when $P_2 \neq 0$, the single critical point expands into a critical surface in parameter space, which separates the region of localized strain from unstable avalanche strain. But, as presented so far, s_0, P_1, P_2, and κ all appear to be independent parameters, which leads to undesirably complex results. The 1D model, on the other hand, suggests that the critical

surface may be independent of s_0, and depend only on P_1 and the product, $P_2\kappa$. If these results remain valid for 2D, the complexity would be reduced to very manageable proportions. We have, therefore, explored the role of s_0 and found that although the critical surface does in fact depend on s_0, its dependence is slow compared to that of the other parameters. Also, the critical surface does not depend exactly on the product, $P_2\kappa$, but P_2 and κ do exhibit an inverse relationship, such that the product can be taken as a rough approximation. Consequently, we are led to propose a modification of Eq. (5) for 2D which we expect to be roughly valid,

$$\bar{a}_c = P_1/2 + P_2\kappa \approx 0.65\,. \tag{6}$$

The constant, 0.65, was determined from the percolation simulations for the case $P_2 = 0$ above.

According to these rough approximations, the critical point now becomes a critical line, as shown in Fig. 1. In the figure, we show the critical line as straight in accord with Eq. (6), but the reader is reminded that this is only a rough approximation.

This behavior is intimately connected with the physics of the cell wall response, as shown in Fig. 2. This figure shows the fractal shape of the spanning cluster overlaid with cells where the strain is greater than some arbitrary large value. These latter cells are dominated by the term in P_2. In these (low probability) cells, the amplification factor is large compared to unity, and a large strain develops. In cells surrounding these sites, however, the strain decays because of the subcritical value of P_1. Percolation through the entire system occurs when the decaying strain surrounding a weak cell can, on the average, reach another weak cell before the strain has decayed to below unity. This general behavior is just that expected from the previous qualitative predictions for 2D based on the 1D model.

In light of these results, the simplified 2D model as encapsulated in Eq. (6) will be adopted as the standard model in what follows. One must always recognize, however, that the simplified model is only a rough approximation to the actual 2D strain percolation problem. But the clarity of the general physical picture which the simplified model permits is well worth the loss in precision.

We have not yet raised the question of how the internal parameters depend on the external observable parameters of total strain in an individual slip band $\langle s \rangle$ (the order parameter in this problem), and the applied stress, $\delta\tau$. It is this connection between the internal parameters and the external observables which gives the model its ultimate usefulness in the stress-strain relations. This dependence can be answered only by an appeal to the cell physics which underlies the model, either by experimental observation, or by direct dislocation simulation. But even though this information is not available, in the remainder of the paper, we explore what can be learned by the fact that such a relationship exists, even if its precise form is not known.

From the solutions of the 2D model described above, we know that a critical surface exists in the space of the independent internal parameters (a critical line in the case of the variables, P_1 and $P_2\kappa$), and that this surface separates a region of localized strain from an avalanche region. All the internal parameters are, in principle, functions of the external driving force, $\delta\tau$. But we need greater specificity in the functionality existing between the external variables and the internal parameters in order to obtain useful information. In the simplified model adopted with internal variables, P_1 and $P_2\kappa$, we will assume that only $P_2\kappa$ depends significantly on $\delta\tau$. Our reason is that the unzipping of weak locks should

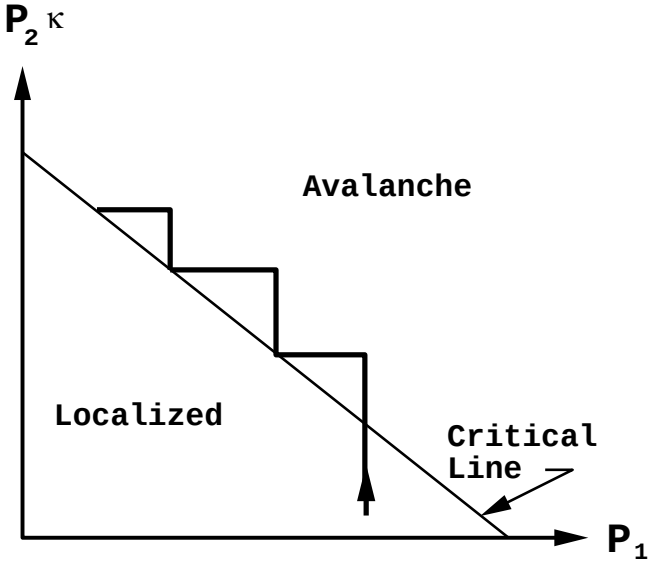

FIG. 1. Critical line, showing the path taken for increasing $\delta\tau$.

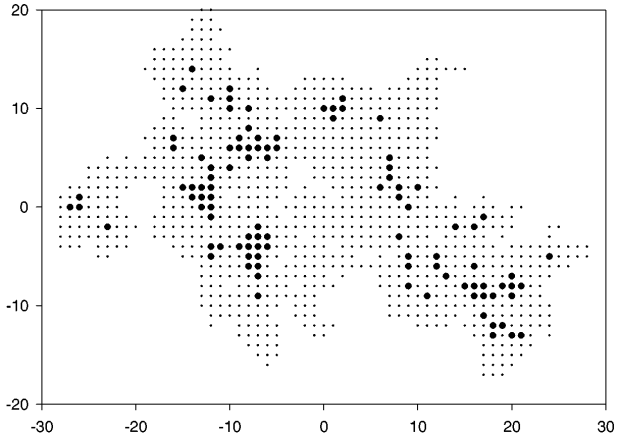

FIG. 2. Spanning cluster. Large circles represent cells with large strain; small circles represent cells with smaller strain. $P_1 = 0.2$; $P_2 = 6.07$; $\kappa = 0.07$.

be a very nonlinear function of the external stress, while P_1 will depend only on $\delta\tau$ in linear fashion. This nonlinear dependence, in our opinion, should overwhelm the weaker dependence of P_1 on $\delta\tau$. This reasoning is very judgmental, and, to repeat, must be confirmed by a more detailed study of the underlying cell physics.

Thus, if the system is started at a subcritical value of $\delta\tau$, and the external stress is continuously increased (Fig. 1), the system will ultimately reach the critical surface, where a strain excursion takes place as the system begins to go into an unstable avalanche. If the system does not sustain an immediate global instability (i.e., the Portevin-LeChatelier instability [7] is inhibited), then some of the new dislocations will be absorbed in the existing walls, thereby hardening them. The hardening walls will in turn stop the dislocation population surge and bring the system back to a new point on the critical surface. Thus, the deforming metal "lives" on the critical surface, and in the case under discussion, the state of the system moves to the left in Fig. 1 as $\delta\tau$ is increased. Such a system is self-organizing [8].

In the simplified model, we will assume that the strain hardening primarily affects the stable wall strength parameter, P_1, and neglect any effect of strain hardening on $P_2\kappa$.

These results, which link the internal variables with the external stress-strain relation, can be given a quantitative form by writing the expression

$$\frac{d(\delta\tau)}{d\langle s\rangle} = \frac{d(\delta\tau)}{d(P_2\kappa)}\,\frac{dP_1}{d\langle s\rangle}\,\frac{d(P_2\kappa)}{dP_1}. \tag{7}$$

In this equation $\langle s\rangle$ is the average calculated strain in a critical strain cluster induced by $\delta\tau$ and is related to the strain, S, in the total system by a sum over the slip bands, $S = \sum\langle s\rangle$. The left-hand side, therefore, expresses the stress-strain law in differential form. The right-hand side is obtained by simple chain rule differentiation of the left, recognizing the functional dependences already proposed. Thus, the first term on the right relates the internal variable, $P_2\kappa$, to the external stress, the second is the wall hardening law, and the third is found from the universal character of the percolation model itself. For the simplified model in question, $d(P_2\kappa)/dP_1 = -1/2$. The first two terms must, of course, be determined from independent dislocation simulation studies or from experiment. We emphasize that the various terms on the right side of Eq. (7) must be evaluated on the critical line (or more generally on the critical surface). This requirement means that the critical state must be known when working at the individual cell level, and this can be an important practical difficulty. However, information such as that provided by the 1D model could prove useful in this connection. More generally, any analytic approach which can be developed for the 2D case, such as a mean field approximation, would have the same usefulness.

In conclusion, we do not claim a "derivation" of the stress-strain law, but we have, instead, successfully formulated a statistical "framework," as we prefer to call it, for converting the physics of dislocation behavior at the single cell level into the desired stress-strain constitutive law. Furthermore, the framework, as we have developed it, indicates what the internal variables in the system might be, and, more importantly, how to obtain them from the underlying cell physics. And finally, we propose that the enormously complex nonlinear and nonequilibrium stochastic problem of metal deformation can be described as a self-organizing critical system, subject to the powerful universality theorems of percolation theory.

It is a pleasure to acknowledge useful criticism and comments from J. Douglas, J. Warren, and R. Minich.

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