Chapter C8

DEVELOPING A CONSISTENT AND REASONABLY COMPREHENSIVE STRUCTURAL THEORY OF PSYCHOSIS

340

SUMMARY OF CHAPTER

This chapter goes still further into details of how the brain is likely to have to organize its coded elements into various types of 'intensive' and 'extensive' sets. It is argued that the best results will be obtained when several such methods are available simultaneously — used in parallel in a pluralistically oriented environment, and that this would be likely to cause considerable difficulties for experimental investigations of the microstructure (where statistical methods are envisaged). These concepts are evaluated in the light of existing clinical and experimental evidence relating to schizophrenia, considering the likely effects of diminished pluralism in producing this sort of impaired mental performance.

C8.1 A closer look at the likely mechanisms whereby implicit "intensive" sets might become organized into the two-or-more types of extensive set

"Intensive" for description, "Extensive" for boxes and leashes — a reminder

To organize thought about the world, one must have workable ways of arranging items into "mathematical" sets. There are several different approaches to this task and, at the risk of repetition, it may help to remind ourselves of the main possibilities, and the "Intensive/Extensive" terminology used by Piaget. One obvious way is to specify some property which all members must have: *Red* objects, or Items which have been *ticked* or fitted with a certain *electronic label*. These are **intensive** definitions, and they have the advantage of flexibility and universality; but they may be difficult to apply in practical activity, especially if we need to act rapidly. For better control, we may need items to be captive in some sense — in a box, or a file, or fenced enclosure, or each tethered to a central bouy by ropes, or in a loop of neighbours each linked bilaterally by some physical-or-virtual attraction, etc. These are **extensive** definitions, with the items being set-members by virtue of their location within an extended region (however that may be defined).

If there happen to be further viable strategies, so much the better; and hybrid techniques should also be useful. The main point is that facility with a variety of these set-manipulating methods offers mental versatility — and we probably spend much of our time translating one type into another in our daily life-tasks. [RRT, 2006]

Practical set-defining Systems?

In the latter half of Section C5.2 we looked at some of the problems of how the brain might develop physical representations of mathematical sets of entities, but without coming to any clearcut opinions as to detailed mechanism. It seems likely though, that any thoroughgoing explanation of psychosis will be closely bound up with details of this type, so it will be prudent to come to rather more definite ideas on such matters of set-organization before proceeding.

Rather than building directly on the proposals put forward previously, let us look at an apparently new alternative suggestion for the basic mechanism — with a view to doing a comparison later on. Looking back then at the pure base sensorimotor level $\mathbf{m^0l}$: Is there any likely scope for set-like organization using only the intensive definition for set-membership? In order to explore some of the implications of such intensive definition, let us suppose provisionally that the ordinary basic linear-elements of this level (as depicted in Figures C5.2/1

341

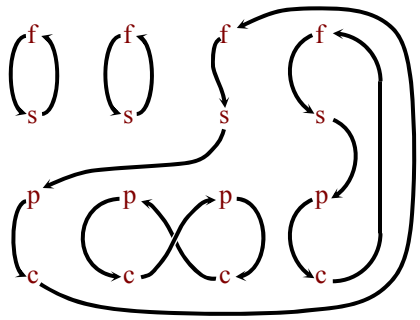


Fig C8.1/1. A possible association-pattern amongst an ensemble of elements, in which each element is required to have one affiliation to another element, (a simplifying assumption).

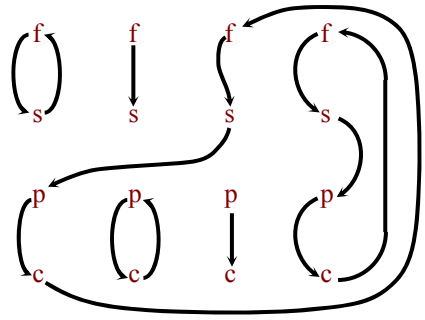


Fig.C8.1/2. Another possible association-pattern amongst an ensemble of the same basic elements, but one in which each element is permitted to have either one affiliation to another element — or none; (a different simplifying assumption).

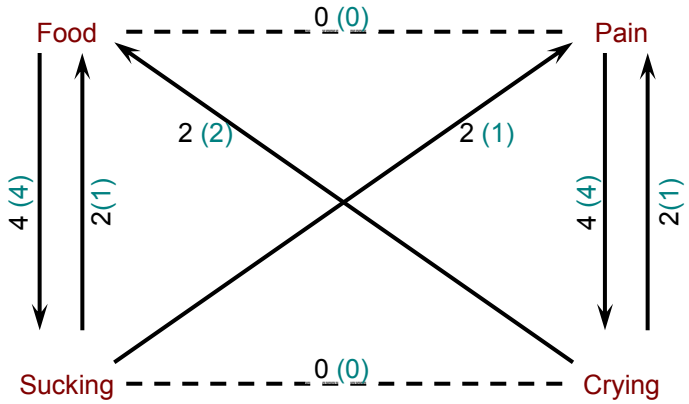


Fig C8.1/3. Strengths of concept associations as depicted in Fig C8.1/1, (and Fig C8.1/2); see text.

and C5.2/2)[73] have an additional ability to keep one[74] name-association reference each — a reference by "name" to some other scheme-element of the same level, without normally activating it. (We may refer to such nodes in the linear string as "β_i" — anticipating their formal equivalence to similar symbolic calls postulated for $\mathbf{m^{½}l}$ elements). But as each scheme is considered to be made up physically of a whole population of linear scheme-elements, and as each element (specified by "i") would presumably be free to form its one association ("β_i") according to its own idiosyncratic history-of-experience, it therefore seems that the scheme, as a whole, will have an effective "network" of *associations* to other schemes — roughly in proportion to the associations actually experienced as significant in the past.

As an illustration of the system proposed here, consider a population of people each of which can like only one other person in the population. If each person does actually like one person, then a diagrammatic representation of the situation would look like Figure C8.1/1, with a complete complement of closed loops; but more plausibly there would be some who would not like anyone at all, and these would result in a number of "*un*closed loops" or linear topologies as in Figure C8.1/2.

Suppose further that the people can be categorized strictly and unambiguously into an exhaustive list of mutually-exclusive sets such as *age-group* or *surname*. In this case we may suppose that each different letter in the adjoining diagrams denotes a different surname, and in calling the surname "f" (say) we will inevitably call forth a reference to "s"; but a call to "c"

[73] Figs.C5.2/**1** and C5.2/**2** are on "original pages 192-193". Likewise for Fig.C5.2/**3**, see o.p.199-200 — and for Fig.C5.2/**4**, see o.p.201-203.

[74] This is a simplifying assumption. In practice the number could well be more, or simply variable.

original page-Nos ↓ 342

might lead to "f" or to "p" — or to nothing at all perhaps, in one random alternative of Figure C8.1/2. If we now leave our analogy of the population of people and return to our postulated population of mental scheme-elements, then we may use the different letters to denote four different schemes (each with a token population of four elements); e.g. f=food-sensation, s=sucking, p=pain, and c=crying. Thus the situation depicted will be roughly as shown in Figure C8.1/3, in which the strengths of the association linkages will be as given by the numbers beside the arrows: plain for the Figure C8.1/1 situation, and in parentheses for the Figure C8.1/2 case.

Some biological considerations, and multi-choice possibilities

343

Unfortunately this still leaves quite a number of questions unresolved, even assuming that the new basic assumption of only-one-reference-per-element is correct. What exactly is the nature of such references likely to be? Are they to be thought of as a sort of (normally inactivated) *"radio call"* to the other elements, or should we think of the other elements as having become *physically attached*? Or should we allow for both, with the first perhaps leading to the second? (Moens, 1973; also Chapters B1 and B2 above). Associated with this problem is the question of whether any "call" of this sort will be addressed to some *particular* individual element (such as the "p" on the left of the diagrams) or simply to all "p"s within reach which are not already captives of some prior or stronger influence.

When faced with this sort of dilemma in a biological context, it seems to me to be best to assume provisionally that all such mechanisms occur to some extent — within a pluralistic environment, and having some sort of statistical mix between the alternative possibilities. We may later find that some or most of these supposed alternatives are untenable after all, but then all we need to do, in a formal sense, is to allocate a statistical-probability coefficient of zero for such cases. In taking such a view, we may find some considerable encouragement in the example of *chemical resonance* in which one considers a number of likely chemical structures based on formal traditional notions of bonding, and then considers that the "actual" structure is a kind of hybrid or random-alternation between these traditional structures. Moreover we should not forget that we are dealing with mental elements which are presumed to function at the molecular level, so we should not be too surprised if we find ourselves invoking principles which are in commonplace usage in chemistry.

344

However the immediate task in this section is to try to be definite and specific, even at the risk of being somewhat inaccurate on points of subsidiary detail. Perhaps the best compromise is to lay down what seem likely to be the predominant trends of structural development, then try to apply them collectively to clinical and experimental observations, and meanwhile make amendments of emphasis wherever these seem to be necessary. (After all, this is probably the strategy which the brain itself adopts, especially at the sensori-motor level). Moreover it might well be that *different* alternative methods will predominate at different stages or for performing different types of task.

If the non-tactile "radio-call" association contact between elements were to be the predominant method of linkage, this would probably be highly flexible but also it would presumably be rather cumbersome and unreliable. As such, it would seem to be a good candidate for the initial primitive-and-impressionable organization for the new-born individual; and it is not too difficult to envisage other arrangements evolving out of it. In such circumstances, an excitation of an "s" element will elicit a call for some other type of element — let us say that it specifies a "p" — and this will operate in a crude way to signify that "p" is also to be considered as being a fellow-member of the same set (and not, in this case, as something to be put into effect — a distinction which we will come back to shortly). For this purpose, we may reasonably assume that the call is addressed indiscriminately to *any* "p" which happens to be within range,

345

rather than to any particular one, thus introducing a random component into the operation whenever there is any actual difference between elements which pass as "p"s.

For a more reliable arrangement we might consider the alternative of an actual tactile linkup between such elements. (Note that as long as we are only admitting the possibility of one-or-none symbolic references from any one element, then such a physical linkage system does seem feasible — without the likelihood of unmanageable branched tangles which might be expected from other super-linear linkage systems. Of course this tangle-problem would disappear in proportion to the *looseness* of the physical linkages — presumably vanishing altogether when the linkages no longer deserve the description "tactile" but have reverted to the "radio-call" linkages which we have just considered). In fact though, in the final analysis, the distinction between "tactile" and "radio-call" linkages is probably less clearcut than we tend to assume in our every-day lives:- Infra-red "radio-call" signals would certainly be of an electromagnetic nature, but then it might be plausible maintained that the relevant types of chemical bonding involved in a "tactile" link would also be electromagnetic in character — though probably with a different type 346
of phase relationship between the electric and magnetic components, as in the distinction between near-field and far-field properties of radio antennae (Walter, 1970; Skilling, 1962).

Molecule-like list-codes

However, be that as it may, we are now considering the closely connected "tactile" type of linkup: so our task now should be to contemplate the likely consequences of such an arrangement, and the mechanisms whereby it might feasibly develop. If the system is to be manageable, it seems likely that the individual elements which are to make up such a chain should retain their individual properties largely unchanged, and if the chain is subject to physical stress we might expect that breakages should occur *between* these elements rather than within them. In these circumstances, a feasible geometrical configuration would be a comb-shaped structure with the individual elements comprising non-rigid "teeth" of the "comb" — on the lines of the arrangement as drawn in C5.2/3 though now, of course, with a physical connection of some sort to replace the more tenuous linkage implied by the dotted lines in that earlier diagram. To make this idea clearer, we may now conveniently look at a modified diagram [Fig C8.1/4] — re-drawn to show the new features which we are now considering. It will be seen that the shape is, in fact, more that of a double-toothed dog-comb than one of the conventional type; and the broken lines are now drawn between the constituent elements to emphasize the postulated looser connection between them:-

348 The main thing to notice about this new diagram is that it lacks any "α" label to act as a clear-cut reference-point to the set-as-a-whole, so that its only means of reference must presumably be via the labels of its individual members (a_i) but especially that of the head-member for that particular geometrical arrangement — a_1 in this case. Such an extra role for "a_1" would evidently lend itself to ambiguity, but then a little thought will suggest that ambiguities of this type may be uncomfortably common during the early stages of the development of a generic concept. It is not unreasonable to suppose, for instance, that our early concept of "dogs in general" consists of a list of particular dogs, of which *one particular dog* serves as the paradigm and label for the whole diffuse concept. (This might next be augmented by similar parallel lists for which a *different* paradigm serves as the lead-in; the two separate lists later coalescing statistically due to frequent parallel usage). As another example, we might note that Piaget himself violates logic in this way and uses the term "Concrete Operations" ambiguously — sometimes *including* and sometimes *excluding* the "Pre-operational" sub-period; (Furth, 1969). (Here the inclusive case seemingly amounts to a list-like organization, whereas the exclusive case can be considered as if it were an isolated "member", independent of any list-structure).

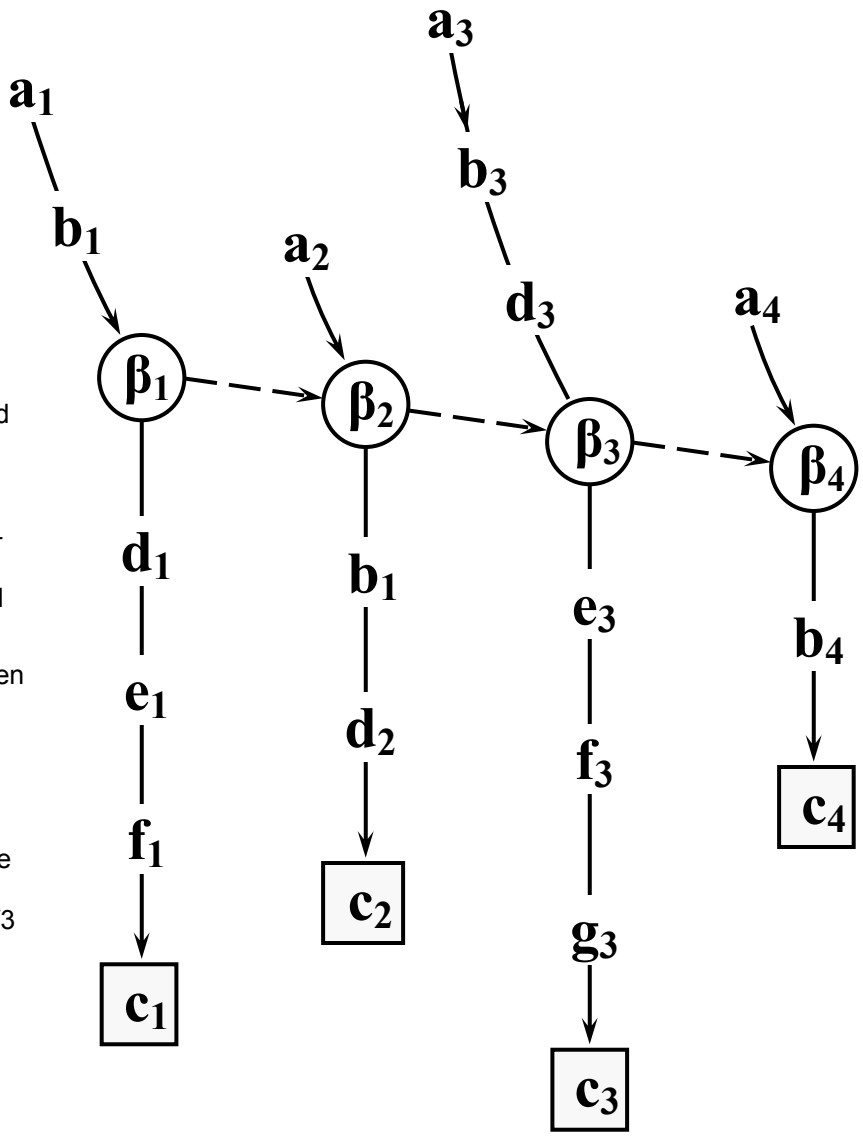


Fig C8.1/4.

Another conceivable arrangement of linear elements formed up to represent a set or "list" of elements.

This may be seen as an alternative to the structures depicted in Figures C5.2/3 and C5.2/4 — though this present structure is less organized (more syncretic), and as such might represent a more primitive method which might, or might not, be a developmental precursor to the other types.

(The broken lines are here seen as a comparatively loose physical bonding, though we have also been considering cases in which such linkages are simply communication channels using "radio-like" free signals such as would be shown dotted in Figures C5.2/3 and C5.2/4).

(347)

349

There are other differences too, of course. Closely associated with the absence of an "α" label is the fact that this ensemble has neither a specialized *master element* (as in Fig C5.2/3)[73] — nor a specialized *name-code* (as in Fig C5.2/4) to represent the set, as such. Clearly there is also, by design, a difference in the locations of the weaker linkages. In addition, in the version which happens to have been shown here, there is less of an "intensive" uniformity among the member elements as compared with the elements as detailed in Figs C5.2/3 and C5.2/4; so one is more inclined to wonder about the mechanism which would have brought such elements together into the same extensive set in the first place — and kept them there. This is the all-important question on which it is now vital to get as much clarification as we can muster.

What could hold the mental list-elements together?

In Section C5.2 it was suggested that there might be a credential-monitoring segment, γ, in the unifying master element Fig C5.2/3 or elements Fig C5.2/4; but there was no detailed suggestion as to how this might operate, and anyhow it is even less convincing here where we are considering a confederation which lacks any master element as such.

Elsewhere in this work it has proved conceptually useful, when faced with apparently-teleological phenomena, to invert the naive concept of causal direction — thus assuming an arbitrary undirected "mutation" which will, on average, be unhelpful to the organism, but will in the long run produce an enduring new adaptive structure by a Darwinian process of selection. Similarly here we may suppose that if an ensemble of the Fig C8.1/4 variety does not prove to 350
have a reasonable norm of (competitive) usefulness, then it will be quietly pushed back into the melting pot. So far so good, but we still need to offer an explanation of the mechanisms involved.

There is no great problem in visualizing how the β_i nodes might invade existing elements in an arbitrary manner, thus producing the necessary mutations. The difficulty, as before, is in showing how efficacy could be monitored. In fact though, there would seem to be a solution to this difficulty on the basis of the synchronizing cross-feed principle of Fig C5.2/2[73], used in conjunction with a trial-and-error procedure:-
In the earlier account of this Monod-and-Jacob system (in Section C5.2), we were thinking in terms of ready-made genetic elements — and moreover these were massed replicates of the same basic structure, arising from a common chromosomal source. But consider the possibility that such cross-feed support systems might arise occasionally between arbitrarily mutating elements which just happened to be within effective communicating distance of each other. As it will presumably be an important property of any meaningful set that its members will tend to be of *simultaneous* relevance, such a synchronizing mechanism would appear to be well suited as a *first step* mechanism for defining the set-membership extensively; but it now remains for us to explain how such membership will relate to the intrinsic intensive properties of the individual elements.

Evolution sequence: Association → Salient member → Label-for-whole

Darwinian survival of the fittest does not apply only to individuals, but also to *ensembles* of 351
individuals; similarly an individual element representing a particular mutant coding will be more likely to survive (other things being equal) if it has happened to achieve an affiliation to a useful ensemble of elements. So as long as there is a plentiful supply of mutants from which to choose and replicate (and after all, the total population of elements could be vast), then there is a reasonable basis for Darwinian selection amongst the postulated ensembles — simply on the basis of the resulting survival probabilities for their members. Such a process could feasibly account for loose types of concept-association and synchronization of the Monod-Jacob crossfeed variety Fig C5.2/2,[73] or indeed for the sequence of single "calls" depicted in Fig C8.1/4 if this is a viable proposition — or any tenable combination of such cross-references between simple member elements. However it is difficult to see how this, on its own, could explain the more organized type of set representation of Figs C5.2/3 and C5.2/4[73] in which the set has come to be both well defined, and also callable and/or nameable in its own corporate right — like a legally formed public company, largely independent of the status of its constituent members.

Probability and Stepwise Evolution

In Section C5.2 and elsewhere, I suggested that some specialized and newly mutated "master elements" might have the power to set themselves up as arbitrary lists of other elements *and also* then erase their own references to those listed members which did not conform to that list's 352
arbitrarily preset criterion for membership — thus eliminating from extensively-defined membership all those elements which did not have the "right" intensive definition. Unfortunately however, satisfying these various different conditions simultaneously by trial-and-error must be regarded as tending towards the highly improbable, even given that there are likely to be a very great number of elements capable of mutating in potentially useful ways and assuming also that a single successful ensemble could be rapidly replicated by "reinforcement". Although ultimately, as in genetic evolution, we must apparently accept a progression by trial-and-error as the only

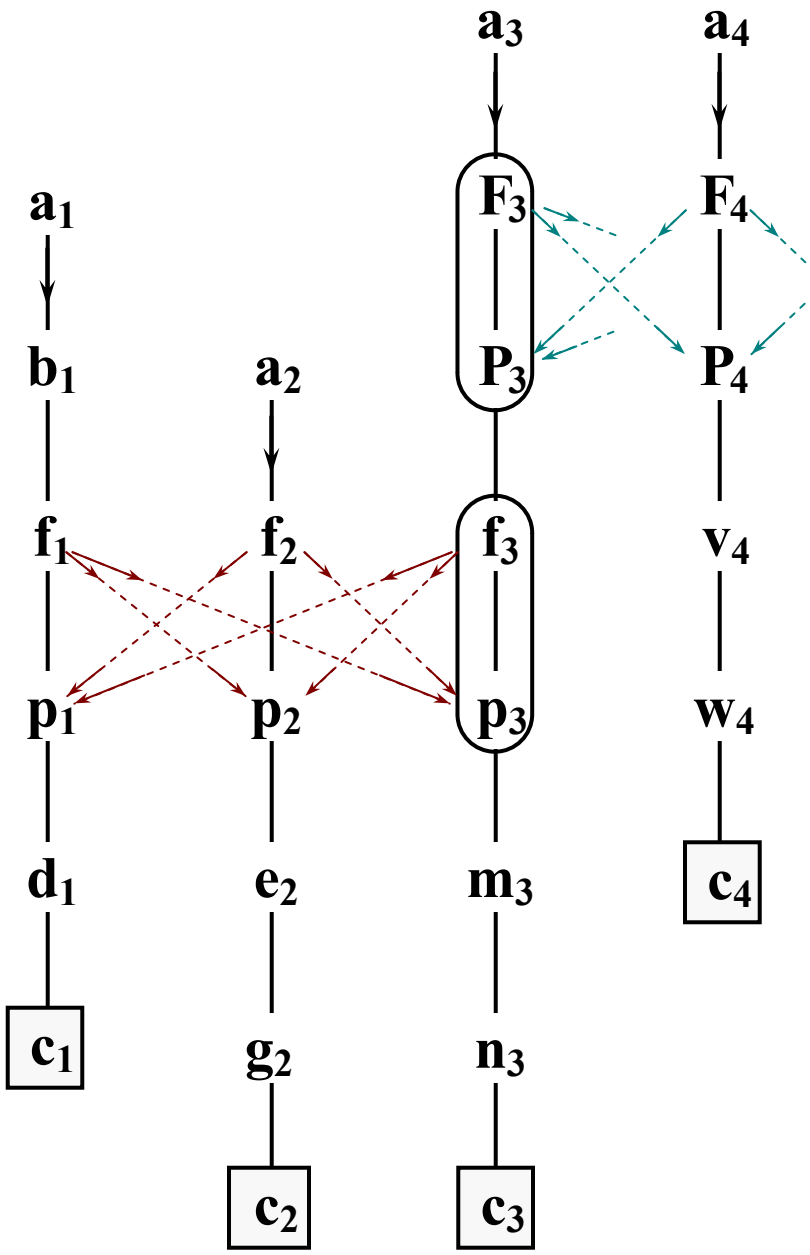
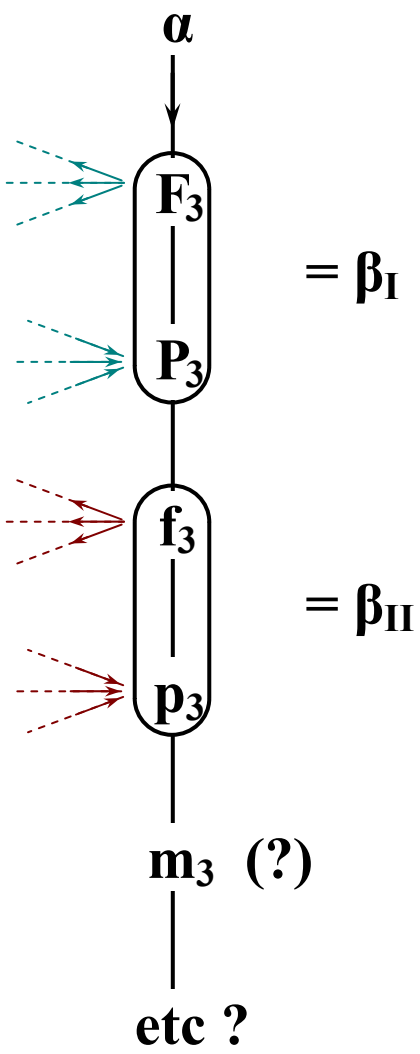


Fig C8.1/5.
One simple representation of a syncretic extensively-defined set. In this case the association-linkages happen to centre on the "a_3" string, giving it a privileged access to the other members. It is thus well situated to capitalize on any chance mutations which would tend to produce versions of it which happen to have properties conducive to its being a "master element" in its new form. (The cross-feed significance of "f" and "p" nodes is the same as in Figure C5.2/2).

possible alternative to a teleological or divinely-guided biology, there are nevertheless limits to what we can reasonably expect trial-and-error to accomplish *in a single step*. Thus we might reasonably credit the occasional spontaneous generation of a virus, but not that of a fully functional amoeba; and it is similarly rather too much to expect the right fully-organized sort of framework for a useful extensive set to appear spontaneously, except perhaps on rare occasions.

But we have just seen that there is at least one plausible trial-and-error process for a part of the job of developing an extensive-definition of sets, so it is now open to us to see whether there might be *other* mutational steps which could result in the same sort of sophisticated system as those considered in Section C5.2 though in a more credible stage-by-stage manner. Let us therefore suppose that our cross-referenced syncretic type of thought-association cluster has evolved within the brain, as postulated in the above paragraphs, and try to explain how this could evolve further into the well-controlled extensive sets envisaged in connection with Figures C5.2/3 and C5.2/4. 353



(355)

Fig C8.1/6.
This depicts the sort of change which might occur to the "a_3" string of Figure C8.1/5, resulting in a "master string" similar in function to that depicted in Figure C5.2/3, but arising from rather different circumstances. The main change would probably be some alteration to the label part such that the a_3 label-coding would become differentiated from other a_3 exemplars, resulting in a new label "α" which could now serve as a reference for the set as a whole rather than for its "most prominent" member. In its new role, other codings on the element, such as m_3, would probably not be maintained by usage and would therefore tend to disappear from the new master-elements — though not from those "a_3" elements which still retained their old function as representatives of the particular member of the set.

It can be seen from Figures C8.1/5 and C8.1/6 that one type of evolutionary change, potentially capable of working successfully, would entail a mutation to the label of one exemplar[75] of one salient member of the set. In favourable circumstances, the new form of the label would fortuitously tally with separately evolved "call" codings on other elements which would then be likely to take on a role of set-manipulation from within the next higher level of the $\mathbf{M^nL}$ hierarchy — in a way similar to that envisaged for the fortuitous growth of control over reflex schemoids during the sensori-motor stage.

356

Thus we now have a rather more credible postulate on how a well-controlled set might evolve — in *two* steps instead of the single step proposed in Section C5.2, and which for the moment we may simply refer to as "step l" and "step 2". In view of the supposed potential for occasional further development of group-like properties, we should also bear in mind the likelihood of there being potential for another — "step 3" — as well; but as this seems likely to have only secondary relevance to our present concern with causes of psychosis, we need not develop this further idea in any great depth here. Suffice it to say that it might well involve a selection in favour of mutations which enhance the symmetry and orderliness of syncretic ensembles like the (over-simplified) one depicted in C8.1/4; but it is reasonable to suppose that "step 2" could be a prerequisite for this type of development, or at least be of some considerable assistance.

[75] Here we should bear in mind that, according to the current theory, there would normally be many such exemplars existing redundantly in parallel; so any single one of them could readily be released for other purposes.

Rival "α-masters" as a possible factor in psychosis

However, returning to the discussion of step 2 itself, it rather looks as though such an evolutionary process might occasionally run into trouble when there are rival claimants among the set's membership for the role of "the most salient", or at any rate when these or other circumstances lead to there being rival "master-string" α-structures whose properties are in some way incompatible. Such rivalry for the "empty throne" may well be quite common in the early stages of step 2, and perhaps even the later stages as well, without necessarily causing any great disruption. Moreover as long as rival formulations can be kept communicationally separate by 357
compartmentalization, then there may be no harm in such an arrangement even in the long term. However if some important conceptual structure of a higher $\mathbf{M^nL}$ level has been built on the basis of one set of α-schemata, and if these become eclipsed by, or confounded with, a new set of α-schemata before the system has had sufficient time to adjust, then this higher $\mathbf{M^nL}$ system is likely to be thrown into major confusion whenever the second type of α-schemata is in the ascendancy. To what extent this could be avoided by gradual transition, or by psychotherapy or whatever, would presumably depend very much on the particular system and the prevailing circumstances.

Anyhow we shall here take it as a postulate that confusions of this type are a decisive factor in the production of clinical psychosis, and it seems to follow from this that we should expect a given "simple" psychosis to be specific to a particular organization at a particular $\mathbf{M^nL}$ level. — But then there can be no guarantee that such "simple" conditions will not spread their effect in a chain-reaction which would similarly unbalance other systems, and perhaps provide exacerbating positive feedback into the original system as well. Nevertheless if this postulate is true to any significant extent, then we might expect that experimental and clinical studies which rely on *correlation*-seeking procedures will produce more mystification than enlightenment on the nature of psychosis because any observable behavioural syndromes are more likely to reflect the second- 358
ary chain-reaction processes than the nature of the original "simple" psychosis, or its connection with *step 2* difficulties.

Other Set-Definition Methods — (beyond Intensive and Tethered-extensive)

So far the discussion in this section has been concerned only with the "tethered" type of extensive definition for the sets. Members have been considered as being either physically bound together, or more likely it is their representative "names" which are physically bound, and these are presumed to be in potential contact with the member elements by means of *intensively defined* calling mechanisms. We have not yet brought the present discussion back to the "bounded" type of extensive definition, but already it is beginning to become clear that any straightforward trichotomy of set-definition types may be an oversimplification. For one thing, we have just been reminded that tethered extension is likely to operate *in close cooperation* with intensive set-definition; but there are also reasons for believing that intensive definitions themselves are possibly specific to particular distances-from-source, or even from sourc*es* (plural). Such possible effects have already been discussed above in Section C6.7; but it will assist the present discussion if we recall the essential points here, and consider their likely implications for the methods of defining set-membership.

For one thing it was argued that, thanks to optical dispersion of the calling-signals, the actual 359
electromagnetic "key" pattern which such signals would present to reception sites would *vary* according to the distance which the signal had passed through the dispersive medium. This has obvious implications for an extensive definition of the sets involved (over and above the intensive "callsign" properties) because the effective target region for any particular coding-and-distortion-tolerance would seem to be a spherical shell, of specific dimensions, centred on the emitting source. [As a matter of detail however, we should bear in mind that unless the dispersive medium

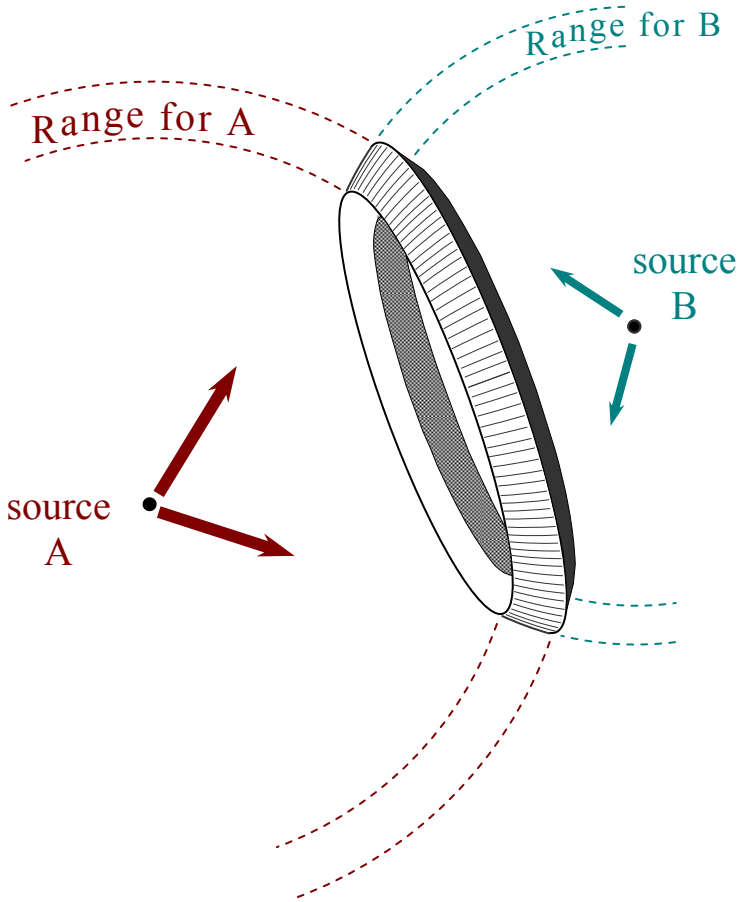


Fig C8.1/7.

An illustration of the sort of region which is likely to be implicitly specified when two range-specific signals are required simultaneously for effective reception. In practice, the shape is likely to be much more irregular because the diagram shown here assumes unjustifiably that the medium will be homogeneous and isotropic throughout.

(360)

happened to be homogeneous and isotropic, the actual shape of the reception-region would not be such a neat geometrical shape. And in view of the likelihood that the bulk of any dispersion would be caused by such inhomogeneities as myelinated fibres, this caveat is hardly to be ignored. On the other hand though, we may happily interpret this concept *topologically*; and in some circumstances we might even get away with statistical approximations — though this should certainly be regarded as dubious pending an in-depth mathematical treatment of the whole dispersion issue]. Anyhow, the essential point here is that there is not only a bounded extension criterion superimposed upon the supposed intensive definition of sets, but it seems that the two processes may actually interact, and that the boundaries of effective areas may have much more intricate shapes than one might at first suppose.

361

But Section C6.7 went further and considered the possibility that, in some circumstances, the effective signal would be an interation-effect or interference-pattern from two or more separate sources, whose in-phase operation is likely to have a special significance worthy of detection. Anyhow the geometrical implication for such a process is shown in Figure C8.1/7, assuming a homogeneous isotropic medium and two relevant emitting sources. Here the effective receiving-region has been narrowed down to the ring-shaped overlap of two spherical shells, and of course any case which required the interaction of any further sources would entail an even more selective target area. (As before, this neat picture is likely to be severely disrupted by inhomogeneities in the dispersive media, but the general principles should still apply).

It will be convenient to apply the term *Range-Bounded Extension* to this potential method for defining a set, whatever the number of emitting-sources involved: one, two, or fifty.

As it would now seem that a signal may be guided to its "intended"[76] destination by two largely-independent mechanisms (a code-specific receptive-site, and a range-specific "broadcast beam"), we would appear to be dealing with mutually cooperative redundant systems, both fulfilling roughly the same function. This means that the system might well be able to continue 362
functioning even if one of the mechanisms is disrupted — though we might well expect there to be some loss of efficiency as a result of the reduction in redundancy, and that this will show specific features depending on which of the mechanisms has been disrupted. Such phenomena seem likely to have some bearing on the attempt to explain psychotic symptoms, as we shall see.

Sets Defined by Barriers such as Cell Membranes

But when we were discussing bounded extensive definitions earlier, we were more inclined to envisage the physical set boundaries as being actual physical barriers such as cell membranes. Such a view is clearly still tenable, though it will be expedient to make a clear distinction between it and the above Range-Bounded type; so let us refer to it as *Envelope-Bounded Extension*, or *Barrier-Bounded Extension*, depending on whether the boundary is seen as being complete (such as a cell-or-vesicle membrane) or merely a non-enclosing "wall" (such as the endoplasmic reticulum). It remains then to explain how such set definitions might become established, and postulate the particular purposes they might serve.

One process whereby such categorization-by-location would probably be set up is that of embryological cell-differentiation. Thus it would seem from the work on brain-mapping (see Section C5.4 above; especially concerning Hubel and Wiesel) that for the more permanent ($\mathbf{M}^{-1}\mathbf{L}$) parts of the brain organization, there is a ready-made orderly array of "pigeon-holes" into which incoming processed signal patterns are sorted. So no-matter whether such cells operate as a single integrated unit, or whether their essential activity is at the molecular level, in either case the cell's overall function will be specialized and its effective processing will evidently be limited to a single restricted "topic". It is rather less clear to what extent these orderly arrays of cells really are "ready-made" by "purely" genetic means. As with other embryological developments, 363
we should be on the lookout for shaping effects arising from early attempted usage; — ortho-maturation again. (We shall come back to this point again in the fourth paragraph below, in connection with the supposedly less primitive origin of such categorization of elements in later development).

Spontaneous Clumping and its Interaction with Boundaries

In addition to any such genetically inspired specialization within cells, we should not be entirely surprised to find a comparable type of specialization occurring *spontaneously*, very much in the same way as land use will tend to become locally-specialized *even in the absence* of legal constraints and natural geographical features (which might perhaps be compared to genetic factors). Moreover the reasons for such clumping would appear to be similar in that there are certain advantages in having particular types of entity in close proximity to each other — be they communicational entities, or economic, or cultural/emotional — so aggregates will tend to continue to build up, within limits, once they have actually begun.[77] Such beginnings may have

[76] It will, I hope, be clear now from previous discussion that this channelling of information is not to be attributed to teleological or vitalistic forces. We should rather think of such channels as evolving arbitrarily (with a large percentage of failures) and then (sometimes) being adopted by an existing system because of an apparent usefulness which the system discovers in them. Of course, once the channel has become established, then it does make some sense to talk loosely about "intended" destinations.

[77] For theoretical considerations of the consequences, to the homogeneity of the system, of various simple assumptions about inter-element affiliation: see Goel (1970), Goel and Leith (1970), Leith and

been inspired by genetic factors or whatever, or they might simply have occurred in any arbitrary location wherever several potentially-cooperating elements happened to occur simultaneously. If micro-pipette electrodes detect some sort of functional specialization at different sites, notably cell-bodies or nodes of Ranvier, then this would hardly be surprising even within the so-called "association cortex", but such findings need not necessarily imply that the electrode has detected all the available information at these locations, nor that the specialization is total and exclusive. Nevertheless such phenomena are clear evidence of some degree of "Envelope-Bounded Extension", or something very like it. 364

In fact, the existence of "natural" boundaries in the form of cell-membranes and similar barriers, will presumably greatly influence the way in which the local aggregations develop — just as natural geographical boundaries profoundly influence the way in which man or beast will develop a specialization in land-use. The isolation of any given area from its neighbouring areas will increase the likelihood of homogeneity *within* that area, and its difference from its neighbours. The boundary will also act as some sort of a brake on any tendency for the aggregate to grow uncontrollably at the expense of the system as a whole; (and it may well be that this formal similarity to the problem of cancerous growths is no mere coincidence). Such restriction on potential communication also brings us back to Ashby's important point that "connections" within the brain should be severely limited, and not superabundant as one might suppose.

Just what sort of local homogeneity should develop will presumably depend on various factors including the ratio of locality size to the effective range of signals within the locality. Thus we might expect the operation of large localities, such as the Purkinje cells, would be significantly different from the operation of small localities. The degree of isolation will presumably also be of considerable importance (and subject to manipulation): excessive isolation would effectively exclude the locality from the general system, while vanishing boundary-effects would of course remove the special feature we are considering. 365

Coming back then, to the question of cells which are supposedly already pre-organized by genetic means, it may well be that there is no *strong* influence brought to bear to determine what specialization the cell should adopt — but that this would not usually be necessary, since only slight "hints" from genetic, potential-usage, or near-neighbour sources would generally suffice to steer development in the "right" orthomaturational direction. For regions of the brain where there is no predictable pattern to such "hints", then we will presumably find no detectable regularity of function between individuals; hence the so-called "association areas".

Consolidation into LTM, and its Breakdown in Psychosis

We have by now distinguished at least four different basic methods which the brain might plausibly use to establish which elements belong together in "sets". Post-learning "consolidation" into more suitably organized forms of memory would seem to entail the ability to reorganize such sets from one type of physical embodiment into one-or-more alternative form. It seems likely, for instance, that this is the sort of activity which results in the conversion of Short Term Memory into Long(er) Term Memory — a process which can be sabotaged by bilateral hippocampal lesions, as in the case of a particular patient "HM", (Scoville and Milner, 1957; Milner, 1966; Kimble, 1969; Milner, 1970), alias "Mr Henry" (Iversen, 1974) — though one should be cautious about assigning a detailed role to the hippocampus on this evidence, or in attributing this effect to *all* concept-consolidation phenomena. 366

So far then, broadly speaking, we have considered four different types of set-definition: three varieties of extensive definition plus the intensive definition — though we should recall the argument that intensive definitions are likely to be complicated by range-effects arising from

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Goel (1971). Similar considerations also apply, of course, to phase theory in physical chemistry.

optical dispersion and hence probably confounded with the Range-Bounded Extension type of definition. Anyhow, bearing in mind that such mechanisms are likely to be operating pluralistically in parallel, we now have some basis for postulating the type of malfunction which could result in psychotic behaviour:- any breakdown in extensively-defined set-organization would be rather like emptying one's filing-cabinet onto the floor and tearing the pages out of the diary! Assuming, that things were properly labelled (i.e. intensively defined) then one could, in principle, carry on with considerably reduced efficiency — on the "Seek and ye shall find" basis.

367

This example emphasized pre-existing "files". Alternatively or additionally we might consider what our plight would be if *no new files or lists* were to be allowed in any sort of reliable extensive form (even though we night be allowed access to existing files). Here again, some sort of coping behaviour might be possible, but our performance would clearly be seriously impaired. Moreover we may presumably envisage various degrees of such breakdown in both cases, so that we should expect different gradations in the symptoms — or even major qualitative differences as critical thresholds are reached. (Incidentally we might perhaps also expect some impairment of performance from the opposite extreme of too rigid a file-structure).

Another noteworthy point is that such breakdowns *need not be universal*, applying to all realms of the patient's thought, (though in some circumstances they might be — e.g. in response to some generally active toxic agent). Thus a disruption effecting language and logic performance in the left hemisphere, need not necessarily have any direct bearing on musical ability in the right hemisphere; or different subject-material might be differentially effected even if, from the macro-anatomical point of view, they were stored at the same site. In particular, a disruption at one $\mathbf{M^nL}$ level might well occur in isolation from the performance at other $\mathbf{M^nL}$ levels; and this is a point which entails some important consequences:-

Symptoms varying greatly according to which "M^nL"-level fails

368

Let us refer back to Table C5.4/I and work down the $\mathbf{M^nL}$ scale considering the types of symptom which are likely to result from a deficient extensive-definition capability at each level. Such a failure at the $\mathbf{M^3L}$ level would be commonplace, and indeed we might doubt whether the $\mathbf{M^3L}$ level exists at all; but anyhow most people seem to manage perfectly well despite this. Failure of $\mathbf{M^2L}$ "formal operations" brings us to one type of symptom often associated with psychosis, though it occurs in other circumstances as well: this entails a failure of logical or mathematical coherence — in a degree considered undesirable within the culture concerned (after all, no man can match a computer in some features of this skill). Such lapses from "secondary process thinking" (to use Freudian terminology) are commonplace of course; even for "normal" people they occur during dreaming, under intoxication, fatigue, and pressure of events. Those people having a chronic deficiency here but not at any lower level, would quite likely pass as "normal" in fact; though presumably they would be classified as "having a low IQ" or as being innumerate, or perhaps even credited with socio-economic class attributes such as "semi-skilled" or "unskilled". Indeed this is a feature which we might do well to investigate in some detail, though it lies outside the scope of this present work.

369

Failure at $\mathbf{M^1L}$ would be more serious, producing behaviour which most people would regard as bizarre — such as devising *ad hoc* sets of objects with scant regard for the culture's view of which objects "should" belong together. (Here, as has often been remarked, there is a tenuous distinction between "madness" and "creativity". After all, sometimes the culture could benefit from a re-drawing of its conceptual boundaries). However there is nothing very creative in the more syncretic thought of the more severe cases. Such scant following through of mere shreds of association suggests a serious lack of the ability to manage sets — i.e. to manage lists of more than one object at a time; and this tends to confirm the suspicion that such patients are just making do with unsupported intensive definitions, whereas the normal person would make

considerable use of one-or-more type of extensive definition to support the process — both regarding grouping entities found useful as a set in the past, and also on an immediate Short Term Memory basis.

Even more serious conditions are likely to arise from defects at the $\mathbf{M^0L}$ and $\mathbf{M^{-1}L}$ levels. The $\mathbf{M^{-1}L}$ case scarcely belongs in our present discussion because its organization is probably different, with perhaps no bearing on the intensive/extensive issue in its present form; and any developmentally-important defect will probably be of a non-psychological nature — acknowledged as "organic" by the medical profession. $\mathbf{M^0L}$ failure would constitute an inability to comprehend objects as such — a non-acquisition (or loss) of the subconscious realization of 370
the mathematical group-like properties of those significant ensembles in our environment which *we* call "objects". Because objects are, on the whole, remarkably consistent in displaying their properties in a comparatively simple form, it will be most unlikely that such learning will not occur (or be lost) unless physical $\mathbf{M^0L}$ handicaps render such lessons virtually inaccessible. Such a blockage at the senses or their nerve-tracts, or indeed in the would-be set-handling mechanisms of the brain, will presumably leave the unfortunate individual in a vegetative state, as depicted in the film "A Day in the Death of Jo Egg"; and borderline cases such as deaf-and-blind babies need most carefully organized experiences of touch, texture and olfaction if they are to have anything like a normal development (Tyrrell, 1977). Once basic concepts of this type have been achieved, it seems hard to believe that they could be lost again in view of their presumed stability — unless perhaps under extreme trauma and sensory-deprivation, or an elaborately rigged environment of ephemeral dissolving pseudo-objects! Regression back down through $\mathbf{M^0L}$ therefore seems unlikely.

Here we have been discussing *objects* (as semi-rigid geometrical entities) as if this were the only type of development which depended directly on the $\mathbf{M^0L}$ sensori-motor stage. This may be a somewhat simplistic view, considering that interpersonal relationships are also developing at 371
the same time. Of course these relationships may well be interpretable in terms of objects — after all, as we have seen earlier, one's self is a key prototypal object against which to assess other objects, and parents constitute other key objects. But it is not difficult to see that there will be a qualitative difference, at some stage, between objects of the more geometrical type and those of the more human type; and this will presumably mean that there is a possibility that one type might come to malfunction even though the other might develop properly. There is some possibility then, that we may have here a vulnerable developmental point at which an inadequate or inappropriate set-organization could lead to *autistic* or *psychopathic* tendencies. This however is a side-issue, and I shall not pursue the matter further here.

Addressing the correct M^nL in Daily Life — an important skill

Another logically possible type of malfunction within the brain's set-organization is what we might call "meta-linguistic confusion" — the incongruous lumping-together of elements which properly belong to *different* $\mathbf{M^nL}$ levels. Thus sharp*ness*, a quality of primary perception properly belonging to the interface between $\mathbf{M^{-1}L}$ and $\mathbf{M^0L}$ might become reified and be treated mentally as if its internal coding were that of an actual object (belonging at the $\mathbf{M^0L/M^1L}$ interface) — and all this without any ($\mathbf{M^2L}$?) awareness which could render the activity into an acceptable *metaphorical* usage. Similarly the confusion could occur one step further up the $\mathbf{M^nL}$ scale, between an *object* and a *set* (perhaps the set to which it properly belongs — "this slug" 372
versus "slugs in general").

During development, such meta-linguistic confusion is probably natural, inevitable, and commonplace (as implied by the mechanisms postulated earlier in the present section, in relation to Figure C8.1/4; but normal development presumably brings a fair measure of mastery of such mysteries, through closure-seeking processes and the establishment of a specialized

"administrative" set-organization whereby elements are generally attributed to their proper **M^{n}L** levels. In principle this could be effected by the establishment of extensive definitions for the respective levels; but in view of the apparently small number of such sets, and their very large membership, it would seem to be a more economical plan to use some form of *ad hoc* intensive definition for this purpose — and in the circumstances this could well take the form of a "tag" system such as those discussed previously in connection with punishment/reward, affect, and feelings of familiarity. Alternatively perhaps there is no such explicit administrative set-structure at all, and its function is adequately implied by the massed membership-patterns of the ordinary sets, evolving in a "proper" **M^{n}L** way, in the light of experience.

It is of some importance to pay special attention to possible differences in set-organization for different aspects of mental activity. In assessing clinical evidence with a view to finding 373
meaningful categorisations amongst symptoms, it will be potentially very useful to have in mind likely differences in the underlying mechanisms, because any separate anomalies in these mechanisms might reasonably be expected to be the cause of separable behavioural abnormalities — or at least different emphases in the behavioural patterns. One such possible difference in mechanism is that between "administrative" and "ordinary" sets, which we have just been considering. Another is the distinction according to subject-matter; and another is the important distinction between **M^{n}L** levels, which also raises the question (discussed in Section C3.2 and depicted in Table C5.4/I) of whether the same material substrate is used for all **M^{n}L** levels, or whether for instance the lower levels operate wholly or partly within the more ancient and primitive parts of the brain, such as the tectum. It is not necessary here for us to arbitrate decisively between such alternatives, but the existence of a conceptual framework which poses such choices might well make it easier to devise more plausible interpretations of clinical observations than those currently offered.

Summarizing Section C8.1

To summarize the discussion of this section:- In general we have been exploring the implications of the supposed existence of alternative physical embodiments of mental set-structure — the more fundamental intensively-defined type, and at least three types of the more instantly-accessible extensively-defined variety. Some consideration was given to the question of 374
how one type could be "translated" into another, though it will be clear that the treatment of the subject was not exhaustive (such as to offer a formal internal closure on the issue), and indeed such an attempt would seem to be premature pending a detailed quantitative investigation of the biochemical and physical issues thought to be involved. In particular, the point raised about the likely observational confounding of Range-Bounded Extension effects with those of Intension (because of optical dispersion, especially if this is a variable) is likely to create great difficulties for any detailed systematization of the theory; though of course it might be possible to demonstrate that this is a non-problem in practice. Anyhow the essential point here, for present purposes, was to give a feel for the sort of processing which might be taking place, and how this might manifest its malfunctions at the clinical level — a matter which we turned to in the concluding part of the discussion.

[A further remark, on the specific issue of the suspected range-related nature of intensive definitions, might usefully be made here. We have seen that even though a molecular "label" may be receptive to a given signal-pattern after it has traversed optical path *x*, this will be no guarantee that it will be receptive to "the same" signal when it has travelled a different distance *y*.[78] This leaves us with a further question:- Does the "*x*-distance" version of label have a 375

[78] There may well be more to this matter of optical distance than we would expect at first sight. Thus electromagnetic distinctions between "near field" and "far field" may well be involved — and/or the question of whether there is chemical-bond contact of any sort.

better chance of evolving into the "*y*-distance" version than any other random label which happens to be present? And if it does, is it likely to evolve via a gradual Lamarckian progression — perhaps as it gradually shifts position; or will the change take the form of a sudden mutation? The trouble with the Lamarckian gradualistic conceptualization is that it is difficult to devise a mechanistic non-teleological explanation of how the process might occur — except perhaps in exceptional "freak" circumstances when the natural course of chemical progression just happened to coincide with rather involved biological needs, without requiring any guidance from feedback mechanisms.

As for the primary question which implicitly asks whether there is any point in evolving an "*x*-distance" version when it is really a "*y*-distance" version which we will ultimately need, the answer seems to be twofold:- Firstly there probably is some advantage — some "transferability of training" — but no absolute advantage from the molecule's own private point of view. Secondly, and more importantly, the advantage which might be gained from such approximation to the optimal should be seen from the viewpoint of the system as a whole, rather than as a "promotion-system" for individual elements. In this light, we should see the advent of roughly-appropriate mechanisms and procedures as creating a suitable environment for further progress, by focussing the developmentally-influential ongoing activity onto more relevant transactions. But once the interim structures have served this purpose, aiding the evolution of still better structures, it is more than likely that they themselves will be bypassed as obsolete, *C'est la vie!* 376

Even if the structures discussed here turn out to be purely fictional, it will perhaps still be worth posing similar questions for any other structural evolution which seems likely to be occurring; — and considering whether the same suggested answers are pertinent in the new context.]

To conclude this sectional summary, we should recall several general principles of operation which have been considered as likely to apply. Firstly *parallelistic pluralism*: there are likely to be a number of alternative methods and structures for many tasks within a biological system, especially within the mental activities of the higher vertebrates; and these alternative systems should be considered as potentially operating in parallel — and perhaps in cooperation. Sabotaging one will therefore not necessarily stop normal functioning, though it might impede its efficiency; so traditional notions of "the controlled-and-designed experiment" should be evaluated with some caution. 377

Secondly, although there may be such alternatives, some types are likely to be much *more common* or more readily implemented by the system; so we may often be justified in ignoring some-or-all of the theoretical alternative varieties. Thirdly, as we have just discussed parenthetically a couple of paragraphs ago, some alternatives may serve as *evolutionary staging-posts* — a practical necessity for the proper development of the system, but expendable in themselves. It may be that they themselves undergo the change "required" for the system, or the changing function may pass to other physical subsystems. Fourthly, and finally, such changes are seen as having a *basically random* fortuitous inception — on essentially Darwinian lines — and the apparently teleological nature of such "guided" developments is seen as stemming from a selection process, which is indeed guided, but on a post-hoc basis — judged on performance.

C8.2 Interpretations of Clinical Schizophrenias and Other Conditions, on the Basis of this Theory

In any attempt to devise a generalized theory to account for a wide and poorly integrated area of clinical phenomena, it is likely to be a useful policy to seek out some single work which offers a reasonably balanced and comprehensive yet condensed coverage of the whole field. As well as essential description, this should include a balanced outline of existing partially-successful theories. For our present purposes, an eminently suitable survey of this type is available in the form of Chapman and Chapman's book: *Disordered Thought in Schizophrenia*, (1973); and page

or chapter references during this section will refer to their work unless otherwise stated. Accordingly we shall now look at various distinguishable themes which arise within their book — often from the differing viewpoints of individual workers in the field — and try to reconcile them with the reductionist approach which has been discussed above. We may consider the themes in their approximate order of apparent importance, as judged by their salience in Chapman and Chapman's survey:-

(1) Faulty clusterings of concepts.

This includes the formulation, traceable through Stransky and Kraepelin of a "loss of inner unity of thinking, feeling, and actions" (Chapman & Chapman, page 8), such as not sticking to the point; or (page 9) citing Bleuler, the patients "lose … logical ordering of their trains of thought. … familiar associations … are absent", while "the most unnatural combinations … are formed, because their incongruity is not perceived …". Subsequently, in Chapter 5, Bleuler's (1911/1950) theorizing is discussed in some detail as the concept of "Broken Associative Threads"; though the authors complain (page 110) that "Bleuler never explicitly defined 'associative thread'..[but] implies that [it] .. is not an observable event, but a construct …". 379

In the light of our foregoing discussions, these observations and comments make interesting reading. Taking the authors' criticisms about observability first: If the theories developed in the present work are anywhere near the truth, then it is small wonder that Bleuler was unable to hazard any very credible guess as to what the "threads" might be in material terms, nor any details concerning how they might operate. And even if he had, then he would have been in an even worse position than we are now when it came to suggest ways in which such a mechanism might be directly *observed*. Clearly then, these threads would have had to be *constructs* in this sense.[79] But it is one of the arguments of the present work that this need not mean that the construct is necessarily limited to being a mere convenient fiction (Section C1.1), though this *could* be so. After all, as it is argued there, there is a case for suggesting that even our most respectable "observations" are fundamentally still constructs in the strict qualitative sense — though well separated from *acknowledged* "constructs" on some scale of corroboration and hence of indubitability.

Not that we need accept Bleuler's "threads" at face-value, but it should now be apparent that 380
a comparatively trivial translation can be made into the conceptual framework of the present theory of "call-signs" (intensive definitions of one type) and physically constrained sets (extensive definitions), both of which may be construed as having some functional and topological affinity with the "thread" concept. Clearly the present account does attempt a more detailed development, but this is not to belittle the pioneering conceptualizations of Stransky, Kraepelin, Bleuler, and others.

If we accept the plausibility of the conceptualizations of the previous section, then we will not find it difficult to attribute the patient's faulty thought-associations to a break-down in one-or-more of the postulated mechanisms by which the brain is supposed to represent *sets*. To be more specific, it is probably a failure of Tethered or Range-Bounded Extension — leaving the patient to make his way, as best he can, with the more primitive and ubiquitous Intensive-type of definition for his sets. This would seem to account for his "default solutions" in the form of syncretic thought (supposedly based on intensive definition), and arguably also for any "overinclusion" which he might exhibit (Chapter 9). Similarly any "Excessive yielding to Normal Biases" (Chapter 6) might also be seen as essentially the same mechanism; and the same again with the overusage of (Tag-using Intensive?) emotional associations, as expressed in the

[79] like the formal status of the "tape" concept (Chapters A1 and A3, above), before any structural interpretation was attempted.

passage from Jung (1906) quoted on page 226: "The pure laws of association play quite an insignificant role when confronted with the unlimited power of the emotional constellation." All these manifestations may be seen as tendencies inherent pluralistically in the normal individual adult, but which come to the fore when the controlling and order-enhancing influence of extensive set-definition fails to operate properly.

Some interesting points are raised by the view that the schizophrenic might, in various ways, be showing signs of regressing back to an earlier stage of development, (Chapter 10). Here the emphasis seems to be on the loss of the ability to think abstractly — a matter which we will return to below under item "(4)" — but presumably any type of regression, anywhere on the $\mathbf{M^nL}$ scale, would come under the same heading. In general, when the individual loses some of his repertoire of set-handling ability at one of these levels (which we will call the "$\mathbf{M^iL}$" level), then as an additional-or-alternative strategy to making-do with syncretic thought at that level, he may happen to try reverting to some well-tried mode of thought belonging to the $\mathbf{M^{i-1}L}$ level; and this is clearly just a new form of words to describe "regression". (In fact any reversion to syncretic thought should also be regarded as regression — indeed it might be argued that it is itself an $\mathbf{M^{i-1}L}$ phenomenon; though such fine distinctions do not materially alter our present argument). 382

It is worth noticing that schizophrenic and regressive conditions cannot really be identified with normal childhood stages, nor with the mental orientations arising normally in more primitive cultures. Chapman and Chapman (page 215), in discussing the study by Bolles (1937), re-state one difference as: "The children, unlike schizophrenics, were able to shift from one approach to another in solving a problem and were able to disregard differences and grasp similarities." Assuming that the assessment of "comparable mental age" (page 214) is above serious reproach in the light of the present theory, this is likely to indicate that the schizophrenics have an additional deficit — in the sets which organize the sets-under-consideration, a matter which we will discuss below under "(2)". In any case we cannot ignore the fact that schizophrenics have bouts of comparative normalcy in which, presumably, the set-structure makes a partial recovery for long enough to operate on *still-existing* higher-level *elements*, such as schemata for abstract thoughts, which would simply not be accessible to children. Similarly, different background histories of experience are likely to produce different numerical weightings amongst the codings for different categories of element, and hence influence the "strengths" of mental-association differentially. Thus: "The younger children probably learned the stronger meanings of words earlier, since they are the more common meanings" (page 220, discussing Chapman et al (1961)). 383

We have seen that a large number of the symptoms of schizophrenia may plausibly be attributed to this syndrome of a "faulty clustering of concepts", which means apparently that some aspect of the individual's ability to handle sets has somehow broken down. In fact we have so far concentrated on the simpler variations on this theme, leaving others to be discussed below as special cases. The type which we *have* looked at here seems to centre on one $\mathbf{M^nL}$ level at a time, though it is by no means clear whether actual patients customarily show this same simplistic bias; and it is more than likely that such an impression is merely an artefact arising from the way we organize our collection and analysis of data — using *collective* populations of subjects, and expecting our data to produce *isolated orthogonal dimensions* at arbitrary levels of resolution chosen by *us*. Nevertheless, if we take this impression at face value, we are left to consider which single $\mathbf{M^nL}$ level or levels suffer from this failure, and produce their respective symptoms — some of which will be classed as "schizophrenic", while perhaps others will not. In fact this issue has already been dealt with in Section C8.1 where it was suggested that it was the $\mathbf{M^1L}$ level which produced behaviour sufficiently bizarre to be regarded socially as schizophrenic, while not yet so debilitating as to be regarded as physical or vegetative. In Piagetian terms then, this places the more essentially "schizophrenic" phenomena within the orbit of *Concrete Operational* failures: an inability to manipulate objects, symbols, and social-objects in a coherent "group- 384

like" fashion involving set-manipulation, though rote performances and pre-set schemata might seem to work adequately in isolation.

Of course it might be pondered that such a state at one $\mathbf{M^nL}$ level could precipitate a similar deficiency at one-or-more other levels — resulting in a confounding of symptoms within the one patient. (And this might occur even if just one $\mathbf{M^nL}$ level were a sufficient initial cause for schizophrenia). In such circumstances, it would be somewhat misleading to state baldly that single simple $\mathbf{M^nL}$ phenomena were the cause of the whole syndrome of symptoms, even if this were strictly true in an indirect way.

(2) Shakow's Major and Minor Set, and his anomalous "Preparatory Interval" experiments.

Shakow's formulation of schizophrenia, (Chapter 12, Chapman and Chapman), attributes the symptoms to a "loss of major set" — where the word "set" is here used in the psychologist's sense of "mental set" toward a particular subject-matter, and not the mathematical sense of a collection of entities which we have been using above. (This is perhaps just one of those unfortunate clashes of terminology which is bound to arise from time to time when once-separate disciplines start to overlap. One might well argue that there is some common element of meaning between the two usages, but that does not help us much when we are trying to delineate precise meanings, especially at the exploratory stage as at present. Nevertheless, both usages are so well ingrained in their respective fields, that it seems prudent just to make the best of the situation — differentiating the ambiguous word "set" by adjectives such as "mental" or "mathematical" wherever the distinction is not clear from the context). 385

In Chapman and Chapman's words: "Shakow defines 'set' as a state of readiness to respond to a particular stimulus and 'major set' as a state of readiness to respond appropriately, that is, in a way that facilitates adaptive behaviour" (bottom of page 244). These definitions are couched in an operationalist-oriented terminology which is of rather limited value for our present objective of postulating structure, but they do serve as a useful benchmark. 'Minor' or 'Segmental' set appears to be left simply as meaning "Non-major set"; thus (bottom of page 248): "Examples are [1] apparently aimless response, [2] response to isolated aspects of the task, [3] repetition of previous responses, and [4] response to irrelevant and internal stimuli" — thereby accounting "for the positive symptoms" of schizophrenia.

It will be instructive to look at each of these items in turn, and attempt to give interpretations, in structural terms, in the light of the present theory. 386

Taking [M] 'major set' first, we may reasonably suppose that a "readiness to respond appropriately" will depend crucially on the adequate availability both of task-relevant schemata at the Concrete-Operations ($\mathbf{M^1L}$) level and also of situational-and-social schemata at the same or higher levels. Moreover, for efficient execution of anything but the most trivial of tasks, such schemata would seemingly have to base their organization on some sort of extensively-defined (mathematical)-set structure — and mere intensive-definition would not really be adequate. So if this *is* what 'Major Set' entails, or largely entails, then its loss could be confidently expected to produce schizophrenic symptoms, and indeed we could then reasonably identify the situation with the failures discussed above under "(1)".

Looking next at [2] "response to isolated aspects of the task"; this could be seen as a *failure to integrate* the various aspects of the task into one whole extensionally-defined set, and *maintain* it in functional form for long enough to make proper use of it. This could take the form of ▪ failing to form an integrated inner mental *perception* of the task or the objects involved in it, or it could be ▪ a failure to marshal the relevant *action*-orientated schemes appropriately, but it is perhaps more probable that the failure would be ▪ general and encompass all such mathematical-set 387

transactions, to a greater-or-lesser average extent. Accordingly, wherever there is a non-materialization of an effective mathematical-set, its role is likely to be taken by some arbitrary would-be *member* of it[80]. But a *member* is not the same thing as any *set* it might belong to, and if the individual proceeds on the misleading assumption that it *is* the same, then we might well expect his behaviour will reflect this by his only taking cognizance of detached *parts* of what we would regard as the total situation. (Incidentally, this could also be taken as one explanation for the phenomenon of "narrowed attention").

Two paragraphs back, the distinction was made between *task-relevant* schemata (at the $\mathbf{M^1L}$ level), and *situational-and-social* schemata (which were seen as either $\mathbf{M^1L}$ or $\mathbf{M^2L}$ phenomena, and quite possibly both).

Anyhow, the essential point here is that the very existence of a "task"-or-aim is often simply a particular aspect of the ongoing social situation — which normal people will usually take for granted and categorize correctly without conscious thought; whereas the faulty mechanisms within a schizophrenic's mind are likely to make the same mess of such socially "obvious" things, as they would for detail of the task itself. This would seem to account for two more of the symptoms attributed to 'Minor Set':- [1] "apparently aimless response" and [4] "response to irrelevant and internal stimuli". After all, when one comes to think of it, it requires a considerable amount of developmental sophistication to divine what (if anything) one's "proper aim" should be within a social context, or which stimuli are to be considered "relevant" given a particular complex context in which it is often *social convention* which defines what should be regarded as salient. If the patient's mathematical-set-structure is unable to build up an adequate model of the ongoing social situation, in the terms which the culture expects, then he will assuredly wander into gross irrelevancies. Even if (like a computer) he were able to handle the task material itself with perfect proficiency, he would still need an absurd amount of guidance (like a computer!) if he is to avoid perpetrating "idiotic howlers"; but of course such proficiency might always desert him too, and in any case there is no guarantee that the guidance could be implemented. 388

This leaves [3] "repetition of previous responses" (now encoded internally). This may be regarded as similar to divining the social situation, as discussed in the previous paragraph, in that both entail some sort of monitoring and control of more basic action-oriented schemata. The emphasis is now rather more introspective, on the lines of: "What are my own aims? And am I making any progress towards them? So, what is the next step?" However if we choose to claim that these aims and self-monitoring skills derive ultimately from the culture[81] — then the distinction becomes rather tenuous. 389

[1+4+3] In any case, it seems plausible to attribute such self-monitoring activities to the workings of the postulated mathematical-set structures, and thus essentially just another example of the same basic phenomenon — though the actual sites of the relevant ultra-micro structures might differ for the different aspects of the patient's overall repertoire, so the severity of malfunction need not be the same for the various symptoms, nor for the various $\mathbf{M^nL}$ levels which could be involved.

We should not pass by this phenomenon of "repetition" without remarking on its apparent formal similarity to Piaget's "Circular Reactions" in infants (Chapter A3, above). Not that we need read much into such a similarity, but some such schizophrenics may well not be properly

[80] — some of whose molecular elements might (in happier times) be capable of mutating into the nucleus of just such a set, on the lines discussed in connection with Figure C8.1/4, above

[81] — or perhaps even if they are merely due to experience based on the physical inevitabilities of the environment

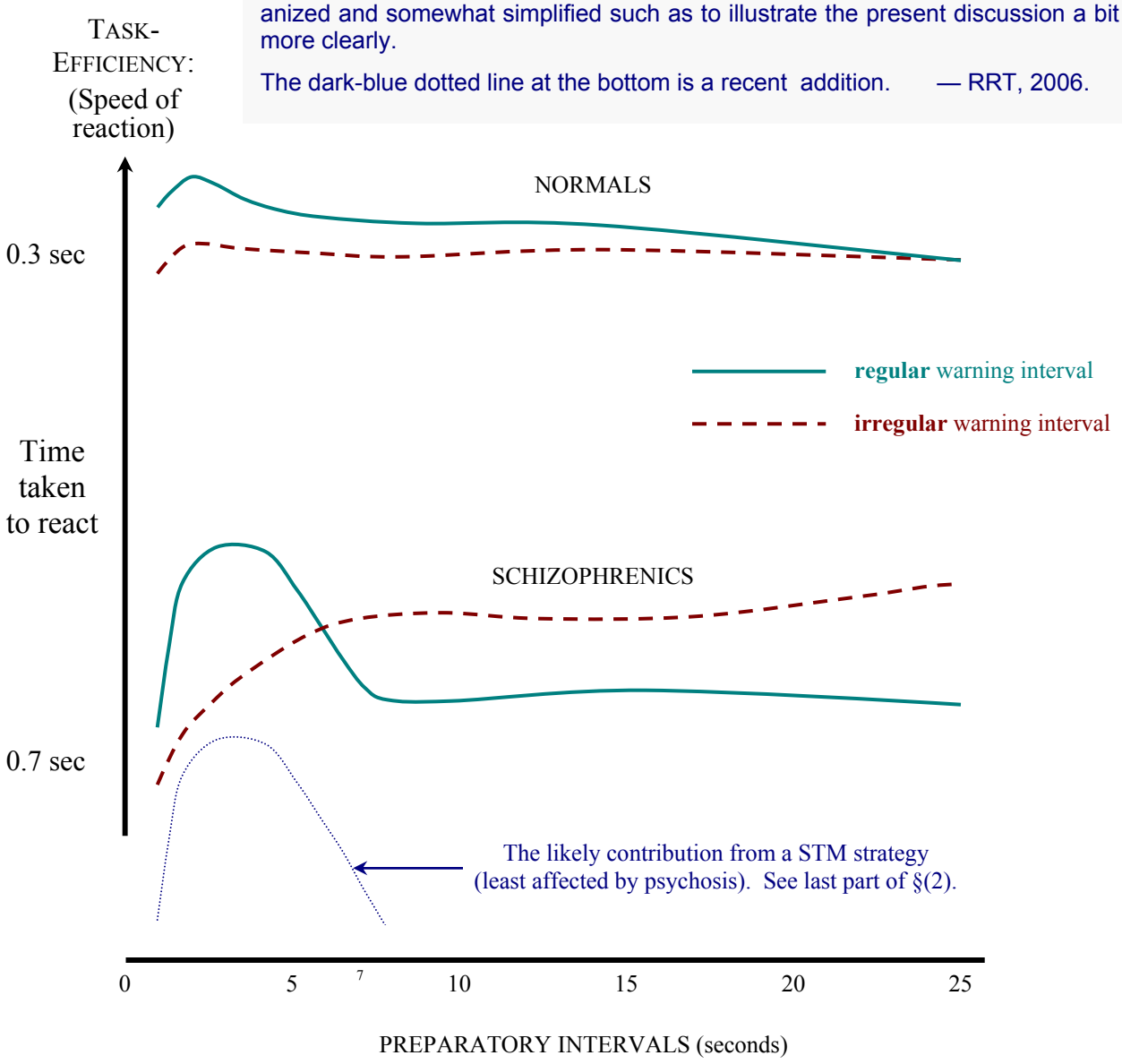
aware that "I have already just done that!" — and this is probably true *initially* for infants as well. Or if the patient or infant *is* aware of the repetition as such, then they presumably feel that the action is rewarding in itself, without questioning whether it makes sense (within some wider logical-or-social context) to just do it again.

390

Fig C8.2/1. At this point, the original 1978 version of this work replicated Rodnick and Shakow's (1940) diagram of "Mean reaction times of twenty-five schizophrenic and ten normal subjects at the various preparatory intervals of the regular and irregular warning procedures".

Partly for copyright reasons, those experimental findings have here been reorganized and somewhat simplified such as to illustrate the present discussion a bit more clearly.

The dark-blue dotted line at the bottom is a recent addition. — RRT, 2006.



Reaction speed, apparently depending on facility in handling mathematical sets

We may turn now to Shakow's findings of an anomalous pattern of reaction-times for schizophrenic patients (pages 244-246, Chapman and Chapman), see Figure C8.2/1. In these experiments there were two stimuli: a preliminary bell, and then (1 to 25 seconds later) a light to which the subject was expected to respond as quickly as possible. If the subject had any way of knowing how soon to expect the light after he had heard the warning-bell, then one might expect him to be able to use this knowledge somehow such as to give him a quicker reaction when the light-signal duly arrived. Thus if a given trial occurred within a block of trials in which this "preparatory interval" was (say) 10 seconds for each case, then the subject might be able to make some use of the realization that this was the case; whereas if there were no such regularity in the length of the preparatory interval within a block of trials, there would seem to be no such helpful knowledge available for utilization. 391

If, for the moment, we look only at the result in Figure C8.2/1 for preparatory intervals of more than 6 seconds, then we notice that the normal subjects do indeed perform better for the "regular" presentations (except when the difference vanishes for the very long 25 sec. delay); but the schizophrenics actually performed *better* for the *irregular* presentations! Meanwhile the performance for these same schizophrenics *was* slowed down *as expected*, but only for those cases in which the preparatory interval was less than 6 seconds — and moreover a plausible explanation for this "crossed-graph" inconsistency was offered by Zahn, Rosenthal, and Shakow (1963). This explanation (page 247, Chapman and Chapman) traced some of the mental-set (as to expected-delay) back to the delay encountered in each *immediately preceding* trial,[82] rather than to any constant value which the block-as-a-whole might have; and furthermore it seems that schizophrenics are *more inclined to use this strategy*. In the context of our present discussion, the important questions to ask are now: "What do we suppose is actually happening in micro-structural terms? And why should the structural configuration of a schizophrenic's brain favour one strategy, while that of a normal brain will favour another?". 392

In fact it would seem that we need not look very far because this phenomenon can be seen in terms entirely consistent with those used a few paragraphs ago. The normal subject's thinking will be ordered by set-structures which entail (i) the logico-social concept of the experimental *trial-block* as a meaningful entity existing over an extended time interval, and (ii) the logico-social concept of a *random mix* in which it is futile (or even counterproductive) to expect consecutive elements to be alike. On reflection, we can see that these concepts are by no means so trivial that we can merely take them for granted; and indeed their proper use is probably beyond the powers of a schizophrenic precisely because he will have troubles with mathematical-set structures, especially if he has to hold them over the time taken for a trial-block to be completed. 393

The type of set-failure which we have been considering here will presumably relate to "monitoring-or-controlling" sets; but we should recall that we were earlier concerned also with the supposed "action-oriented" sets and their embodiment in action/perception schemata. If, in general, we expect the schizophrenic to be having difficulties with these sets as well, then we might expect to find evidence for this too somewhere in the graphs. In fact it is easy to see from the graphs in Figure C8.2/1 that the average performance-rate of the schizophrenic is about half that of normal subjects (as mentioned in passing on page 244, Chapman and Chapman), and it seems reasonable to attribute this to an inefficient action/perception set structure.

But there are some grounds for suspecting that we can infer still more from Figure C8.2/1. The Zahn *et al* (1963) explanation accounts for the increasing efficiency-with-delay in the

[82] a strategy which just happens to pay off for the longer delays, given the particular experimental design.

"irregular-presentation" curve for schizophrenics, and with about the same sort of changes in gradient and curvature as shown on the figure — provided we are to trust the *consistency* of the ordinate speed-scale as we move left-or-right along the abscissa[83]. It rather looks then, as though the *absolute* values of the speed-ordinates might be reliable guides to underlying phenomena. If 394
we accept this argument, at least provisionally, then we might well ask what significance should be attached to the large "peaking" of performance at about 4 seconds delay for the regular/schizophrenic curve, and its much smaller counterpart at 2 seconds delay for the regular normal curve.

Two types of time estimate, with crossover at about 7 sec — implications in set-manipulation

This raises a number of diverse thoughts of a rather anecdotal nature, which I have no intention of developing very far here. However they may suggest some interesting lines of investigation, so I shall at least place them on record. To start with, there is the undocumented observation that computer-users operating an interactive tele-type terminal will normally be quite happy to wait for *up to 10 seconds* for the machine to respond, but any further delay is likely to cause irritation and impatience, or at least a conscious awareness of the delay. Next we might compare this to the supposed time-span for Short Term Memory which, judging from Wickelgren's (1970) graphs, would seem to have a half-life of about 12 seconds, though perhaps a more generally acceptable figure would be around 7 seconds. One also gets the (perhaps misleading) impression that it is more difficult to make reasonably accurate estimates of time intervals greater than about 10 seconds, without resorting to such conscious and artificial devices as *counting* the estimated seconds. Accordingly, one might be forgiven for suspecting that there 395
is something qualitatively different available for processes which do not last much longer than about 10 seconds, and that this something may be intimately bound up with the recognized phenomenon of Short Term Memory (Wickelgren, 1968; Peterson and Peterson, 1959; Posner and Rossman, 1965).

This of course raises some questions about the role and nature of "biological clocks". Concerning this I would suggest (i) that there *may* be "purpose-built" *slow transmission-lines*: non-TEM nerve-fibre modes (see Part B, above), *or else* linear molecules (reminiscent of those depicted in Figures C6.7/3 to C6.7/6, but without the encumbrance of logical gates along their length, so that a signal will traverse the path unconditionally, though subject to a more-or-less predictable delay); and (ii) that there is likely to be a definite *upper limit* to the delays which could be modelled in these ways without the use of a more elaborate mathematical-set structures such as various types of "rehearsal", and (**M²L**?) counting procedures — each of which would probably have a practical upper limit also.

Anyhow, we might at least reasonably expect that the one-to-seven second delay intervals might be easier to embody into a useful schemoid *somehow*, thus accounting for the "peaking" of performance; and that this might well be of extra benefit to schizophrenics, if only because it 396
leaves them with a technique which is less adversely affected than others which they might otherwise have had to use.

Concerning the question of explaining why the same does not apply in the case of "irregular presentations", it rather begins to look as though *for these short delays* there is a whole mechanism left more-or-less intact despite the schizophrenic condition, and that under these circumscribed circumstances the schizophrenic *is* able to benefit from the regularity of presentation in the same way as the normal subject — (though still subject to the *overall* slowness of reaction which we discussed earlier). In other words, this could be taken to support a view that there is a

[83] and not *just* trust it to have the right comparative relationship with other values at each different "preparatory interval" value

pluralistic choice of mechanisms capable of taking advantage of the block-regularity when this exists, but that one of these mechanisms can only work for the shorter-delay conditions. And if we accept that there are likely to be two or more moderately-autonomous mechanisms, then we might reasonably expect that in the normal individual each such mechanism will tend to specialize in some particular function. One (or more) is bound to be involved in the more-or-less permanently established schemata which presumably constitute "Long Term Memory", while other likely roles for other such mechanisms will involve Short Term Memory-and/orAttention, or Consciousness (Popper's "World 2": Chapter A1, above).

397

Given such postulated alternatives with moderate autonomy, then it would not be altogether surprising to find cases in which malfunctions had affected them *selectively* — leaving one (or more) intact despite disruption elsewhere. In the Shakow studies it seems likely that it is the more STM-oriented which has escaped serious disruption; but this need not be taken as representative because it is easy to imagine that if any of his would-be subjects had been suffering from such a disability, then he might well have found it impossible to get any sensible laboratory-oriented responses from them at all. So it may well be that any patients suffering from this debility, alone or in combination, tend to be overlooked in systematic studies of schizophrenics. Indeed such patients could conceivably be classified as suffering from some other clinical condition, and not be regarded as "schizophrenics" at all. Anyhow this may serve as a further pointer to the possibilities of subcategories within and across apparently-adequate clinical classifications.

(3) Loss of Abstract Thought and Logic.

During Section C8.1 above (original-pages 368-369, subhead: *"Symptoms varying…"*), we considered what symptoms were likely to arise from defects at the various $\mathbf{M^nL}$ levels; and it was suggested that the symptoms most characteristic of schizophrenia were likely to have their origin from $\mathbf{M^1L}$ malfunctions. However it was also noted that loss of the ability to think abstractly and logically was taken as a significant extra symptom suggesting a schizophrenic state, and that this defect was to be considered as a malfunction at the $\mathbf{M^2L}$ or "Formal Operations" level. If both types of symptom are both indicative of schizophrenia in some measure, and if they are traceable to inadequate mathematical-set-organizations within *different structures*, then this raises questions about what relationship, if any, there is between these structures.

398

Broadly speaking, we might expect the relationship between $\mathbf{M^1L}$ and $\mathbf{M^2L}$ defects to be either: (A) one in which some structural influence outside the two respective structures themselves comes to adversely affect them both in comparable degrees. (For instance, if we accept that the $\mathbf{M^2L}$ and $\mathbf{M^1L}$ structures both use the same material substrate — on some sort of recursive-like basis, as suggested in Section C5.4 and its Table — then the common cause could well be some defect within this common substrate). Or alternatively (B), a defect at one of these levels might be instrumental in precipitating, maintaining, or exacerbating a similar defect in the other. In fact there is no obvious reason to rule out the possibility of both types of causality occurring — either in different patients for each type, or even both within the same patient. [So the controversy over "organic" versus "psychogenic" aetiologies (Chapman and Chapman, pages 147-148) could perhaps be a contest over a non-issue — some schizophrenics suffering from (say) generally-acting toxic effects within the substrate, some from the direct-or-indirect effects of misleading experience acting psychologically (see below), and some suffering from both.]

399

In the case of the psychogenetic causes, it has already been hinted here and in Section C8.1 that if the $\mathbf{M^2L}$ level has become defective to a socially-obvious extent, then it is probably some defect at the $\mathbf{M^1L}$ which is to blame — and not the reverse. After all, if the $\mathbf{M^2L}$ structures take as basic elements the schemata established by $\mathbf{M^1L}$ closure (as is supposed by the current theory), then any breakdown at the $\mathbf{M^1L}$ level will tend to erode the schemata on which the $\mathbf{M^2L}$ level

depends — either by directly disrupting these schemata, or more probably by simply failing to maintain and develop them properly. So if these various suggestions are correct, then such serious loss of abstract thought should either be thought of as a *secondary consequence* of more fundamental $\mathbf{M^1L}$ difficulties, *or else* as indicating some malfunction of the substrate common to both levels — or as a sign that the two different pathologies are occurring together.

There is also the likelihood of (C) a more involved *interactive* relationship between $\mathbf{M^1L}$ and $\mathbf{M^2L}$. Consider, for instance, the orthodox view of abstraction (Chapman and Chapman, page 144) as the ability to single out "a common property from a range of stimuli" on each of a number of objects. What mechanisms is this skill likely to entail? It rather looks as though the 400
individual needs to be able to manipulate sets of various types of schemata, to represent objects, their properties (here *reified* as if they were objects) and also actions connecting object-like things with object-like topologies. If such a task is to be feasible, one might reasonably suppose it essential to be able to organize these disparate entities in set-structures which stood higher in the $\mathbf{M^nL}$ hierarchy, than any of the entities directly involved. As the highest of the entities listed was what we would class as belonging at about the $\mathbf{M^1L}$ level, it would therefore seem to follow that any organizing set-structure would need to be at the $\mathbf{M^2L}$ level or higher. If this should happen to be true, it would go some way to reconciling the above "orthodox" view of the nature of abstraction, with the formal entailment of the $\mathbf{M^2L}$ level proposed here. But it also gives some indication of the possible interactive relationships which might exist between $\mathbf{M^nL}$ levels.

The possible choice of two or more different aetiologies for $\mathbf{M^2L}$ defects (perhaps also augmented by a volition-defect, which we will discuss next below, and maybe yet other causes) might shed some light on the diverse assortment of deviant behaviour which Goldstein[84] considered to exemplify *his* concept of "concreteness", and hence indicative of schizophrenia. 401
Thus Chapman and Chapman (page 148) write: "Goldstein's attempt to unite these diverse behaviours under a single principle tantalizes the reader with the hope that, if only he can penetrate Goldstein's true meaning, he will obtain insight into the essence of schizophrenic thinking." Whether the present account gets anywhere near this "true meaning", or an acceptable substitute for it, is a matter for current debate. Anyhow, the comment continues: "…Despite his emphasis on loss of conscious volition [see below] as the [unifying] core phenomenon of the concrete attitude, Goldstein described concreteness in such diverse ways that clinicians with widely differing views can all find it congenial. Almost any conceivable error can be interpreted as concrete."

The comment then goes on to point out that Goldstein denied the appropriateness of "intercorrelations between measures of the various kinds of concreteness". Insofar as the different types of symptom do have independent causes, on the lines suggested above, then this criticism of intercorrelational studies would seem to be well-founded; and as it is suggested at the finish of this work, there is often a tendency to expect too much in the blind use of such techniques. However it is probably overstating the case to claim that such tests could tell us nothing useful: the above arguments imply possibilities of *some* inter-correlation; and even a null 402
result would be quite informative, implying a need for independent therapeutic treatments.

(4) Loss of Volition or Conscious Control.

It will be recalled that, in Sections C6.3 and C6.4, a previous attempt was made to postulate what the formal structural organization of "consciousness" might be. Unfortunately, though not altogether surprisingly, the resulting suggestions were not particularly convincing in view of the

[84] Goldstein (1939, 1944/1964, 1959); Goldstein and Scheerer (1941, 1953); Goldstein and Salzman (1967).

sparseness of detail proposed,[85] so that it is difficult to test or corroborate the ideas with any precision, even if we are content just using *internal closure* criteria (see Chapter C1) — never mind any external experimental criteria of the more conventional type! However, for whatever value it might have in the present discussion, we may recall that it was supposed that two necessary conditions for consciousness were *attention* and *self-concept*, and that concepts such as *sentience* might conceivably be explicable in these terms.

Attention is discussed at the beginning of Section C6.3 (and in the third paragraph of Section C5.5)[86] in terms of a metaphorical fitting of a limited number of privileged schemata into "a room" or "control centre". The implication is clearly that some sort of extensively-defined set is crucially involved, that this set and its members have a temporary privileged access to the individual's ongoing activity, and that there is some comparatively rigid limit to the number of direct members that can be admitted within the "attention span"[87] at any one time (G A Miller, 1956a, 1956b; Pascual-Leone, 1970). So, whatever the details of the physical mechanism which presumably underlies attention, it seems likely that it may be subject to broadly the same sort of defect as the $\mathbf{M^1L}$ and $\mathbf{M^2L}$ mathematical-sets which we have been considering above — though not necessarily at the same time, nor necessarily at the same physical sites, nor necessarily for the same aetiological reasons. 403

Self-Concept is considered above in paragraphs 6 to 8 of Section C6.3,[88] where it is suggested that the most central aspects of it are probably at the $\mathbf{M^1L}$ level, though there is also a case for placing them within the $\mathbf{M^0L}$ level — or perhaps some unique amalgam of both. Whatever the details, one's self-concept or *ego* or *personal identity* is likely to be a much more involved construct than the schemata for ordinary external objects; but it still seems plausible, as far as we can see at this stage, that this complicated self-concept will nevertheless consist ultimately of a basic extensive-set schematic structure of physical molecular elements — essentially similar to that postulated for other extensive sets. 404

Putting these two supposed components of consciousness back together, we might reasonably expect that the total phenomenon will still be describable, at least in principle, in terms of the various mathematical-set structures which we have been discussing. Of course there is also the further complication that an exhaustive model would have to account, not only for consciousness itself, but also for the way in which it *controls* ongoing activity: more-or-less correctly in normal individuals, or inadequately in individuals behaving according to the Goldstein formulation of Schizophrenia *as* a loss of conscious volitional control. However there is no obvious reason why the additional feature of a control-link should make the basic postulate of molecular set-structure untenable, though the complications implicit in this problem are formidable:-

Thus we may recall the topological dilemma concerning where consciousness should belong in the $\mathbf{M^nL}$ hierarchy; (Sec C3.2/paragr 7, and Sec C6.4/paragr 4).[89] Although no fully detailed account of likely mechanisms emerged from these deliberations, they did at least produce a logical solution to the immediate dilemma by placing consciousness, as such, in a category of its own — standing aside from the linear hierarchy of the $\mathbf{M^nL}$ scale. It thus seemed to occupy a potentially privileged position of relatively easy control-access to schemata of all levels, on average; though to the extent that there exists a body of subconscious and unconscious thoughts, 405

[85] It is hoped that future theoretical investigation will remedy this deficiency.

[86] Original pp.224-225 (see margin): "[…]", which also invokes Section A1.4.

[87] One might profitably enquire whether each $\mathbf{M^nL}$ level might have its own private attention-span, and explore complications raised by this.

[88] Orig.pp.246-247: "The second point … Piagetian description."

[89] Respectively: Orig.p.172 "As for consciousness…" —— and O.p.265 "Section…"

it will be obvious that this privilege is certainly not total. Anyhow, to mark this supposedly exceptional status, it was suggested that consciousness be considered as being in an exceptionally-named level: the $\mathbf{M^{top}L}$ level.

So what then should we make of Goldstein's notion that "loss of conscious volition" is, or should be, the defining principle for identifying schizophrenia. From a strictly practical point of view, this is most unhelpful "because its presence or absence is almost impossible to judge" (Chapman and Chapman, page 149). And even as a theoretical basis there is precious little, at this stage, that one could do in the way of structural development if one were committed to this definitional dependence on "consciousness" as the fundamental construct. Not that the existence of such potential inconveniences necessarily means that Goldstein is wrong; though we might well pray that he is, because if he is right then there will be a great deal of theory for us to clarify in considerable detail before we are likely to have a reasonably accurate insight into the nature of schizophrenia. Nevertheless it is hoped that we do not reject his ideas merely because of wishful thinking in this direction!

But even if he *is* right, it still seems likely that any deeper, more-micro, level of explanation will involve disturbance of one or more of the brain's postulated extensive-definition set 406
structures; and it remains possible that whichever of these structures happen to be faulty in any given type of schizophrenia the faults will all fall within a reasonably circumscribed range of types of structural error afflicting essentially similar mechanisms, (at the molecular level, or whatever).

Finally it is perhaps worth considering that if Goldstein does happen to be wrong on this point, and loss of volitional control is merely a secondary symptom, then its logical status is likely to be much the same as that postulated for logical and abstract thinking in the immediately preceding discussion: item "(3)". In this case, the comments made there concerning causal chains from $\mathbf{M^1L}$ deficiencies to $\mathbf{M^2L}$ symptoms, are likely to apply comparably to causal chains from $\mathbf{M^1L}$ to "$\mathbf{M^{top}L}$" — or wherever we finally decide that consciousness should properly be located in the functional topology.

(5) A Negativistic Attitude, or Paranoid Tendencies.

It has already been argued in Section C7.7, that paranoia is primarily a *neurosis* rather than a psychosis, but that its nature is such as to encourage a certain amount of disorganization within some of the mathematical-set structure acquired by the brain, thus tending to produce some psychotic symptoms. (This question of *causes* of set-deterioration will be discussed below in its own right). But, as with many syndromes of this general type, it is quite on the cards that there 407
was actually a small amount of unwarranted and socially disadvantageous set-disorganization in the first place — along with a small amount of neurosis — and that the two tended to exacerbate each other in a positive feedback situation, so that the collective behavioural syndrome eventually escalated until it became socially evident. In such a situation, it is not a clearcut issue to attribute causality; in fact it constitutes an example of the "chicken and egg" dilemma. And even if the neurosis was indeed the root cause initially, it is likely that it would have passed unnoticed in many cases if there were no supporting feedback loop operating subsequently via psychotic disruption. In other words, it may not always be possible or desirable to make a complete logical distinction between the two types of disorder.

Accordingly it is perhaps sensible to include this negativistic syndrome under both headings: neurosis *and* psychosis. At any rate, it does often appear in the psychosis-category; and its symptoms are offered as correlating with a diagnosis of schizophrenia. Indeed this seems to be the main characteristic of the patients discussed by Laing (1960/1965, 1961/1971), as pointed out earlier in Sections C6.3 and C7.7. Thus if social "objects" such as public institutions come to be *seen* as malevolent, then the individual will tend to reject such "objects" in the apparent-cause of

bolstering his own self-identity schema. Such withdrawal of cathexis will sometimes take on an aggressive form such as diminished-cooperation, like Kraepelin's (1919) patient who persistently answered numerical questions with answers which all seemed to be deliberately wrong — a phenomenon described by Bleuler as "intellectual negativism", (Chapman and Chapman, "evasion" page 9). Work propounding this viewpoint of the basic nature of schizophrenia includes Sullivan (1924, 1925, 1944/1964) where the patients are seen as having lost the motivation to communicate, and Haley (1959) where the concept of "motivated retreat" is used — as in Laing (1960, 1961); Chapman and Chapman, pages 228-230)

408

C8.3 Some Comments on the Accepted Dimensions for Classifying schizophrenias and Related Conditions

Elsasser has suggested that the crucial difference between physics and biology lies in the fact that physics deals with mainly-homogeneous systems, while in biology it is inevitable that the systems be very markedly "inhomogeneous". This inhomogeneity has been traced down to molecular level by experimental procedures for some aspects of biological function, such as genetic inheritance, though not in others such as memory — at least not in adequate experimental detail. Nevertheless it has been a principal objective of the present work to offer credible suggestions as to how memory *could* be accounted for at the molecular level, in terms of a very considerable degree of inhomogeneity — though not to the extent of leaving it in complete chaos.

409

But as all too often occurs in practice, there is a price to be paid for progress. To the extent that our model of the brain becomes more inhomogeneous in its basic micro-structure, the more we should come to suspect the validity of those modelling techniques which we have come to value for homogeneous systems such as "the perfect gas", or the civil engineer's "three-dimensional space". After all, even the real gases encountered by the physicist are not really "perfect" and under some conditions they produce "behaviour" which is not at all in accordance with Boyle's law — thus leading to seemingly arbitrary artefactual models such as the cubic equations of van der Waals. In fact it is not until we enquire deeply into the basic structural nature of gases that we can make reasonable progress towards removing the appearance of arbitrariness which the (modified) cubic equation presents. In a similar spirit, we might well be wary therefore, of blindly transferring the mathematical assumptions of simple-paradigm behavioural psychology down into the realm of the molecular elements which are, according to the present theory, the basis of inhomogeneity underlying psychological phenomena.

In particular it may be doubted whether psychological systems can really be adequately and meaningfully expressed by "dimensions" in a Cartesian-coordinate system. It would appear that this assumption is commonly made, and moreover made quite unconsciously (as if the successful use of this conceptualization in applied physics were sufficient guarantee of its universal applicability!) This belief in the applicability of Cartesian coordinates underlies many of the experimenter's statistical tools, such as multiple-factor analysis, so the question is of some general relevance. Indeed the results of such procedures are unquestionably often technologically useful — just as Boyle's law is often technologically useful. There may even be some discoverable structural basis for the constructs inferred and named on the basis of such analyses (Cattell, 1965), though the technique itself will not be much help in elucidating what that structural basis might be. However, one might perhaps feel that the method assumes away many of the interesting problems, so that any findings which do eventuate will merely have scratched the surface — leaving the "real workings of government" well hidden.

410

The sort of structural effects which are likely to conspire against this approach include: internal feedback and delay, internal interaction between elements or parts of the system, and effects arising from the fact that the system is actually made up of heterogeneous discrete elements rather than a continuum. Now the interesting thing is that there is a good chance that, in

principle, one could by-pass these difficulties by using a finer "level of resolution" into a large number of subsystems, to such an extent that *none* of the complications just listed are *internal* any more. In other words, one can hope for a reductionistic Utopia in which each individual *sub*system is free from complications like internal feedback, and whatever feedback there is will 411
be *between* such subsystems. Experimentally though, this will probably not be much help if these subunits are too small, too delicate, and too numerous to be measured sufficiently; and even if we can infer the basic structure by other means, we may then still have huge problems in turning such information into a usable model — though we might, if we are lucky, find a special trick of the trade such as "Statistical Mechanics".

Having said all that, let us re-examine some of the "dimensions" offered in the literature as being indicative of schizophrenia. And, more to the point, let us try to see past the orthodox behaviouristically-oriented formulation and comment on the likely structural basis for the dimensions in question.

Process/Reactive

The *Process-Reactive Distinction* is discussed in Chapman and Chapman as probably constituting more than "merely two end-points on a continuum of premorbid adjustment" (page 334). The *Reactive* type is likened to Kraepelin's manic depressive (page 28) and is seen as having a good chance of recovery, while the *Process* type of patient is likened to Kraepelin's dementia praecox as a "typical" schizophrenic with: "inadequate prepsychotic adjustment, with little interest in other people or the activities of life … develops gradually … with no identifiable precipitating stress. … usually … affective flattening, … prognosis is poor, … deteriorating course".

412
Without attempting any very detailed analysis of these distinctions, some preliminary observations on the basis of the current theory might assist us to orient ourselves to the issues likely to be involved. One impression about these latter symptoms is that they seem more likely to have their origins deep within the personality structure of the individual, with significant components from experiences in early life, or in inherited biases within the mathematical-sets' substrate, or both. Another impression is the likely importance of experiences involving affective relationships, or the genetically-determined nature of whatever structures serve to "encode" affective relationships (whether these be "tag"-involving procedures as has been suggested here, or some different arrangement).

As a tentative working hypothesis therefore, it might be useful to think of Process Schizophrenia as stemming from the individual's failure to acquire *stable* object-like *socially-oriented* structures, at-or-about the Sensori-motor stage ($\mathbf{M^0L}$); — or as having acquired them without the "appropriate" affective affiliations (which might also have a direct or indirect bearing on their ultimate stability). We might then wish to distinguish between ▪ those cases which arose genetically, ▪ those generated through damaging experiences (of different sorts, perhaps), and ▪ those with various admixtures of these two influences. Behaviourally speaking though, there might be little difference between such categories.

413
By contrast, we might suppose that Reactive Schizophrenics suffer from comparable disruptions at some higher $\mathbf{M^nL}$ level, probably the Concrete Operations ($\mathbf{M^1L}$) level, which would have presumably developed later and be more amenable to readjustment — as well as being less far-reaching in their consequences. whether or not genetic components are likely to make much contribution toward Reactive Schizophrenia would seem to depend on whether the relevant parts of the two respective $\mathbf{M^nL}$ levels operate within substrates which are both subject to the same genetic influences. Thus if both levels are likely to suffer the same genetic disruption, then presumably the Process-symptoms will pre-empt any would-be development of Reactive symptoms; so anyone diagnosed as simply Reactive would presumably be free from any

such *generally-acting* genetic defects — except perhaps in an auxiliary role. But of course, if each **M^{n}L** level operated within a substrate which was genetically independent in origin (with respect to mechanisms relevant to our present discussion), then we might well expect to find occasional genetic components in any pre-disposition to Reactive Schizophrenia or manic-depressive personality, (and without them also promoting Process Schizophrenia at the same time).

Hebephrenic/Paranoid

The *Hebephrenic-Paranoid Distinction* is implicit in the comparative properties discussed above in Section C8.2, in the two items "(1) and (5)" respectively, or indeed in the special *supposedly neurotic* affiliations of the latter "paranoid" item *vis a vis* any of the other four; — and this developed a theme introduced earlier in Section C7.7. 414

Chapman and Chapman make some interesting comments concerning this supposed-dimension:

> "Although the distinction between paranoid and nonparanoid patients accounts for some of the heterogeneity of performance among schizophrenics, the distinction is not clearly one of different disorders. As Kraepelin himself pointed out, few patients fit unambiguously into one subtype, and even those who do *may shift from one symptom picture to another* … A patient may, for example, initially show predominantly paranoid symptoms, and later show predominantly hebephrenic or catatonic symptoms. Such changes indicate that these symptoms represent different stages of the same disorder rather than distinct disorders. Not all patients, however, shift symptom patterns". (Page 30, emphasis added).

Such a dynamic evolution is clearly within the spirit of the above-mentioned danger of progression from a neurotic beginning into more-psychotic states. Whether these successive stages are to be regarded as representing "the same" disorder or not is perhaps a little beside the point and a matter of mere semantics; the crucial issue is to decide what the essential cybernetic dynamics are likely to be, and not to try to clothe such phenomena in a terminology which implies static configurations.

"Progress on this problem is hindered both by the dearth of theory to guide the research and by the failure of research workers to use objective and reliable criteria for diagnosis of the paranoid subtype." (Chapman and Chapman, pages 333-4). The current theory might perhaps help to fill this breach, or to inspire other theories which do; but of course that remains to be seen. 415

Acute/Chronic

The *Acute-Chronic Distinction* is discussed by Chapman and Chapman (pages 321-331). Changes in symptom patterns over time are hardly surprising if the brain is viewed as a dynamic and more-or-less self-organizing system, as we have just seen in the preceding comments about paranoia. In such a system it is likely that an abnormal operation of set-structures at one **M^{n}L** level, or even just an isolated part of such a level, will sometimes disturb the consolidation or maintenance of set-structures elsewhere — bringing the possibility that there will be *eventual* profound changes in the overall system. (A useful rough analogy is offered by consideration of the long-term economic consequences of a substantial increase in the price of oil!) In some cases the progression may be propagated through largely internal processes, such as those postulated for the paranoid's decline into hebephrenia; in others the change may be largely brought about by external influences such as hospitalization or other institutionalization influences. But from a strictly cybernetic viewpoint, it is not necessarily important to distinguish between such *internal* and *external* mediators of the exacerbating feedback loops; rather we should ask which loops can be broken through our "autonomous" intervention. Thus we might, because of socio-political reasons, be powerless to change the "external" influences of hospitalization, and yet we might conceivably be able to stop the progression by medication — though whether this should be called "internal" or not is a moot point, and it constitutes another semantic issue. 416

Such chronicity should, of course, be taken into account; and it has considerable practical implications. But it looks as though it should properly be regarded as merely secondary as far as symptom-analysis is concerned, and not a particularly informative basis from which to try to derive a useful fundamental "dimension". Of course if we were to get beyond a mere consideration of "time served" and look more deeply into the apparent changes of state in the various subsystems within the total system, then this would be real progress (and probably Utopian progress at that!); but it is questionable whether such information could be conveniently fitted onto one linear dimension, or perhaps even onto *any* Cartesian coordinate space.

Two other separate criteria, arising out of Goldstein's overly inclusive category of "concreteness", have been proposed as dimension-like dichotomies [90] by McGaughran (1954), and McGaughran and Moran (1956, 1957) — as outlined by Chapman and Chapman (pages 156-8). 417

Public/Private

Of these, the *Public-Private Distinction* might plausibly be attributed to the *acceptance* of the conceptual categories used conventionally within their culture, in the case of non-paranoid subjects, or *rejection* of such conceptualizations in the case of paranoids — either as an aggressive gesture against society in retaliation for its supposed malevolence, or for some arbitrary reason which has resulted in the past in a "malevolent" response from society, thus engendering the feeling of persecution lead to the paranoid state. Anyhow the findings of McGaughran and Moran (1957) and of Silverman and Silverman (1962) found a correlation of "privateness" of response with "paranoid-schizophrenia" diagnosis which tends to support this view of their common origin.

Open/Closed

The other dimension-or-dichotomy arising from these studies is the *Open-Closed distinction* which refers to the use of *open*-ended categorizations versus *closed* unique "categories" which tend to constitute trivial one-member sets. The latter one-member tendency turns out to be associated with brain-damaged patients rather than schizophrenics — those whose sortings under a test were of the type that Goldstein described as 'stimulus-bound', or as an excessive response to the 'immediate experience of the given thing or situation in its particular uniqueness'." From this, and from the typical responses given, there is some basis for believing that these effects in brain-damaged patients are symptoms of an underlying defect in which some of the ability to 418 place objects into conceptual mathematical-sets has been erased completely; so that the individual makes do with individual items instead of the sets which might have contained them, and even for these single items he is dependent on the support of perceptual input due to the presumed shortage of adequate set-structures to constitute the normal attention-span.

By contrast, those particular types of schizophrenic which were used in these studies did seem to be able to apply concepts like "redness" to the objects which were presented to them, and therefore presumably they were able to form. mentally the mathematical-sets entailed in this type of classification. It is less clear whether or not these subjects had other well-defined set-forming aberrations, such as difficulty with $\mathbf{M^2L}$ control of such activities or whether their performance with solid objects is matched by a similar competence with "social objects". It might perhaps be worth investigating the possibility that in the brain-damaged patients, there is a more uniform disruption across all types of mathematical-set activity at all levels, while those with experience-based schizophrenias have defects centring on that particular aspect of set-organization which is 419

[90] Despite McGaughran's suggestion that these should be seen as continua, it is arguable that this view is only applicable to experimental measures, and not to underlying phenomena; or even if they do represent some statistical reality, there is nevertheless a discrete basis to them at the micro level.

most related to the schizogenic experience. Or else, looking at the question more generally, perhaps all we should postulate is that there will be a *different distribution* of set-handling deficiencies, without attempting to specify in advance just what the respective distributions should be; though from our present structure-oriented point of view this rather amounts to admitting temporary defeat! But of course our "intelligent guesses" at structure depend on there being some important and coherent rationale as a historical background to existing mechanisms — and arbitrary brain damage is unlikely to have much relevant rationale behind it! 419

Attention focus

The Narrowed-versus-Broadened Attention Distinction — and related effects such as over-inclusion and distractability. The literature on this type of symptom is reviewed in Chapman and Chapman (Chapter 13, pp 253-285), and some of the theoretical issues have already been touched on in the present work.[91] However our present aim is not so much to encompass the whole field as to test the potential explanatory power of the current structural theory in isolated typical cases, and in particular in those situations where existing explanations have the most difficulty in accounting for the facts (even if, in the end, their explanations do seem to be correct). Accordingly we shall confine our attention here to the comparative studies and interpretations offered by Broen (1966) which are discussed in Chapman and Chapman (pages 278-280). ;

Let us look first at Broen's concept of "*response disorganization*" and re-interpret it in terms of the current structural theory. Consider that the individual will generally need to discriminate between *"dominant" responses* (which are, in some sense, to be considered as appropriate) and *"competing" responses* — represented presumably by schemes or schemata which are physically available, but are *not* appropriate in the above sense. When stated in this way, it becomes easy to identify Broen' s *dominant responses* with member-schemoids *listed in an appropriate set-structure* and thus having "extensive definition". This then would seem to be the relevant organization, and its absence would then be the "response disorganization" envisaged by Broen. The practical result of this deficiency would indeed be expected to be "that the frequency of the various response alternatives become more nearly equal for schizophrenics than for normal subjects". 420

In the present work, we have often come across the idea that there are likely to be many categories of set-structure: notably between $\mathbf{M^nL}$ levels, but also probably for various different areas of activity (presumably also corresponding to different physical areas within the brain, though not necessarily distinguishable in macroscopic anatomical terms). Anyhow it would appear that Broen makes at least some distinction of this sort by singling out *"focusing responses"* for special consideration as what amounts to the operation of a particular set-structure designated for *attention-holding* purposes. By way of contrast, the interpretation which follows below will envisage at least *two* relevant organizations for attention — corresponding to $\mathbf{M^1L}$ and $\mathbf{M^2L}$ thought processes respectively. 421

Given certain circumstances which we will come back to shortly, the patient's attention is said to become "narrowed" — thus "reducing the range of stimuli they observe" down to those with the strongest "pull" or saliency. A promising structural interpretation of this progression is as follows:- The patient's ability to comprehend *objects in extensive sets* is diminishing, due to some breakdown in set-organization in the relevant part of the $\mathbf{M^0L}$-$\mathbf{M^1L}$ interface, leaving the patient to do the best he can with the primitive techniques of *intensive-definition* calling of schemata. Consequently we might well expect that the actual object-schemata which actually reach attention will be those "shouting the loudest", so to speak — which probably means either

[91] Section C5.5, para 3: o.p.223- "[…" — Section C8.2(2) para 4: o.p.386-387 "Looking next…" — and Section C8.2(4) para 2: o.p.402-403 "Attention is…"

that their elemental molecular representatives are in a majority in some sense, or that sense-impressions from them have some sort of monopoly over the sensory-input channels. Accordingly the patient will tend to fixate on these "salient" items, having lost the ability to give a balanced consideration to the full range available to him in the real external world; or to put it in Piagetian terminology, his attention will no longer be "decentré". moreover, when the patient does shift his attention to some other item such a move is likely to follow syncretic associations rather than "logical categories" . 422

In other sets of circumstances, also outlined below, there is a loss of the patient's apparent ability to guide himself to the use of "appropriate" criteria when sorting objects. Instead he will seemingly be content to make indiscriminate use of *any* attribute which he happens to light upon as being common to some of the objects, including such features as "shadows or scratches" which normal subjects automatically assume to be irrelevant. (This assumption of irrelevance *could* have a social-norm or formal-logic component; but it is perhaps more likely that it reflects a basic $\mathbf{M^0L}$ awareness of the mathematical-group properties of *objects* as such, and therefore a feel for what is not-real-object but mere sensual artefact like shadows and perspective — accessory details which the $\mathbf{M^1L}$-stage artist will sensibly ignore until perhaps he acquires the $\mathbf{M^2L}$ sophistication which enables him to rationalize their inclusion again for particular purposes).

Anyhow it would seem that, although the patient is apparently able to form mental extensive-sets of objects (as long as other pathologies are absent), he is *not* keeping proper extensive sets for some other feature of the task. This other feature could be the *properties* of objects including their mathematical-group-like nature, suggesting that the patient's mental encoding of objects must be tending to become mere empty ciphers devoid of genuine object-like characterization; — the name or form only, without the substance. Alternatively the phenomenon could be due to the loss of some set-structure which would normally provide guide-lines as to which would-be characteristics of objects are worthy of attention. On the face of it, this seems less likely — except insofar as *social* conventions may be a component in the choice of "appropriate" features. 423

We may now try to apply these formulations to the first of the two dilemmas which Broen considered, but concentrating on the non-paranoid patients for whom the relevant effect was greatest (as we might expect on the theoretical grounds outlined earlier). The dilemma lay in the apparently contradictory findings of Payne (1962) and Chapman (1961), because while Payne found more "overinclusion" amongst his *acute* patients, Chapman found more of *his version* of "overinclusion" in his *chronic* patients. (Broen himself suggests a solution to this dilemma using a somewhat different conceptual approach. It can plausibly be argued that his explanation is formally compatible in its essentials with the one given below; but that is an issue which we will not pursue here in any depth).

Payne's concept of "overinclusion" amounted to his subjects using the very "shadows or scratches" which we have just been discussing. Such a "response disorganization", arguably in the form of a loss of appreciation of the "objectness" of objects, is in keeping with the sort of defect that one might expect from a non-paranoid schizophrenic — if the current theory is on the right track. There is, however, no obvious reason why such symptoms (and their underlying mechanisms) should diminish over time as originally-"acute" patients turn into "chronic" patients". On the other hand though, it is quite plausible that this condition's underlying mechanisms might remain more-or-less constant and yet the manifest effects arising from them might disappear somewhat with chronicity due to the masking action of some other development. That, in fact, is the essential point made in Broen's explanation, and he identities the new development as the "narrowed observation" with which we began about four paragraphs ago. 424

By contrast, Chapman's (1961) version of "overinclusion" involved the gratuitous addition of items which were approximately correct by virtue of some sort of association. Such errors can

reasonably be identified as syncretic thought, lacking the set-structure guidance of reliable extensive-definition of object sets, and so indicative of one form of mental decline. Not only that, but it is not difficult to imagine that such a deficiency could pre-empt the manifestation of Paynes's type of overinclusion — though the argument is scarcely clearcut enough to actually make reliable *predictions* of this sort. (That, of course, is unfortunate; and it is to be hoped that future developments will remedy this situation).

425

The second dilemma investigated by Broen (1966) was the apparent disparity between the findings of Venables (1964) and Chapman (1956). According to Venables, chronic schizophrenics attend to a range of stimuli which is narrower than normal; and of course that is very much what we might expect from the theory of schizophrenia adopted in the present work, and the discussion of six paragraphs back in particular. However Chapman (1956) had found that the same general category of patient (chronic schizophrenics) were more distractable than normal subjects, and this seemed to imply that they therefore had a wider span of attention than the normal. In retrospect this might perhaps be seen as a semantic dispute over the term "wideness of span":- Does it mean the wideness of a narrowly-focussed but highly-rotatable searchlight beam? *Or* does it mean the wideness of a parachute-flare? Anyhow Broen offered what seems to be a very credible explanation along these lines, attributing the different experimental implications to the centripetal and centrifugal effects of the more salient items in the two respective cases; and such an explanation would appear to be entirely compatible with structural accounts of syncretic thought (in default of extensive set-structure) as expounded here.

In closing

Some closing comments about the strengths and weaknesses of such explanatory exercises are, perhaps, called for at this stage. Attention is (apparently) a phenomenon of some considerable complexity; and the current theory, with its emphasis on potentially-detailed substructure, is likely to have a lot to say about complex phenomena. Indeed the trouble is that it is likely to have too many possible explanations to offer, and the difficulty will then be in deciding which ones, if any, are likely to be correct — and in what combinations, and under what circumstances. In other words, we have a situation in which the stock criticism "It explains everything but predicts nothing" might be partly justified. Nevertheless we should perhaps be content *for the moment* to have a theory which *does* tend to "explain everything" — that is, one which offers too many plausible structurally-based solutions rather than none at all. However, to the extent that it is true that such explanations "predict nothing", this would be a clear indication that further work and elaboration is called for to fill in structural, qualitative and quantitative details of the theory and its implications.

426

C8.4 Aetiological Issues: By what mechanisms are Psychoses likely to Originate?

It is clear that there are at least two different categories of cause for schizophrenia: "Anybody who at this stage of our knowledge takes a wholly genetic or wholly environmental view of aetiology is clearly in error" (Venables, 1975); so obviously any theoretical approach to the problem must be compatible with both. Moreover it seems likely that there are also different sub-categories within each of these two areas: different parts of the presumed structural organization which are affected differently by *different genetic* factors, and also various qualitative types of initial disruption to set-organization which are likely to arise from *different experiential* anomalies. Any truly detailed explication of these causal chains will probably have to await a detailed exposition of the underlying mechanisms, be they molecular or whatever. But meanwhile, we have now developed the present theory sufficiently for us to be able to indicate the likely broad outline of the processes leading to psychoses in general, and those relatively enduring psychotic states which we include under the term "schizophrenia" in particular. The experience-based aetiologies seem to be the most difficult of these to conceptualize, so we shall

427

leave them till last; but the others can now be outlined quite briefly, given the conceptual framework which has been elaborated in the present work so far. Let us start with the schizomimetic effects as discussed in Chapman and Chapman (Chapter 15):-

Drugs.

For any theory which postulates that crucial activities are being carried on at the molecular level, it is patently obvious that drugs are likely to produce specific disruptions to particular parts of the proposed mechanism Offhand, for instance LSD and Mescaline might both stop the efficient activity of at least some of the extensive-set mechanisms — though arguably there would be a differential impact on different categories of such mechanism. Then maybe Sernyl 428
somehow interferes with the proper operation of the supposed "tag" effects, leading to feelings of unreality or depersonalization. Likewise perhaps Amphetamine has an impeding effect on the schemata related to the ego, thus promoting a predisposition toward paranoid feelings. (These illustrations are offered merely as impressionistic examples, and should not be taken too seriously as they stand). Anyhow, given the present conceptual framework and a measure of good fortune, systematic investigations along these lines could conceivably be highly rewarding.

Brain Damage.

The present work has been primarily concerned with an ultra-micro view of the brain's supposed activities, but clearly there are also meaningful things to be said about the same system when viewed in grosser terms (just as there is value in viewing aspects of economic phenomena both at the level of the individual *and* at the level of the firm — or industry, or nation). However our more macro pronouncements are likely to make better sense if we have a proper understanding, of the micro-phenomena, which are the basis of the macro-phenomena; — and having made better sense of the macro data we will then be in a better position to use it to shed further light on the micro-phenomena. In other words, there is room for profitable mutual cooperation between these two viewpoints.

Thus the concepts involving intensive and extensive set-defining mechanisms postulated 429
here, offer some new dimensions to the interpretation of the significance of brain lesions and any tendency there may be for the patient to recover from them; while of course brain-lesion data is very useful in clarifying some of the "geographical" mathematical-set-tendencies within the brain, and also its behavioural consequences provide useful benchmarks against which schizophrenic behaviour patterns can be usefully compared. If, for instance, we can identify the behaviour patterns of brain-damaged patients in terms of set-structure failure (and this seems likely), then it is likely to be an informative exercise to account for different distributions of the *types* of such failure in the two cases — with a reasonable chance of thereby shedding some light on the way the brain system is organized, and what aspects are most vulnerable to the different types of disruption.

Sleep Deprivation.

Section C6.6 of the present work suggests that sleep may constitute special consolidation-periods in which set-structures are vetted for their "internal closure", and that the coherence or "sanity" of surviving mental configurations is therefore likely to depend on this sleep activity. Accordingly we should not be surprised if sleep deprivation produces symptoms which resemble the more permanent ones of schizophrenia. Moreover we can reasonably go further and suggest that a selective deprivation of orthodox sleep (if that were possible) would selectively impoverish 430
the $\mathbf{M^0L}$ level initially, while selective deprivation of REM sleep will preferentially upset the $\mathbf{M^1L}$ coherence. As an interesting further speculation we might consider whether *meditation* or "time to collect one's thoughts" might serve the same sort of function for the $\mathbf{M^2L}$ level of logical thought; so perhaps such "high pressure" treatment as *Speeded Performance* could be expected to

produce psychomimetic symptoms in the form of a trend toward non-logical Primary Process thinking. Flavell *et al.*'s (1958) findings might conceivably be interpreted in these terms, though of course it is unwise to make too much of such an analysis *post hoc* — and one should rather design new experiments specifically based on the new conceptualizations. (Chapman and Chapman, page 317).

Sensory Deprivation.

It has been part of the general philosophy of the present work that the development and maintenance of useful set-structures within the brain (like useful concepts in science) depend on two complementary processes. These are the testing of *internal* consistency (as just discussed in connection with sleep), and secondly the testing of the *external* consistency of one's mental structures *vis a vis* reality, by means of a more-or-less continual interaction between these domains. Clearly any prolonged sensory deprivation will interfere with this latter process and therefore, according to our theory, some progressive deterioration of some aspects of mental 431
organization are likely. (This indeed may be taken as a special case of a more general biological principle that organization tends to depend on the relevant structure actually being used. For example, an unused limb will tend to atrophy or develop inappropriately — apparently because its development and maintenance actually depend on the cues given by the stresses of life interacting with the system). Anyhow, suffice it here to say that such schizomimetic symptoms arising as a consequence of sensory deprivation would seem to be very much compatible with the current theory.

Incidental Minor

Other schizomimetic-inducers could be interpreted as acting through some aspect of the brain's overall set-structural organization *other than* the actual set-structure in question. In other words, it looks as though the self-stabilizing activities of this focal schema are specifically disrupted by the interfering "exogenous" action of some other schema within the total system. It will suffice for our present purposes if we merely identify the conditions which might plausibly be operating in this way, and also suggest the likely route of their presumed intervention.

- *Relaxed Attention* is produced by conscious intent, so we may presume that it is the complex ego-schema which deliberately lets certain set-supporting activities lapse, temporarily.
- In *Distraction* there is a (voluntary *or* involuntary) overload on the attention, thus disrupting its proper function; and the source will presumably be the intrusion of irrelevant stimuli and their 432
internal coding — unless perhaps we choose to blame the ego-schema again, for the "voluntary" situations.
- *Hypnosis*, insofar as we understand it at all, does seem to be exactly such an intervention — instigated and controlled by that other special schema representing supposedly-social demands: the superego.
- *Operant Training* may be devised by the experimenter to produce deviant responses; and this presumably operates through normal "ordinary" set-structures — and probably via essentially commonplace mutations to the focal set-structure itself, or to some other such structure normally instrumental in maintaining it.

Perception manipulation

Disruption of Perception such as Aaronson's (1967) use of posthypnotic suggestion to induce the subject to believe that the normal "unquestionable" laws of nature had ceased to operate. Such a situation (which did in fact produce psychotic symptoms) would presumably have been seen by the subject as unwelcome evidence that he was losing his control over happenings in the external world. It thus turns out to be (subjectively and temporarily) a case of a general experiential arrangement which *does* lead to schizophrenia, and which rather looks like being the

main-or-only experiential cause of schizophrenia. We shall come to this issue almost immediately, and conclude this section on that note; but first let us deal with the question of inheritable pre-dispositions toward schizophrenia:- 433

Genetic Factors promoting Schizophrenia.

When an engineer sets out to design an aircraft or a bridge, he will have to assume the competing demands of maximal strength versus minimal weight-or-cost — and probably other troublesome variables as well, like pollution-rate, resonance-frequency range and specificity, wind-resistance characteristics, and so on. If the final project is to be viable, taking the total existing conditions into account, then the "dimensional space" into which the design must fit will be comparatively small — and probably very much more restricted than the naive commentator might expect. Similarly, if the body is to avoid undue risk of infection and yet not have such an active immunological system that it destroys its own useful structures, then there will be a definite limitation to the range of acceptable "designs" for such a system; (though in practice here, the "design" will be by the rather uncomfortable process of natural selection).

It is not necessary to know all the details (about how molecular-based set-structures might operate) for us to realize that such a system is bound to have many aspects where the details of adjustment have to be "pre-designed" in much the same way. Thus it will be no good to the individual if his set structures are so stable that no new evidence can make any impression on these structures (nor *find* an appropriate mutant version); but on the other hand the individual will also come to grief if his set-structures are so adaptable that no encoding of experience-based discoveries can be relied on to retain its information unchanged. The compromise required here is presumably between over-creativity and ultra-conservatism, but the basic principle of finding the proper balance between conflicting demands appears to be the same. Moreover, in the absence of "genetic engineering", the acceptable range will have to be found and maintained by the same unfair survival-of-the-fittest principle as that applying to immunological systems — though perhaps we are now talking about the survival of "mental health" rather than life itself. However the settings of these values will only "set the stage"; and actual "mental survival" will also depend on experiential factors:- 434

Schizogenic Environmental Conditions.

It will be helpful to start by comparing some of the likely properties of ordinary mental schemata with those of the special ego schema. For one thing, the ramifications of the ego will almost certainly be much more far-reaching than those of any other schema; and this presumably amounts to saying that the ego-structure will be very much "bigger", in terms of membership and internal-closure-loops, than any other mental schema. Thus, if the individual has managed to assemble any recognizable ego-set at all, then the mutual corroboration amongst the closure loops of this complex set-structure will probably make it substantially more *stable* than any other mental schema. So on primarily structural grounds we have some reason to believe that the ego-schema has a privileged stability status, and that is likely to mean that responses to its attempted disruption will appear to be qualitatively different from similar attempts on the integrity of other existing schemata. 435

If we look at the same issue from a functional viewpoint, it seems reasonable to regard the ego-structure as the *sine qua non* of a humanoid being's mental organization. In other words, like the King in chess, its integrity is equated with that of the system as a whole and cannot be regarded as negotiable, whatever the price. (Not that the brain would have been assembled according to any consciously argued "design", but presumably nature has had to keep trying until the appropriate arbitrary mutants present themselves for selection). So, on these grounds too, we

might expect the ego to be especially protected from any serious threat to its essential integrity, even though other ordinary set-structures might come to be radically altered or superseded.

In this context, it is interesting to consider Masserman's (1971) view that psychoses (and other mental dysfunctions) arise from particular types of uncertainty concerning the environment — and the individual's own ability to cope with it:- "Doubts and trepidations about our capacities to predict and cope with impending and important events induce the internal physiological signals we interpret as 'anxiety', and variably actuate the symbolically evasive (phobic), hopefully repetitive (compulsive ritualistic), regressive-dependent (depressive), reactively overassertive (paranoid), dysaffective and dereistic (schizoid), and other individualized attempts at mastery or denial which, depending on the extent and duration of their deviation from current cultural norms, are then labelled 'ideosyncratic', 'neurotic', 'sociopathic', or 'psychotic'". 436

Moreover we can get a clearer insight into the type of uncertainty which is involved here. The account given by Kimmel (1971a) traces the laboratory evidence on this effect to Yerofeeva (1916), and especially to Shenger-Krestovnikova (1921) whose experiment, with dogs, entailed a learned discrimination between circles and ellipses. Uncertainty was introduced in the latter case, by progressively using ellipses which were more-and-more circle-like — thus presumably eroding the dogs' confidence that they could really cope with a discrimination which they thought they had mastered. Anyhow the result was: "a neurotic-like breakdown without the use of any aversive stimulation whatever". One might perhaps quibble as to whether this might actually be classed as an aversive stimulus, though obviously it is not what one normally understands the term to mean; but we may recall that the current theory takes it that the accomplishment of internal closure should be regarded as rewarding-in-itself and accompanied by subjective feelings of satisfaction, so presumably a loss of internal-closure will be unpleasant. Anyhow we can see that the two conceptualizations seem to be compatible, and the merely semantic difficulty should cause us no real problem. 437

If we can agree that the dog *does* feel this situation as casting uncertainty into its felt-ability to cope — as an attack on its ego-schema in other words — then according to the principles of privileged-protection for the ego put forward above, the dog will go to almost any extreme to try to redeem the situation. One interesting point is that the resulting breakdown was described as "neurotic-like" rather than psychotic. Perhaps we should not read too much into the distinction, but suppose we do take it at face-value; *Why* is it a neurotic response rather than a psychotic one? And what has happened to our explanation for schizophrenia?

One clue to the latter question is the course supposedly taken by some cases of paranoia. There we had a condition with an arguable status as an initial neurosis, but with inbuilt tendencies to progress into psychosis. One interesting possibility is that whereas "paranoia", as usually categorized, entails a more-or-less *conscious* mistrust of *social* objects and processes; perhaps some other types of neurosis could be described as a less conscious, lower $\mathbf{M^nL}$ level, mistrust of rather more-basic aspects of the environment. Such neuroses might perhaps then run a similar risk of progressing into psychosis, though, if they were low $\mathbf{M^nL}$ types, they might tend toward more hebephrenic or "Process" categories of schizophrenia. 438

As for "why … neurotic?", there is no obvious clearcut answer (if indeed the claim is well founded). Suppose however we were to postulate a wider category of paranoid-like neuroses on the lines just suggested, and envisaged them as all resulting from various different sorts of dangerously-close attacks on the ego; then from a structural point of view we might expect to find some rather desperate attempts to save the closure of the ego-structure by "amputating" that part of it which seemed to have become contaminated, and re-closing the remainder of the ego-elements into a somewhat diminished ego-structure. (This, of course is substantially what Laing suggests (1960); but the main difference is that we are here considering a basically similar

process for a wider range of basic structures associated with the ego-structure, and not just the more social-object phenomena of traditional paranoids).

There is though, something appealing about the supposed origin in an attack on the ego-structure. That, after all, is what neurotic defense-mechanisms are supposed to be intimately involved in. Moreover, it could be this very feature which makes for the greater stability and duration in schizophrenia proper — as opposed to mere schizomimetic psychoses which perhaps always result from attacks on schemata *other than* the ego itself.

439

C8.5 Which Direction For Future Research?

We started in Section C1.1 by contemplating Chapman and Chapman's (1973) ideas concerning future research into schizophrenia; and it was suggested that a mere tightening up of statistical design (on operationalist/behaviourist lines) as proposed by them, was not likely to produce greatly improved insight. Since then, some quite complex structures have been postulated here, on theoretical grounds, to account for schizophrenia and associated phenomena; so it is now pertinent to ask whether Chapman and Chapman's proposed conventional and objective approach could ever have led to such complex conceptualizations, unaided by the "less respectable" techniques such as those used here.

Note that even if the theory offered here should turn out to be totally wrong, it would nevertheless have served to show that the underlying mechanism *could* plausibly be very much *more complex* (though orderly) than most psychological or computer-models would have us believe; so that doubt must remain concerning the practical usefulness of Chapman and Chapman's proposal for future research.

It should be recognized that there are important uses for theories which spell out structural details of the supposed micro-structure, and that one of these uses is *to guide the experimenter* to those correlations-or-whatever which it might be most profitable to investigate. If the lead should turn out to have been a false one, then at worst one has simply just done *yet another* experiment which has not done much to further our insight; but in practice it is likely that we can achieve some worthwhile modification of the structural theory which will enable us to try again.

440

In fact, in general, we may expect a useful interplay between the two approaches at many stages of an investigation: from initial considerations to concluding interpretations. Indeed, according to the epistemological arguments outlined in Section C1.1, there is little basis for adopting either experimental-investigation, nor internal-closure-seeking as *the* superior approach; rather we should consider the two as complementary. So let us, by all means, press on with suitable experimental studies; but we would do well to press on with structural theoretical developments also — and use these two approaches in a mutually guiding and supporting collaboration.