

The Loosening of Associations

Bleuler's system and its philosophical ground — the *Lehrbuch* of 1920, the two genealogies of association, and the reply to Schneider



Anri Ê to Oigen Buroirā アンリ・エーとオイゲン・ブロイラー

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NOTA IN LIMINE — PREFATORY NOTE

This is Paper I of the three-part English edition of the author's study of Bleuler and Henri Ey, based on the Japanese original, v27 (2026). The present paper reconstructs Bleuler's system as a system — the *Lehrbuch* of 1920, the philosophical ground of the loosening of associations, the answer this makes possible to Schneider's objection, and the late psychobiological works. What French psychiatry made of that system is the subject of Paper II, *The Cradle of Delusions*. Paper III, *The Natural History of the Soul*, is a *commentarius*: a section-by-section reading of the *Naturgeschichte der Seele* of 1921, the chief of the late works treated summarily in § 7 below, undertaken to establish what Bleuler had and had not committed himself to before the *Psychoïde* of 1925. The complete argument remains the Japanese original. Sources shared between the three papers carry different reference numbers in each, since each list is self-contained. So, it should be added, is each paper: where a premise of the argument here is developed at length elsewhere — in the companion papers, or in the author's studies of Schneider — the premise is stated and documented in this paper as well, with its primary text quoted among the *Texts* below, and the cross-reference is offered for amplification and not in place of evidence.

Abstract

Eugen Bleuler is, in the English-speaking world as in Japan, effectively a one-book author. The 1911 monograph on the schizophrenias is read; the *Lehrbuch der Psychiatrie*, the *Naturgeschichte der Seele*, *Die Psychoide* and *Mechanismus, Vitalismus, Mnemismus* are not. Henri Ey, who translated the monograph in 1925 and lectured on Bleuler in 1940, insisted that the four later books were where the system stood — and then, in a footnote to his own lecture, admitted that he had not treated the textbook. This paper takes up what Ey left undone. It reads chapter A of the third edition of the *Lehrbuch* (1920), the “Psychological Guide,” from the inside, and sets it against the corresponding chapter of Kraepelin’s sixth edition (1899) to show that the two men were asking different questions: Kraepelin what is observed, Bleuler why it occurs. It then argues that the concept on which Bleuler’s whole system turns — the loosening of associations — cannot be understood within the associationist tradition to which it is conventionally assigned. Association descends from Aristotle under two incompatible understandings: as *contents* deposited one by one by experience, the line running through Locke and Hume to Wundt; and as a

capacity, the connecting function itself given to mind or brain in advance of experience, the line articulated classically by Kant, Herbart and Griesinger. Only the second permits the inference Bleuler actually drew, from loosened associations to a cerebral process — an inference his own citations nowhere explain, since the 1911 monograph names none of these philosophers, and the reconstruction offered here is accordingly presented as a reconstruction. Read on that ground, Kurt Schneider’s dismissal of the fundamental symptoms as diagnostically useless is not a refutation but a change of level — and Schneider’s own confession that the somatic basis of schizophrenia was for him *ein Glaubensbekenntnis*, an article of faith, marks the distance precisely. The paper closes with the late psychobiology, where engram theory and the *Psychoide* extend the same monism, and with the modern approaches — predictive coding, aberrant salience, the computational analysis of speech, the shared genetic architecture of five disorders — that have arrived in the neighbourhood of Bleuler’s intuitions by other roads; what such arrivals do and do not establish is weighed in the closing sections.

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1. Introduction: the books that were not read

Bleuler survives in the literature as the author of one book. Eugen Bleuler (1857–1939) wrote a great deal; *Dementia praecox oder Gruppe der Schizophrenien* (1911) is what is read.^[1] It is in English in Zinkin's translation of 1950 and in Japanese since 1974; it is cited, excerpted and taught. Around it stands a body of work that is, for practical purposes, unread. The *Lehrbuch der Psychiatrie* exists in English only in A. A. Brill's authorized translation of 1924, made from the fourth German edition and reissued by Dover in 1951, and is almost never consulted. *Naturgeschichte der Seele* (1921), *Die Psychoide als Prinzip*

der organischen Entwicklung (1925) and *Mechanismus, Vitalismus, Mnemismus* (1931) have never, so far as the present author has been able to establish, appeared in English. The result is a Bleuler who appears in the histories as a clinician with a happy terminological instinct — the man who renamed dementia praecox — and not as what he took himself to be, the author of a general psychopathology with a philosophy of mind underneath it.

Henri Ey knew better, and said so. In the lecture he gave in 1940 and mimeographed in 1945 he wrote that “the work of the Zurich master is, in my eyes, the masterpiece of the psychiatry of this century,” and that “the study of the schizophrenic states by itself

represents an extensive and fundamental conception of general psychopathology, complemented by the four principal works that followed.”^[4] The five books he had in view were the monograph of 1911, which reconstructed the concept of schizophrenia; the *Lehrbuch*, first published in 1916 and reaching in its third edition of 1920 five hundred and twenty-six pages, which organized the whole of psychiatry from chapter A, the “Psychological Guide,” to chapter N on the individual mental illnesses; the *Naturgeschichte der Seele*, a psycho-biological monism; *Die Psychoïde*, which proposed an organizing principle of living matter; and *Mechanismus, Vitalismus, Mnemismus*, a reckoning with the foundations of biology. Ey also knew, and recorded, that Bleuler in a letter of 1932 set a higher value on the *Naturgeschichte* than on the schizophrenia monograph.^[6]

And then, in the eighth of his own notes to that lecture, Ey conceded the gap: “his textbook, which I have not touched on in this paper, does not — one might almost say absolutely does not — apply his mnemistic conception of the mind-body relation.”^[4] The admission is the warrant for the present paper. Ey read Bleuler as the purest exponent of a third way between mechanistic organicism and pure psychogenism, and he was right; but he built the reading out of the

monograph and the late psychobiology and left the textbook out. The textbook is where Bleuler set down what he thought a mind was before he said anything about what happens to a diseased one, and it is therefore where the system is most visible as a system.

This paper reads it. Sections 2 to 4 take the third edition of 1920 — Bleuler’s own last word before his son’s revisions began to overwrite the book — and work through its architecture, its debt to and quarrel with Kraepelin, the psychology of chapter A, and the pathology of chapters B and N.^[2] Section 5 turns to the concept everything rests on and asks what philosophical premises are required to hold it: the loosening of associations is normally filed under a tradition that cannot in fact support the use Bleuler made of it, and the misfiling has consequences. Section 6 brings Kurt Schneider’s objection against that background and shows what it does and does not establish. Section 7 follows the argument into the late works. Section 8 draws the results together.

The companion paper, *The Cradle of Delusions*, takes up the other half of the story: what happened when this system crossed the Rhine — Ey’s summer of translation in the Pyrenees in 1925, the confrontation with Henri Claude at the Geneva–Lausanne

congress of 1926, Minkowski's Bergsonian reading of autism, and the long institutional afterlife of Bleuler in French psychiatry down to ICD-11.

A word on the edition used, since the bibliography of the *Lehrbuch* is unusually tangled and has misled more than one commentator. Eugen Bleuler himself saw the book through six editions; after his death in 1939 his son Manfred took over the revision and carried it, through nine further editions, to the fifteenth of 1983. The complete Japanese translation, published in three volumes and finished in 1990, is made from that fifteenth edition — of which Manfred wrote that each revision had completely rewritten more than a quarter of the whole. A reader who goes to the Japanese *Lehrbuch* for Eugen Bleuler's own thought is therefore reading, in large part, his son. The third edition of 1920 stands closest to the monograph, is Eugen's unaided work, and retains his original chapter design. It is the text used throughout here; the full edition history is given in the notes.^[3]

2. The *Lehrbuch* of 1920: architecture and intent

Bleuler built the textbook, as he says, to give physicians the knowledge they need at the

bedside. But the first edition's preface states a commitment that goes well beyond a practical handbook: psychiatry without psychology, he writes, is nosology without physiology. And under it stands a methodological principle he repeats and observes: that unclear concepts passing current as clear obstruct real understanding far more than ignorance does. The book is, from beginning to end, an exercise in making concepts precise before using them.

The architecture follows from the commitment. Chapter A, the "Psychological Guide" (*Psychologische Wegleitung*), sets out systematically the psychological concepts psychiatry will need — consciousness and the unconscious; sensation, perception and representation; association and thinking; intelligence; memory; affectivity; attention; suggestion; autistic thinking; and personality. Chapter B, "General Psychopathology" (*Allgemeine Psychopathologie*), treats the pathological deformation of each of those functions in turn. Chapter N, "The Individual Mental Illnesses" (*Die einzelnen Geisteskrankheiten*), describes the diseases themselves, from the organic psychoses through the group of schizophrenias, manic-depressive illness, the neuroses and mental deficiency. The sequence is itself an assertion: that no mental illness can be understood before the normal

working of the mental function it deranges has been understood.

What that assertion amounts to becomes visible only in contrast with Kraepelin. The second chapter of the sixth edition of Kraepelin's *Psychiatrie* (1899), "The Phenomena of Insanity," is built in four parts — disorders of the perceptual process, of intellectual activity, of the emotional life, and of will and action — reproducing with great tidiness the faculty-psychological triad of knowing, feeling and willing (*Erkennen–Fühlen–Wollen*) that eighteenth-century psychology had bequeathed.^[5] Each part is assembled as an inventory of phenomena observable from outside; in the section on thought processes one finds flight of ideas (*Ideenflucht*), *Zerfahrenheit*, obsessional ideas, catalogued as forms of symptom. And Kraepelin states his governing interest in the introduction: what may justly be demanded of a physician is the prediction of what is to come. The longitudinal chain that runs from observed symptom to prognosis is his central clinical proposition, and psychology is admitted as an auxiliary to it.

Bleuler's "Psychological Guide" begins from a different question altogether. Where Kraepelin asks what is observed, Bleuler asks why it occurs. If Kraepelin records the shapes

of symptoms from outside the window, Bleuler goes into the room and tries to grasp from within the mechanism by which mental phenomena come about. The difference is thoroughgoing from the first page of chapter A. Working under his rule against unclear concepts, Bleuler first establishes a normal psychology systematically, and only then describes pathology as its quantitative deformation. The formula that follows — the difference between the patient and the healthy person is only quantitative — stands in direct opposition to Kraepelin's clean dichotomy of the sound and the diseased.

From that methodological position one choice follows necessarily: association is installed as the central concept of psychiatry. For Kraepelin the association of ideas is one item in the vocabulary of symptom description, a term for naming an observed disturbance of thought. For Bleuler it is the answer to a question — how does thinking in fact link itself together? — that had been shaped over a long descent, from Aristotle's account of recollection through the association philosophy of Locke and Hume to Wundt's experimental psychology. The word-association experiments Bleuler conducted with Jung at the Burghölzli between 1904 and 1906 lie on the natural extension of that line. And if it is the strength or weakness of the

affinity between associations that establishes the logical order of thought, then a weakening of that affinity — a loosening of associations — is not one symptom among others but the collapse of the condition on which human thinking rests.

The intuition has since found echoes it did not seek. Fletcher and Frith's predictive-coding model (2009) and Kapur's aberrant-salience theory (2003) share with Bleuler's conception of associations governed by context a common explanatory form — the failure of a contextual constraint upon what rises into thought — though, as § 6.1 will argue, what this establishes is a structural analogy between paradigms and not a demonstration across them.^{[21] [20]} What can be asserted more firmly lies elsewhere: the descriptive core of the concept has become measurable. Computational analyses of speech now quantify the semantic coherence between successive utterances, and some of them do so citing Bleuler on their first page.^{[23] [24]} A century on, the loosening of associations is at least no longer what Schneider took it for — a notion that resists all operational grasp. That is why it heads the list of fundamental symptoms of schizophrenia, and why the whole of chapter A can be read as the unfolding of a single conviction.

Kraepelin sought to induce the lawfulness of symptoms from the records of the clinic; Bleuler sought to deduce the pathological state from an insight into the philosophical and psychological foundations of thought. The divergence of direction is a remote cause of what Bleuler would say in Lausanne in 1926 — that reading the French literature on schizophrenia gave him the impression of being a legendary figure to whom words he never spoke and deeds he never did were commonly attributed.^[19] It also hardens, later, into the two lineages that dominate the second half of the century: Schneider's doctrine of primary delusion, converging on a diagnostic cross-section, and Ey's dissolutional theory of delusion, tracing the passage from acute confusion to chronic delusion. Those are matters for § 6 and for the companion paper.

3. Chapter A, the “Psychological Guide”

Chapter A treats psychology systematically as the science that must come before psychiatry. Its sections may be taken in order.

Section a, “Consciousness and the Unconscious,” defines consciousness as the totality of mental processes present at a given moment, and gives the unconscious the

standing of a positive psychic force. Section b, “Sensation, Perception, Representation,” treats perception as an active organization of sense data, and lays here the ground for the later formula that the difference between the sick and the healthy is only quantitative.

Section c, “Association and Thinking,” is the core of the chapter. Bleuler defines association as the force that binds the material of thought, and argues that the strength or weakness of associative *affinity* (*Affinität*) determines the logical order of thinking. The weakness of associative affinity — *Schwäche der Assoziationsaffinität* — is the conceptual heart of what will later be identified as the primary symptom of schizophrenia. What is at stake philosophically in that concept is the business of § 5.

Section d, “Intelligence” (*Intelligenz*), defines intelligence as the capacity to apply existing associations flexibly to new situations. Because a disturbance of intelligence is then understood as derivative from a disturbance of association, dementia (*Demenz*) ceases to be a loss of knowledge and is reconceived as a loss of associative flexibility — a redefinition with obvious consequences for a nosology built around *dementia praecox*. Section e, “Memory” (*Gedächtnis*), connects directly with

the mnemism of the later *Naturgeschichte der Seele*. Section f, “Affectivity” (*Affektivität*), works with the concept of feeling-tone (*Gefühlston*), and understands affective flattening (*Affektverflachung*) as a structural dissociation between affect and the content of thought. Section g distinguishes active from passive attention; section h treats suggestibility as a contributor to the formation of pathological states.

The upshot is that the “Psychological Guide” presents a comprehensive psychology as the basic science of psychiatry. Read from the 1911 monograph alone, Bleuler’s psychopathology looks like a theory of one disease. Read against chapter A, it appears as the clinical application of a theory of mind as such.

3.1 Section k: autistic thinking

The section that matters most for what follows is k, “Autistic Thinking” (*Das autistische Denken*). Bleuler sets realistic thinking, bound to reality (*realistisches Denken*), against thinking detached from it (*autistisches Denken*). The mark of the latter is that it takes no account of contradiction with the real, and follows instead a *logic of feeling* (*Logik des Fühlens*), pursuing goals set by instinct and affectivity.

When we let our fantasy run in play, in mythology, in dreams, in certain morbid states, thinking will not or cannot concern itself with reality; it pursues goals given by instincts and affects. *[T1]*

What deserves notice is that autistic thinking is grasped here as continuous with dream, myth, poetry and philosophy. Autism in schizophrenia is placed as the pathological extremity of a normal autistic thinking. The formula of the later papers — the difference between patient and healthy person is only quantitative — is already contained in the basic conception of this chapter. And it is precisely this principle of continuity that Ey would extract from the textbook.

The continuity is stated at its sharpest not in the textbook but in the monograph of 1911, in a short section on the relations of schizophrenia to dream. There Bleuler writes that the analogy “becomes identity in the cases where the patients treat their dream hallucinations as real ones, form their delusional ideas in the dream and hold to them when awake,” and concludes that dreamlike and schizophrenic-autistic thinking are, for the means of investigation then available, essentially identical (S. 356–

357). This is worth setting beside Chapter A, since it shows that what the textbook treats as one mode of thinking among others is, for Bleuler, the very mode in which every sleeper thinks each night. It is worth setting beside it for a second reason as well: in the same sentence Bleuler adds that delusional ideas — *but not all of them* — are formed in dreams. The restriction is the one he had already entered two hundred and fifty pages earlier in writing that the acute states are the cradle of *many* delusional ideas. What became of that restriction in France is the subject of the companion study.

4. Chapters B and N: pathology as deformation

Chapter B, “General Psychopathology,” is constructed so as to describe the concepts established in chapter A in their pathological deformations. In the section on disturbances of the afferent functions, hallucination is placed not as a product of a primary disturbance of brain process but as a *secondary* symptom, generated by the autistic mental activity which that disturbance has released. The section on disturbances of association and thinking describes systematically the pathological deformation of chapter A’s section c. Affective flattening is understood

not as an extinction of feeling but as a dissociation between affect and thought-content: powerful feeling continues to move in the patient's inner world, but has ceased to connect with its outward expression.

In chapter N, “The Group of Schizophrenias,” from page 280 onward, Bleuler names as fundamental symptoms the disturbance of association, the alteration of affectivity, and autism, and places hallucination, delusion and the catatonic symptoms among the accessory ones. “This disease may halt at any stage,” he writes, “and many of its symptoms may diminish greatly in degree or disappear entirely. We conceive it not as a single disease in the narrow sense but as a group of diseases.” This is the root of Bleuler's position — schizophrenia grasped not as an entity (*Entität*) but as a group (*Gruppe*) — and it is the very point Ey would press with such insistence in the lecture of 1940.

5. The two genealogies of association

To see what Bleuler meant by the loosening of associations — and why he put it first among the fundamental symptoms — one has to separate two lines of descent which both begin in Aristotle and which have run side by side, largely unremarked, ever since.

The first is the line of the *Organon*. The Categories, the Prior and Posterior Analytics, On Interpretation: a system of formal rules for correct inference, with the syllogism at its centre. Through Aquinas this became the spine of scholastic philosophy, and it attached itself there to the definition of man as *animal rationale* — the Latin heir of Aristotle's *zōon logon echon* (Politics 1253a), whose functional argument stands in the first book of the Nicomachean Ethics. To take Aristotle into scholasticism was to place a norm, logical correctness, at the foundation of thought; and early modern philosophy kept the frame, Descartes still locating what is universal in man in *bon sens*, the capacity to reason well. [11]

The second line begins in a different work altogether: *De Memoria et Reminiscentia*, one of the psychological essays of the *Parva Naturalia*. There Aristotle argued that recollection is organized by three principles — similarity, contrast, and contiguity in space and time. These are not rules for correct inference. They are a description of how thinking in fact proceeds from one thing to the next; and for as long as scholasticism kept the *Organon* at its centre, this second, associationist line stayed in the margins. [11]

It came to the centre in British empiricism. Hobbes discussed the “train of thoughts” in *Leviathan* (1651).^[12] Locke introduced the term “association of ideas” in the *Essay* (1690), though he treated it negatively, as a source of the errors that spoil correct reasoning.^[13] It was Hume who, in the *Treatise* (1739), made association a fundamental operation of the mind and gave it three principles of its own: resemblance, contiguity in time and place, and causation.^[14] Through David Hartley and James Mill the line runs to Wundt, who measured the reaction times of association in the laboratory and so converted a philosophical concept into a psychological one that could be measured.^[18]

Here, though, a decisive question has to be put. Locke treated association as a source of error and Hume as a fundamental operation of mind; but for both, associations were *acquired* — laid down by the accumulation of experience. Within the empiricist frame, then, an odd or pathological association is a contingent failure in the work of experience and memory, an individual accident. And on that frame the proposition *loosening of associations is evidence of a cerebral lesion* cannot be built at all. If the connections are merely deposited by experience, their failure has no route to the structural pathology of an organ.

Hartley is the partial exception worth naming, since his associationism was grounded in a physiology of neural vibrations. But in Hartley too the individual associations are formed by the repetition of experience; the capacity for association itself is not yet what the Continental line will make of it, something given to the brain structurally and in advance.

Bleuler’s move therefore requires a quite different philosophical premise: that the function of connecting is built into the brain structurally, prior to experience. Only then does its failure become evidence about an organ. The decisive figure in that line is Kant, and specifically the transcendental unity of apperception. For Kant the power to combine and synthesize representations does not come from experience; it is the condition under which experience is possible at all. The *I think* must be able to accompany all my representations — and it is this unity of apperception that makes every association, every synthesis, possible. On such a frame, a loosening of associations is not a contingent failure of experience but the giving way of the transcendental condition of experience itself.^[15]

Between Kant and Bleuler stands Johann Friedrich Herbart (1776–1841). In *Psychologie als*

Wissenschaft (1824–25) Herbart found mathematical laws in the interaction of representations (*Vorstellungen*): a threshold of consciousness (*Schwelle des Bewusstseins*), below which a representation does not rise into awareness; an inhibition (*Hemmung*) exerted between competing representations; fusion and complication (*Fusion und Komplikation*). These are not deposits of accumulated experience but structural, quasi-a-priori laws of mental functioning. Herbart's psychology marked nineteenth-century German psychiatry deeply, and by way of Griesinger it forms the immediate background of the psychiatric style of thought Bleuler inherited.^[16]

That immediate predecessor is Wilhelm Griesinger (1817–1868), whose thesis — condensed by tradition into the formula *Geisteskrankheiten sind Gehirnkrankheiten*, mental illnesses are diseases of the brain, though his own words in the opening paragraph run rather „haben wir vor allem in den psychischen Krankheiten jedesmal *Erkrankungen des Gehirns* zu erkennen“ — presupposes that mental functions are organ-functions of the brain, given in advance. Symptoms are then expressions of a lesion in that organ. This is plainly not empiricism; one might call it a biological apriorism. Bleuler stands directly in the succession, and the logic by which he made the loosening of associations evidence

of a cerebral lesion belongs to the German cerebral-psychiatric tradition descending from Griesinger — the psychiatric fruit of Kantian apriorism and Herbartian mechanics of representation.^[17]

A caution about the standing of this claim is owed to the reader, and it is best entered as a plain statement of the textual record. The monograph of 1911 cites neither Kant nor Herbart nor Griesinger — not once.^[25] The association psychologist whom Bleuler does cite, and cites more often than any other, is Theodor Ziehen; and Ziehen, in the preface to his own *Leitfaden der physiologischen Psychologie*, declares his allegiance not to any Continental line but to „die sogenannte Assoziationspsychologie der Engländer“, setting his book expressly against Wundt.^[26] Nor is that all. When Bleuler at last discusses the Kantian doctrine of synthesis directly — in the *Naturgeschichte der Seele* of 1921 — he rejects it, deriving the combination of representations not from a synthetic faculty of the imagination but from memory, and answering the doctrine of the a priori with a flat counter-thesis: „Die Kausalität ist eine Denknöwendigkeit, die aus der Erfahrung stammt.“^[27] Whoever asserts a Kantian filiation for Bleuler must assert it against these facts and not in ignorance of them.

The claim made in this section is therefore not biographical but systematic, and the distinction should be stated without embarrassment. What is reconstructed here is the logic of an inference, not the contents of a library: the question is what premises must be in place for *the associations are loosened* to function as evidence of a cerebral process. And for that question the division that matters is not the one between nations but the one between two understandings of association. On the first, associations are *contents*, deposited one by one in the course of experience; their derangement is then an accident of an individual history, and no road leads from it to the pathology of an organ. On the second, association is a *capacity* — the connecting function itself, built into the organ in advance of any particular experience; its derangement is then the signature of a lesion. The inference Bleuler drew stands or falls with the second understanding. Its classical Continental articulation runs from Kant's transcendental unity of apperception through Herbart's mechanics of representations to Griesinger, whose debt to Herbart is acknowledged in his own text and settled in the scholarship;^[28] and that Herbart stood among the theoretical foundations of Bleuler's thinking is likewise the judgment of the modern specialist literature, not an invention of this paper.^[29]

But the capacity view had a second road to travel by: physiologized associationism reached it from the empiricist side — Hartley naturalizing Hume's principles into vibrations, Ziehen housing the associations in the cortex — and Bleuler's own late doctrine naturalizes the *a priori* itself, the *phyloplastic* organization of the *Naturgeschichte* being an inherited structure that does the work of Kant's synthesis without its transcendental warrant.^[27] By which of these doors Bleuler entered, his citations do not permit us to decide, and this paper does not pretend to decide it.

What the sources do permit is this. The received formula, *Bleuler inherited the associationist tradition that descends from Aristotle*, still needs its qualification, but the qualification can now be made precise. Aristotle is the source of both understandings; Bleuler's use of the concept presupposes the second; and the second had been made the working grammar of German-language psychiatry above all by the cerebral-psychiatric tradition descending from Griesinger, within which associations could figure as functions of an organ because the capacity for connection was taken to be one. That Bleuler absorbed this grammar without ever naming its authors is an interpretation and not a documented fact, and it is offered here as what it is — a rational

reconstruction, entered with the historiographical cautions such reconstructions demand;^[30] but it is the interpretation under which the monograph's actual procedure, the untroubled passage from loosened associations to a *Hirnprozess*, first becomes coherent.

Standing where he did, Bleuler formulated the loosening of associations at the crossing point of both traditions. The word-association experiments carried out with C. G. Jung at the Burghölzli (*Diagnostische Assoziationsstudien*, 1904–1906) supply, incidentally, an important reply to Schneider's charge that the concept resists operationalization: the loosening of associations was not a purely impressionistic notion but set out from the start as something measurable by an experimental procedure, the timing of responses. In schizophrenic patients the complex indicators (*Komplexindikatoren*) — delayed responses, absurd responses, repetitions of the same response — were shown to appear characteristically.

But the weight of the concept does not come from that experimental basis alone. Only when one sees that Bleuler stood at the intersection of the two traditions — correct inference as *logos*, in the line of the *Organon*, and the actual chaining mechanism of thought, in the line of the association theory

— does it become clear why he placed the loosening of associations at the head of the fundamental symptoms, and gave it such weight.

If association in Hume's sense is the fundamental organizing mechanism of thought, then the normal functioning of association is the condition of thinking as a human being thinks. And the rational capacity which the tradition since scholasticism has displayed under the definition of man as the being possessed of *logos* is itself supported by a deeper cognitive foundation: the capacity to select and inhibit associations according to context. The patient in Bleuler's celebrated Congo example *knows* where Egypt is — the knowledge is intact — and yet cannot govern his associations according to the logical and spatial priorities the question demands. This is not an error of formal logic; it is a failure of the cognitive control that supports the exercise of *logos* from below. When associations loosen, a person sinks into a dreamlike autistic thinking and loses the capacity for contact with reality. That is not a description of a symptom. It is a claim about the collapse of the condition on which human thinking rests.

The principle of continuity Bleuler stated at Lausanne — that schizophrenia exists in every degree, from apparent health to the

gravest states of dementia — can be reread in this context. In the healthy, too, contextual control of association is not perfect: it loosens in fatigue, in excitement, in dreams. Schizophrenia is understood as the quantitative extremity of that. Which is to say: the Aristotelian definition of man as the being possessed of logos is subject to gradual collapse.

6. Schneider's objection, and the level of the question

Kurt Schneider (1887–1967) proposed the symptoms of the first rank (*Erstrangsymptome*) — thought insertion, thought broadcasting, thought withdrawal, experiences of being made or influenced, voices conversing with one another — as diagnostic indicators of schizophrenia, and in effect dismissed Bleuler's fundamental symptoms, the loosening of associations above all, as useless for diagnosis.^[9]

What has to be seen is that the two men were asking questions at different levels. Schneider's was operational and practical: which symptoms should the clinician use in order to decide reliably whether schizophrenia is present? Bleuler's was pathogenetic: why do these symptoms arise?

That these are different questions is not an inference of the present author's but Schneider's own declaration, made in the very paragraph that introduces the first-rank symptoms — and made, as it happens, by naming Bleuler:

Among the numerous abnormal modes of experience occurring in schizophrenia there are some that we call symptoms of the first rank — not because we *held* them to be “basic disturbances” (*Grundstörungen*), but because they *carry* a quite special weight for the diagnosis, as against non-psychotic mental abnormality and as against cyclothymia alike. This valuation, then, bears only upon the diagnosis. Nothing, however, is thereby said about the theory of schizophrenia, in the way that Bleuler’s “fundamental” and “accessory” symptoms intend, or his and other authors’ “primary” and “secondary” symptoms. [T7]

Schneider was not rejecting pathogenetic claims, then; he was declining to make them, and saying so with Bleuler’s own pair of terms in front of him. That the disclaimer is inscribed even in the grammar of the

sentence — the essentialist reading held off in the subjunctive *hielten*, the diagnostic weighting asserted in the indicative *haben* — is the subject of a separate study by the present author, to which nothing in the present argument is owed. [9]

More decisively, Schneider was candid about the organic basis of schizophrenia:

This is an article of faith, and it does not want for challenges. [T3]

Conceding that the pathological processes underlying cyclothymia and schizophrenia are unknown to us, he nevertheless declared that he would stand by the hypothesis — and hence by the position that these states are *krankhaft*, morbid — in the sense of a heuristic principle. For Schneider, in other words, the organicity of schizophrenia remained a hypothesis held as an article of faith for clinical and forensic convenience, not a piece of demonstrated knowledge. The contrast with what Bleuler declared at Lausanne in 1926 is sharp:

Schizophrenia is a physiogenic affection, that is to say one with an organic basis. [...] The organic origin of schizophrenia can today be demonstrated with all the evidence one could wish. [T2]

For Bleuler organicism was not a faith but a demonstrable proposition, and the loosening of associations was its psychological expression, experimentally measurable from the beginning. That the presentation of the first-rank symptoms was accompanied by no pathogenetic ground whatever, being a purely operational procedure, is a thing Schneider thus said of himself.

A paradox deserves notice here, and it is not a small one. Consider the pathogenetic standing of the symptoms Schneider chose. Thought insertion, thought withdrawal, voices conversing — in Bleuler’s framework every one of these is a *secondary* symptom. They are reactive products which the surviving mental activity generates in response to the primary disturbance of association, and it is exactly because they are such products that the patient can report them consciously, as *experiences*. The symptoms Schneider calls useful for diagnosis are the symptoms Bleuler says may be absent while the disease is

present. Diagnostic reliability and pathogenetic fundamentality tend to pull in opposite directions.

6.1 The Congo example

What of the conceptual precision of the loosening of associations itself? In the discussion at the Lausanne congress — treated at length in the companion paper — Courbon raised an objection to Bleuler’s celebrated Congo example. To answer the question “Where is Egypt?” with “between Assyria and the Congo” is, he argued, a reply a poorly educated ordinary person might equally give: if the man remembers that the Egyptian, Assyrian and Congolese rooms adjoined one another in a museum, then his associations are not in fact loosened. Nor, Courbon added, is such a chaining of ideas impossible as an expression of high spirits. The objection is a legitimate challenge to the conceptual boundaries of the concept — though, as will appear, only half of it hits the mark.

Returning to the intellectual-historical context set out above makes the answer clearer. What Bleuler meant to display in the Congo example was neither mistaken geographical knowledge nor a strange associative content. What he stressed was the

fact that the patient's answer *proved that he knew perfectly well where Egypt was*. The knowledge has not been lost. The problem is why the context of the question fails to suppress such a contingent contiguity association. Courbon's museum association, far from telling against Bleuler, endorses him: in normal thinking the museum memory is there too, but the question *where is Egypt?* activates a logical and spatial contextual constraint which suppresses the contingent memory. In schizophrenia that context-bound control weakens, and contingent associations rise more easily to the surface of thought.

The essence of the loosening of associations can therefore be understood as an impairment of the priority function belonging to the logical and semantic aspect among Aristotle's three principles of recollection — similarity, contrast, contiguity. The Congo example is the result of one of the three normal principles operating, contiguity; in that sense association has not vanished, but the *selective control* of association has been lost. This is the collapse of the precondition under which what the Organon tradition called the exercise of logos — inference appropriate to context — becomes possible at all.

Contemporary neuroscience offers this direction a suggestive parallel, and the suggestion should be taken for exactly what it is. Within the theoretical framework of predictive coding, schizophrenia is understood as a state in which higher-order cognitive models fail to apply appropriate precision weighting to prediction errors arising from lower-order sensory input (Fletcher and Frith, 2009) — a scheme formally akin to Bleuler's conception of associations selected under logical and contextual constraint.^[21] But the kinship is one of explanatory form and not of demonstrated identity. The framework's own architects concede that the evidence assembled so far is “compatible with” the account rather than a test of it, and that even the direction of the central abnormality — priors too weak, or priors too strong — remains unsettled;^[31] its philosophical critics press the harder charge, that a scheme flexible enough to accommodate any finding forfeits empirical content.^[32] The theory of aberrant salience (Kapur, 2003) reads the prominent activation of inappropriate associations as a misattribution of salience arising from dysfunction of the dopamine system, and so appears to give a neuropharmacological underpinning to Bleuler's intuition of a disorder in the control of associative selection.^[20] Here a twenty-

year retrospective, co-authored by one of the predictive-coding school's own principals, must be allowed its verdict: several of the theory's core predictions have failed empirically, and “a strong incentive salience theory of delusions (and hallucinations) cannot hold” — even though the presynaptic striatal dopamine abnormality itself remains among the most robust findings in psychiatry. [33] What the parallels license, and what they do not, is weighed in § 8; the word used there is analogy, and it is used advisedly.

What emerges from all this is that Schneider's critique did not demonstrate an error in Bleuler; it changed the level of the question. Schneider's contribution lies in having given an outstanding solution to the practical problem of operational diagnosis. What the conversion cost was the capacity to answer the pathogenetic question — why do the symptoms of schizophrenia take the shapes they do? Bleuler's theoretical framework, the two-layer structure running from loosening of associations through autism to delusion and hallucination, is what provides the ground on which that question can be answered. Ey's lifelong insistence that Bleuler's doctrine is above all organogenetic rests exactly here.

7. The late Bleuler: mnemism and the *Psychoïde*

That Ey's lecture of 1940 discussed not only the 1911 monograph but the late works in detail is almost unknown in Japan. Ey recognized that Bleuler's psycho-biological writings — above all the *Naturgeschichte der Seele* (1921) — were in France even less known than the work on schizophrenia, and treated them at length as throwing new light on Bleuler's organo-dynamism. He records that Bleuler himself, in a letter of 1932, valued the book more highly than the schizophrenia monograph. [6]

The principal thesis of the *Naturgeschichte* is a psycho-physiological monism built on the theory of *mnemes* — the engram (*Engramm*) — of Ewald Hering and Richard Semon. The fundamental somato-psychic function is memory; everything that has been lived is fixed in the form of an engram; and the engram, so close a relative of the “traces” of classical psychology, is a physical property of the brain which lasts as long as the brain does. Perception, abstraction, representation, thought and intelligence are, on this account, all elaborations of engrams, and the principal form of the activity of thought is associative. All of which is the theoretical deepening of

what chapter A section e of the textbook says about memory.

Ey's estimate of the book deserves to be given entire, because it is not the estimate of a disciple. "Certainly," he writes, "such a psychology remains essentially atomistic, associationist and, all in all, rather uninteresting. Nor have the critics spared it: the severe analysis Jaspers made of this work is well known." [T4] The concession is not a formality, and it is what gives the adversative that follows its weight: "But Bleuler's monism is nevertheless of a distinctly dynamist character, and one may say in this respect that it consists less in reducing the psyche to the brain than in making the psyche penetrate the very organization of the brain." [T5]

NOTE Ey gives no reference for the Jaspers analysis, remarking only that it is well known, and it is reported here as his. A criticism Jaspers did make of Bleuler can be documented — but of the *Psychoïde*, a different book from the one Ey is discussing. It is quoted in its place below.

This mnemistic monism carries the understanding of the organic basis of the loosening of associations a stage further. Association rests on the reactivation of

engrams — *Ecphorie* — and the loosening of associations comes to be understood as an organic disturbance of the whole process by which engrams are activated, selected and inhibited. The same mnemistic monism is systematically formulated as *Mnemismus* in the later *Mechanismus, Vitalismus, Mnemismus* (1931). [8]

In *Die Psychoïde als Prinzip der organischen Entwicklung* (1925) Bleuler introduced the concept of the *Psychoïde*. [7] The *Psychoïde* is the principle that living matter possesses an immanent purposiveness of organization. Once an analogy holds between the *Psychoïde* and the psyche, the roots of mind are lowered into the depths of the vegetative and reflex organization of the body, and the ground is cut from under any grasp of mental illness in terms of a dualistic opposition of mind and body.

It is precisely here that Jaspers entered an objection, and this one is on record. One qualification has to be entered before it is quoted. Jaspers does deliver his criticism in the *General Psychopathology*, but what he is criticizing there is the *Psychoïde* of 1925 — not the *Naturgeschichte der Seele* of 1921, which is the book Ey has in view when he speaks of a severe analysis that is well known. Whether the two are the same criticism cannot be established from Ey's text, and no claim to

that effect is made here. With that reservation, and discussing the relation of body and psyche, Jaspers wrote:

A recent example of this is Bleuler's use of the term "psychoid," which was to denote something common to somatic and psychic life alike: the function of memory, integration, the purposeful character of living structures and forces. The defect here, as with all such schematic ideas, is that a comprehensive viewpoint of this sort may indeed provide us with an ideal construct but this cannot be investigated nor can we use it in order to obtain new knowledge.^[10]

The charge is methodological. Jaspers does not say that the conception is false; he says that nothing can be done with it — that it yields an ideal construct and no research.

Ey's verdict is the exact reverse, and the sentence in which he states it is the hinge of his whole reading of Bleuler: this philosophical position, he writes, "is the only one that permits the establishment of a

psychopathology which does not collide at every step with the relations of the physical and the moral posed in the form of the alternative 'the organic' or 'the psychic.'" ^[T6] Where Jaspers saw a construct that could not be investigated, Ey saw the only ground on which a psychopathology could stand at all. The paragraph in which the sentence stands closes the lecture of 1940, and is quoted entire in the companion paper.

8. Conclusion

What the textbook shows, when it is read rather than cited, is that Bleuler's psychopathology of schizophrenia is an application of a general theory of mind, and that the theory has a philosophical ground which is not the one usually attributed to it. The loosening of associations is intelligible as evidence of a cerebral process only where association is taken for a capacity of the organ and not for a deposit of experience; the two-layer structure of primary and secondary symptoms follows from a psychology established before any pathology is described; and the late psychobiology extends the same monism into biology at large.

The vindication has come, as such things do, from unexpected quarters. Bleuler's original

insight that the group probably contains several diseases anticipated by a century the movement toward the schizophrenia spectrum and dimensional diagnosis in DSM-5 and after. While Schneider's operational turn came to fruition in the institutional authority of the DSM, the legitimacy of Bleuler's conception — a group of reaction-forms rather than a disease entity — is today supported by independent evidence from molecular genetics, in the identification by genome-wide association studies of genetic risk loci shared across schizophrenia, bipolar disorder, autism spectrum disorder, attention-deficit/hyperactivity disorder and major depression.^[22] And the concept Schneider set aside as impossible to operationalize has re-entered the sciences by three doors, which it is the business of a historical study to keep distinct.

There is, first, a documented lineage — and it runs in the unaccustomed direction. The dysconnection hypothesis of Friston and his colleagues names Bleuler's disintegration of the psyche as the psychopathological ancestor of its own central idea: the rare case in which the genealogical claim is entered from the neuroscientific side rather than the historian's. What is claimed there, it should be said, is the kinship of *Spaltung* and not of the loosening of associations itself.^[34] There is,

second, a partial operationalization: computational analyses of speech now measure the semantic coherence between successive utterances — the descriptive core of the loosened association — with clinical predictive value in small cohorts, and in studies that sometimes cite Bleuler on their first page; what they measure, it must equally be said, is the discourse produced and not the selective mechanism Bleuler postulated behind it.^{[23] [24]} And there is, third, a structural analogy: predictive coding and aberrant salience share the *form* of Bleuler's account — a failure of contextual constraint upon what surfaces — while differing from it in their objects, their mechanisms and their evidence, and while remaining contested within neuroscience itself.^{[31] [33]} Demonstration would be too strong a word, and this paper does not use it. But three doors, each of them opened from the other side, are more than resonance.

What French psychiatry made of this system, and at what price, is the subject of the companion paper, where the neuroimaging evidence for the primary/secondary distinction is also weighed.

Texts

Originals of the passages quoted in translation. Bleuler, Lehrbuch der Psychiatrie, 3. Aufl., Berlin: Julius Springer, 1920.

Bleuler, "La Schizophrénie," Rapport de psychiatrie, XXX^e Session, Genève–Lausanne, 1926 (repr. Paris: L'Harmattan, 2001). Schneider, Klinische Psychopathologie, 14. Aufl., Stuttgart/New York: Georg Thieme, 1992. Ey, "La conception d'Eugen Bleuler," in Bleuler (Viallard trans.), 1993.

[T1] (Lehrbuch, chap. A, sect. k) „Wenn wir spielend unserer Phantasie den Lauf lassen, in der Mythologie, im Traum, in manchen krankhaften Zuständen, will oder kann sich das Denken um die Wirklichkeit nicht kümmern; es verfolgt von Instinkten und Affekten gegebene Ziele.“

[T2] (Rapport, 1926) « La schizophrénie est une affection physiogène, c'est-à-dire à base organique. [...] L'origine organique de la schizophrénie se laisse démontrer aujourd'hui avec toute l'évidence voulue. »

[T3] (Klinische Psychopathologie, S. 6) „Das ist ein Glaubensbekenntnis und es fehlt nicht an Anfechtungen.“

[T4] (Ey 1993, pp. 655–656) « Certes une telle psychologie reste essentiellement atomiste, associationniste et, somme toute, assez peu intéressante. Aussi les critiques n'ont-elles pas été ménagées. On connaît la sévère analyse que Jaspers a faite de cet ouvrage. »

[T5] (Ey 1993, pp. 655–656, immediately following) « Mais le monisme bleulérien est cependant de caractère nettement dynamiste

et on peut dire à cet égard qu'il consiste moins à réduire la psyché au cerveau qu'à faire pénétrer la psyché dans l'organisation même du cerveau. »

[T6] (Ey 1993, p. 658) « Cette position philosophique est la seule qui permet l'établissement d'une psychopathologie qui ne se heurte à chaque pas à la position des rapports du physique et du moral sous la forme de l'alternative de « l'organique » ou du « psychique ». »

[T7] (Klinische Psychopathologie, S. 65) „Unter den zahlreichen bei der Schizophrenie vorkommenden abnormen Erlebnisweisen gibt es einige, die wir Symptome 1. Ranges heißen, nicht weil wir sie für „Grundstörungen“ hielten, sondern weil sie für die Diagnose sowohl gegenüber nichtpsychotisch seelisch abnormem, wie gegenüber der Zykllothymie ein ganz besonderes Gewicht haben. Diese Wertung bezieht sich also nur auf die Diagnose. Nicht aber ist damit etwas zur Theorie der Schizophrenie gesagt, wie das Bleulers „Grundsymptome“ und „akzessorische Symptome“ meinen oder seine und anderer Autoren „primäre“ und „sekundäre“ Symptome.“

Notes and References

[1] Bleuler, E. *Dementia praecox oder Gruppe der Schizophrenien*. Leipzig/Wien:

Franz Deuticke, 1911. English translation: *Dementia Praecox, or the Group of Schizophrenias*, trans. J. Zinkin. New York: International Universities Press, 1950. Japanese translation: trans. M. Iida et al. Tokyo: Igaku-Shoin, 1974 — a collaborative translation by four hands, in which the consistency of terminology has been criticized. French translation: A. Viallard. Paris: E.P.E.L./G.R.E.C., 1993. (Cited at 1.)

[2] Bleuler, E. *Lehrbuch der Psychiatrie*, 3. Auflage. Berlin: Julius Springer, 1920. The third edition is consulted directly throughout the present paper, for three reasons: it is Eugen Bleuler's unaided work and so philosophically unmixed; it stands only nine years from the schizophrenia monograph of 1911, and thus on substantially the same intellectual horizon as the text Ey translated in 1925; and its chapter design — chapter A, *Psychologische Wegleitung*, and the rest — preserves Eugen's original architecture, which is what makes the

internal analysis attempted here possible. Japanese translation: from the 15th edition (1983, revised by Manfred Bleuler), in three volumes covering the general part, the endogenous psychoses, the psychogenic psychoses, the organic psychoses and epilepsy; completed 1990. (Cited at 1.)

[3] *Edition history of the Lehrbuch*. Eugen Bleuler's sole authorship (Julius Springer, Berlin): 1st ed. 1916 (532 pp.), 2nd ed. 1918, 3rd ed. 1920 (526 pp.) — these three verified by the present author from PDF copies. The publication years of the 4th, 5th and 6th editions remain unverified. Eugen Bleuler died on 15 July 1939; that he supervised the book himself from the first edition through the sixth is stated by Manfred Bleuler in the preface to the 14th edition. Manfred Bleuler's revisions: 7th ed. 1943 (511 pp., *umgearbeitet von* Manfred Bleuler, with the collaboration of Berze, Luxenburger, Meggendorfer and Scheidegger); 8th ed. 1949 (506 pp.); 9th ed. 1955 (583 pp.);

10th ed. 1960 (629 pp.); 11th ed. 1966 (659 pp.); 12th and 13th eds. unverified; 14th ed. 1979 (706 pp.); 15th ed. 1983 (768 pp., with the collaboration of Angst, Ernst, Hess, Mende, Reisner, Scheidegger and others). In the preface to the 14th edition Manfred states that each revision completely rewrote more than a quarter of the whole, so that from the 7th edition onward the book has in effect been repeatedly rewritten in its entirety. English translation: *Textbook of Psychiatry*, trans. A. A. Brill (authorized English translation), 1st ed. 1924, from the 4th German edition; reissued by Dover, 1951. Japanese translation: from the 15th edition, in three volumes (the general part; the endogenous psychoses; the psychogenic, organic and epileptic psychoses), completed 1990. There is information that the Japanese translation was begun around 1943, but the present author has not been able to verify the details. (Cited at 1.)

[4] Ey, H. « La conception d'Eugen Bleuler. » In: Bleuler, E. *Dementia praecox ou Groupe des schizophrénies*, trans. A. Viallard. Paris: E.P.E.L./G.R.E.C., 1993, pp. 642–658. The text is the last of the lecture series “Contemporary tendencies in psychiatry,” delivered September–October 1940 and first mimeographed in 1945. Ey’s own note 8 is quoted in § 1 above; the passages quoted in § 7 are at pp. 655–656 and p. 658. A Japanese translation with detailed annotations by Prof. Ninsa Kageyama appears as “Document 2” in Henri Ey, *Genkaku 幻覚* [Les hallucinations], vol. 5, *Kishitsurikidōron 2 器質・力動論 2*, trans. N. Kageyama and T. Abe (Tokyo: Kongo Shuppan, 2017). (Cited at 1 and 8.)

[5] Kraepelin, E. *Psychiatrie. Ein Lehrbuch für Studierende und Aerzte*, 6., vollständig umgearbeitete Auflage, I. Band: *Allgemeine Psychiatrie*. Leipzig: Johann Ambrosius Barth, 1899. Chapter 2, “Die Erscheinungen des Irreseins,” sections A–D; the

demand for the *Voraussage des Kommenden* is stated in the introduction. (Cited at 2.)

[6] Bleuler, E. *Naturgeschichte der Seele und ihres Bewußtwerdens*. Berlin: Julius Springer, 1921; 2. Aufl. 1932. Bleuler's letter of 1932 valuing this work above the schizophrenia monograph is reported by Ey, ref. [4]. (Cited at 1, 7 and 8.)

[7] Bleuler, E. *Die Psychoide als Prinzip der organischen Entwicklung*. Berlin: Julius Springer, 1925. (Cited at 7.)

[8] Bleuler, E. *Mechanismus, Vitalismus, Mnemismus*. Berlin: Julius Springer, 1931. (Cited at 7.)

[9] Schneider, K. *Klinische Psychopathologie*, 14., unveränderte Auflage, with a commentary by G. Huber and G. Gross. Stuttgart/New York: Georg Thieme Verlag, 1992. The confession of faith quoted as [T3] stands at S. 6; the paragraph introducing the first-rank symptoms and disclaiming any bearing on the theory of schizophrenia, quoted as [T7], at S. 65. Both passages are reproduced in the

original among the *Texts* above, so that the reader may check the use made of them here without recourse to any other publication. The distribution of grammatical mood across that paragraph, and the wider structure of Schneider's methodological reservation, are analysed in the author's *The Grammar of Diagnostic Caution*, Bulletin of YRIDEI 1 (2026), Article 15-I; the present argument uses only the disclaimer itself, which the quoted text carries on its face. (Cited at 6.)

[10] Jaspers, K. *General Psychopathology*, trans. J. Hoenig and M. W. Hamilton. Baltimore: Johns Hopkins University Press, 1997, vol. 1, p. 224; the passage stands in the discussion of the body-psyche relation in the chapter on the somatic accompaniments of psychic activity. The German original is *Allgemeine Psychopathologie*, Berlin: Julius Springer, 1913; 9. Aufl., Berlin/Heidelberg/New York: Springer, 1973, S. 189 — Erster Teil, Drittes Kapitel, *Die Symptome des Seelenlebens in körperlichen Begleit-*

und Folgeerscheinungen (Somatopsychologie), in the preliminary remarks on *Leib und Seele*. The German pagination is that of the seventh edition of 1959, from which the English translation was made, and is unaltered in the eighth and ninth. The passage runs: „Weil die alte Anschauung ein Bild des Ganzen gab, die Fülle bewahrte, die Einheit von Leib und Seele nicht preisgab, bei allem Seelischen das Somatische mit im Auge hatte, bei allem Somatischen das Seelische, wurde sie bis heute gegen Descartes oft erneuert. So zuletzt in den ‚Psychoiden‘ Bleulers. Er wollte im Begriff der Psychoiden das dem somatischen Leben und der Seele Gemeinsame zusammenhalten: die mnemischen Funktionen, die Integration und die Zweckhaftigkeit der Strukturen und Kräfte. Der Mangel aber ist hier, wie jedesmal bei solchen Entwürfen, daß mit der Totalanschauung wohl ein Schema der Idee, aber nicht ein erforschbarer Gegenstand

wirklicher Erkenntnis gewonnen wird.“ On the object of the criticism — the *Psychoide* of 1925 and not the *Naturgeschichte der Seele* of 1921 — see the reservation entered in the text above. (Cited at 7.)

[11] Aristotle. *De Memoria et Reminiscentia* (*Parva Naturalia*); *Nicomachean Ethics*; *Politica*; *Organon*. English edition: Barnes, J. (ed.), *The Complete Works of Aristotle*. Princeton: Princeton University Press, 1984. French edition: Aristote, *Œuvres complètes*, trans. P. Pellegrin et al. Paris: Flammarion, 2014. (Cited at 5.)

[12] Hobbes, T. *Leviathan* (1651), ed. R. Tuck. Cambridge: Cambridge University Press, 1991. Part I, ch. 3, “Of the Consequence or Train of Imaginations.” (Cited at 5.)

[13] Locke, J. *An Essay Concerning Human Understanding* (1690; 4th ed. 1700), ed. P. H. Niddich. Oxford: Clarendon Press, 1975. Book II, ch. 33, “Of the Association of Ideas.” (Cited at 5.)

[14] Hume, D. *A Treatise of Human Nature* (1739), ed. L. A. Selby-Bigge. Oxford: Clarendon Press, 1978.

Book I, Part I, § 4, “Of the Connexion or Association of Ideas.” (Cited at 5.)

[15] Kant, I. *Kritik der reinen Vernunft* (1781; 2. Aufl. 1787), ed. J. Timmermann. Hamburg: Meiner, 1998. *Transzendente Analytik*, § § 15–20 (*Transzendente Einheit der Apperzeption*). French translation: A. Renaut, *Critique de la raison pure*. Paris: Flammarion, 2006. (Cited at 5.)

[16] Herbart, J. F. *Psychologie als Wissenschaft, neu gegründet auf Erfahrung, Metaphysik und Mathematik*, 2 vols. Königsberg: Unzer, 1824–1825. (Cited at 5.)

[17] Griesinger, W. *Pathologie und Therapie der psychischen Krankheiten*. Stuttgart: Krabbe, 1845; 2., umgearbeitete und erweiterte Auflage, 1861. French translation: A. Doumic, *Traité des maladies mentales*. Paris: Delahaye, 1865. (Cited at 5.)

[18] Wundt, W. *Grundzüge der physiologischen Psychologie*. Leipzig: Engelmann, 1874; 6. Aufl., 3 vols., 1908–1911. (Cited at 5.)

[19] Bleuler, E. « La Schizophrénie. » Rapport de psychiatrie, Congrès des Médecins Aliénistes et Neurologistes de France et des Pays de Langue Française, XXX^e Session, Genève–Lausanne, 2–7 août 1926. Paris: Masson et Cie, 1926. Reprinted in: Bleuler, E. & Claude, H. *La schizophrénie en débat*. Paris: L'Harmattan, 2001. The reprint preserves the full discussion, including Courbon's objection to the Congo example, and is the text used here. (Cited at 2.)

[20] Kapur, S. “Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia.” *American Journal of Psychiatry*, 160(1), 2003, pp. 13–23. (Cited at 2 and 6.1.)

[21] Fletcher, P. C. & Frith, C. D. “Perceiving is believing: a Bayesian approach to explaining the positive symptoms of schizophrenia.” *Nature Reviews Neuroscience*, 10(1), 2009, pp. 48–58. (Cited at 2 and 6.1.)

[22] Cross-Disorder Group of the Psychiatric Genomics Consortium. “Identification of risk loci with shared effects on five major psychiatric disorders: a genome-wide analysis.” *The Lancet*, 381(9875), 2013, pp. 1371–1379. (Cited at 8.)

[23] Tang, S. X., Kriz, R., Cho, S., et al. “Natural language processing methods are sensitive to sub-clinical linguistic differences in schizophrenia spectrum disorders.” *npj Schizophrenia*, 7, 2021, art. 25. Sentence-embedding distances detect tangentiality that clinical rating scales do not; the paper opens by citing Bleuler on the disturbance of language. The authors themselves note the small samples and the risk of overfitting. (Cited at 2 and 8.)

[24] *The measurement literature, in brief*. Spitzer, M. “A cognitive neuroscience view of schizophrenic thought disorder,” *Schizophrenia Bulletin*, 23(1), 1997, pp. 29–50, which opens: “The experimental

association psychology approach to mental associations has been the conceptual background for the concept of schizophrenia”; Spitzer, M., Braun, U., Hermle, L. & Maier, S. “Associative semantic network dysfunction in thought-disordered schizophrenic patients: direct evidence from indirect semantic priming,” *Biological Psychiatry*, 34(12), 1993, pp. 864–877 — against which must be set the meta-analysis of Pomarol-Clotet, E. et al., “Semantic priming in schizophrenia,” *British Journal of Psychiatry*, 192, 2008, pp. 92–97, which finds the hyperpriming evidence inconsistent outside thought-disordered subgroups. For the computational line: Ellevåg, B., Foltz, P. W., Weinberger, D. R. & Goldberg, T. E., *Schizophrenia Research*, 93, 2007, pp. 304–316; Bedi, G. et al., *npj Schizophrenia*, 1, 2015, art. 15030 (five converters only, as the authors state); Corcoran, C. M. et al., *World Psychiatry*, 17(1), 2018, pp. 67–75; Mota, N. B. et al., *PLoS ONE*, 7(4), 2012, e34928,

and *npj Schizophrenia*, 3, 2017, art. 18. For the standing caution: Hitczenko, K., Mittal, V. A. & Goldrick, M., *Schizophrenia Bulletin*, 47(2), 2021, pp. 344–362 (“a better understanding of when and why these models fail will be crucial”); de Boer, J. N. et al., *Current Opinion in Psychiatry*, 33(3), 2020, pp. 212–218, which likewise cites Bleuler and warns of overfitting and the absence of independent validation sets. (Cited at 2 and 8.)

[25] The citation record of the 1911 monograph. The present author has searched the full text of *Dementia praecox oder Gruppe der Schizophrenien* (the copy digitized from the Wellcome Collection original) for the names at issue, inflected forms included. Kant, Herbart and Griesinger do not occur. Neither does the term *Assoziationspsychologie*: Bleuler uses the vocabulary of association as unmarked common property and nowhere names a school. Wundt occurs once, and at second hand — in the survey of rival theories, as the source of

the *Apperzeption* whose disturbance Weygandt had proposed and Bleuler rejects. Ziehen, by contrast, is cited a dozen times and under several distinct works (entries 840–847 of Bleuler’s own bibliography). Wernicke’s *Sejunktion* is discussed and set aside as too anatomical-physiological a term for what Bleuler means by *Spaltung*. Page references are to the digitized copy and await collation against the printed pagination. (Cited at 5.)

[26] Ziehen, Th. *Leitfaden der physiologischen Psychologie*. Jena: Gustav Fischer, 1891, and many later editions. The declaration of allegiance stands in the preface; the announced purpose of the book is to show that Wundt’s *Apperzeption* is a superfluous auxiliary quantity. The edition consulted awaits final verification. (Cited at 5.)

[27] Bleuler, E. *Naturgeschichte der Seele und ihres Bewußtwerdens*, ref. [6], S. 201 (against Kant’s *synthetisches Vermögen der Einbildungskraft*), S. 211 („unterscheiden uns aber dabei wesentlich von Kant“), S. 215

(the counter-thesis on causality), and the section „A priori und a posteriori, Organisation und Erfahrung“, S. 227ff., where the distinction of *phyloplastisch* from *ontoplastisch* reactions naturalizes the a priori as phylogenetically sedimented experience. Herbart is not named in this book either. The passages are read in detail in Paper III of this series, *The Natural History of the Soul*. (Cited at 5.)

[28] On Griesinger's debt to Herbart. Griesinger names Herbart in the psychological chapters: see *Mental Pathology and Therapeutics*, trans. C. L. Robertson & J. Rutherford, London: New Sydenham Society, 1867 (from the second German edition of 1861), p. 27n. For the scholarship: Verwey, G. *Psychiatry in an Anthropological and Biomedical Context: Philosophical Presuppositions and Implications of German Psychiatry, 1820–1870*. Dordrecht: Reidel, 1985, ch. 3, pp. 86–155; Arens, K. “Wilhelm Griesinger: psychiatry between philosophy and praxis,” *Philosophy, Psychiatry, &*

Psychology, 3(3), 1996, pp. 147–163; Marx, O. M. “Nineteenth-century medical psychology: theoretical problems in the work of Griesinger, Meynert, and Wernicke,” *Isis*, 61(3), 1970, pp. 355–370, and “Wilhelm Griesinger and the history of psychiatry: a reassessment,” *Bulletin of the History of Medicine*, 46(6), 1972, pp. 519–544; Wahrig-Schmidt, B. *Der junge Wilhelm Griesinger im Spannungsfeld zwischen Philosophie und Physiologie*. Tübingen: Gunter Narr, 1986. Griesinger's eclecticism should be recorded in the same breath: the mechanics of representations stands in his work beside the reflex physiology of the Müller school and an explicit refusal of localization. (Cited at 5.)

[29] Hoff, P. “Eugen Bleuler's concept of schizophrenia and its relevance to present-day psychiatry,” *Neuropsychobiology*, 66(1), 2012, pp. 6–13: “As for the theoretical foundations of Bleuler's thinking on schizophrenia, 3 authors have to be mentioned: Johann Friedrich Herbart (1776–1841),

Emil Kraepelin (1856–1926) and Sigmund Freud (1856–1939).” Hoff enters no primary citation of Bleuler on Herbart, and his own footnote 3 recalls that the tradition of association theory is much broader, naming Hume — so that the attribution is a scholarly judgment about a shared background and not a claim of documented reading. See also Maatz, A., Hoff, P. & Angst, J. “Eugen Bleuler’s schizophrenia — a modern perspective,” *Dialogues in Clinical Neuroscience*, 17(1), 2015, pp. 43–49; and Moskowitz, A. & Heim, G. “Eugen Bleuler’s *Dementia Praecox or the Group of Schizophrenias* (1911): a centenary appreciation and reconsideration,” *Schizophrenia Bulletin*, 37(3), 2011, pp. 471–479: “Bleuler’s concept arose out of the association psychology of the 19th century, the dominant psychological paradigm of his time.” The contrary estimate deserves recording: Stotz-Ingenlath, G. “Epistemological aspects of Eugen Bleuler’s conception of schizophrenia in 1911,” *Medicine, Health Care and*

Philosophy, 3(2), 2000, pp. 153–159, which describes Bleuler as “methodologically... an empiricist and an eclecticist” and whose apparatus names neither Kant nor Herbart nor Griesinger. (Cited at 5.)

[30] Skinner, Q. “Meaning and understanding in the history of ideas,” *History and Theory*, 8(1), 1969, pp. 3–53 (the mythologies of doctrines, of coherence and of prolepsis); Berrios, G. E. “Eugen Bleuler’s place in the history of psychiatry,” *Schizophrenia Bulletin*, 37(6), 2011, pp. 1095–1098, which warns, in a text on Bleuler himself, against positioning earlier figures as “anticipators or pioneers of a current truth”; Berrios, G. E. *The History of Mental Symptoms: Descriptive Psychopathology since the Nineteenth Century*. Cambridge: Cambridge University Press, 1996; Butterfield, H. *The Whig Interpretation of History*. London: G. Bell & Sons, 1931. (Cited at 5.)

[31] Sterzer, P., Adams, R. A., Fletcher, P., Frith, C., Lawrie, S. M., Muckli, L., Petrovic, P.,

Uhlhaas, P., Voss, M. & Corlett, P. R. “The predictive coding account of psychosis.” *Biological Psychiatry*, 84(9), 2018, pp. 634–643. The authors write that hallucinations “represent a challenge, as two apparently opposing aberrations have been proposed and there is evidence supporting both”; that “an overly flexible or general theory that explains everything will be of little use”; and that “while there is abundant empirical evidence compatible with a predictive coding account, more research is needed that explicitly tests (and potentially falsifies) predictions derived from this theory.” For the state of the question since: Goodwin, I., Diederer, K. M. J., et al. “Predictive processing accounts of psychosis: bottom-up or top-down disruptions,” *Nature Mental Health*, 4, 2026, pp. 60–84, which reports the findings heterogeneous and the direction of the central abnormality unresolved. Neither paper mentions Bleuler. (Cited at 6.1 and 8.)

[32] Litwin, P. & Miłkowski, M. “Unification by fiat: arrested development of predictive processing,” *Cognitive Science*, 44(7), 2020, e12867; Miłkowski, M. & Litwin, P. “Testable or bust: theoretical lessons for predictive processing,” *Synthese*, 200, 2022, art. 462 (“if a theory is so flexible that it can predict just anything, it carries no empirical information”); Colombo, M., Elkin, L. & Hartmann, S. “Being realist about Bayes, and the predictive processing theory of mind,” *British Journal for the Philosophy of Science*, 72(1), 2021, pp. 185–220 (priors and likelihoods selected post hoc to accommodate rather than to explain); Sun, Z. & Firestone, C. “The dark room problem,” *Trends in Cognitive Sciences*, 24(5), 2020, pp. 346–348; Williams, D. “Hierarchical Bayesian models of delusion,” *Consciousness and Cognition*, 61, 2018, pp. 129–147, on the circumscription of delusions. Replies from within the programme exist and should be read beside these. (Cited at 6.1.)

[33] Corlett, P. R. & Fraser, K. M. “20 years of aberrant salience in psychosis: what have we learned?” *American Journal of Psychiatry*, 182(9), 2025, pp. 819–829. On the Salience Attribution Test: “There are no differences in aberrant salience between patients with delusions and healthy controls”; on the anatomy, the dopamine dysfunction is located in the head of the caudate and the associative striatum rather than where the model implies; and the verdict, “we believe that a strong incentive salience theory of delusions (and hallucinations) cannot hold.” That the theory has itself been repeatedly revised should be borne in mind: Howes, O. D. & Kapur, S. “The dopamine hypothesis of schizophrenia: version III – the final common pathway,” *Schizophrenia Bulletin*, 35(3), 2009, pp. 549–562. (Cited at 6.1 and 8.)

[34] Stephan, K. E., Friston, K. J. & Frith, C. D. “Dysconnection in schizophrenia: from abnormal synaptic plasticity to failures of self-monitoring,” *Schizophrenia*

Bulletin, 35(3), 2009, pp. 509–527: “Subsequently, a similar notion, but formulated in terms of psychopathology, was expressed by Bleuler who coined the term ‘schizophrenia’ to denote the ‘splitting’ of different mental domains.” Friston, K., Brown, H. R., Zeidman, P. & Razi, A. “The dysconnection hypothesis (2016),” *Schizophrenia Research*, 176(2–3), 2016, pp. 83–94, which distinguishes anatomical dysconnection (Wernicke’s sejunction) from functional dysconnection (Bleuler’s disintegration of the psyche), while conceding an explanatory gap and calling its own synthesis “rather speculative.” The one sustained attempt to connect the classical Bleulerian symptomatology to predictive coding is offered as a hypothesis: Liddle, P. F. & Liddle, E. B. “Imprecise predictive coding is at the core of classical schizophrenia,” *Frontiers in Human Neuroscience*, 16, 2022, art. 818711. Bleuler is, so far as the present author can establish, absent from the

citation genealogy predictive coding acknowledges for itself, which runs from Kant through Helmholtz and Gregory to Friston: see Swanson, L. R. “The predictive processing

paradigm has roots in Kant,” *Frontiers in Systems Neuroscience*, 10, 2016, art. 79. (Cited at 8.)

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