THE RELATIONSHIP OF DEMENTIA PRÆCOX TO MENTAL DEFICIENCY.*

By STANLEY G. JAMES, M.D.Sydney, M.R.C.P.Lond., D.P.M.,
Deputy Medical Superintendent, Rampton State Institution, near Retford, Notts.

INTRODUCTION.

FROM a clinical point of view, mental deficiency and insanity are usually considered to be separate disorders. The members of each group who require institutional care are collected in separate hospitals and the methods of treatment are on different lines.

In spite of intensive clinical and pathological research the actual causes of the two disorders are unknown in the majority of cases. The pathology of a few small groups in both mental deficiency and insanity is well understood, but this knowledge has not thrown the light that was to be hoped for on the greater bulk of the disorders. It is possible that for this greater proportion the cause is the same in each, and to strengthen this view mental deficiency and a particular variety of insanity can be shown to have a closer relationship than is perhaps generally realized.

This relationship is inferred principally from the consideration of a special variety of mental deficiency, namely, that which is associated with dangerous and violent propensities. Many will doubtless be of the opinion that there is no such special variety, but I submit that such opinion arises from lack of opportunity of studying the condition collectively, since there are only two institutions in this country, the State Institutions, in which this may be done.

CLINICAL MATERIAL.

The special personality of the State Institution patient is characterized by undue sensitiveness to mild provocation, pride, intolerance, euphoria, instability leading to quick resentment or rebellion, insolence or deference, and responsiveness to reward and to withdrawal of reward. It may be objected that these features are those of an ordinary defective and that it is just a question of degree. This may be so, but the extent to which they are carried is so great that, from a practical point of view, they represent a special type.

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The outstanding feature is the extreme emotional instability leading to extreme disorder of conduct. In some instances there is deficient moral sense or inability to appreciate right and wrong, and in others the moral sense is not appreciably lacking, but there is still little or no control over behaviour. In the phase of excitement the patient may be quarrelsome, noisy, screaming or singing at the top of the voice, or truculent and violent, banging, smashing, or otherwise destroying clothing and property, with extreme depravity of personal habits. In the phase of depression, transient ideas of persecution and attempts at self injury or suicide are encountered. Curious instances occur of a mixture of the two phases, features of both being present at the same time.

In the majority of cases the outbursts are periodic.

The emotional changes appear to be the cyclothymic experiences of a person of sound mind carried to an abnormal degree, and one concludes that one is dealing with a number of manic-depressives. This conclusion is proved inaccurate by the observation that, even at the height of the manic phase or the depth of the depressive phase, there is no loss of insight or lack of accessibility. Identity, circumstances and environment are in full consciousness, and are undistorted and not the least confused. In fact, particular attention to the surroundings is always much in evidence.

A considerable number of partly developed and fully developed psychoses are, however, encountered. These may be considered to have developed on top of previously existing deficiency or they may be psychoses which began at an early age and remained indistinguishable, during childhood, from deficiency with dangerous and violent propensities. My own observations lead me to adopt the second explanation, and to regard this special variety of mental deficiency as intermediate between and linking together ordinary deficiency and insanity. In many instances, particularly in the dementia præcox group, the clinical picture is finally that of pure psychosis as found in a mental hospital, with no trace of mental deficiency at all. Such of these whose best interests appear to be served by so doing are removed to mental hospitals, under Section 16 of the Principal Act.

With a view to investigating the incidence of insanity among cases of mental deficiency with dangerous and violent propensities, the patients admitted to Rampton State Institution during the past seventeen years have been reviewed. The total number of admissions is 2,382. The present population of 1,207 has been investigated personally and information concerning the remaining 1,175 admissions has been obtained by an examination of the records. The results of the investigation are set out in the following tables.

In the first column are the serial numbers of the cases and the ages given are those at the present time. The column headed "crime" indicates convictions, and "stigmata" refers to the physical abnormalities of congenital origin that are associated with mental deficiency.

TABLE I.—*Female Patients with Signs of Dementia Præcox.*

No.	Age.	Crime.	Stigmata.	Hallucinations.	Delusions.	Type.
1	46	+	+	+	+	Para.
2	54	+	−	−	−	Kata.
3	41	+	−	−	+	,,
4	47	+	−	+	+	Para.
5	42	+	−	−	+	Kata.
6	39	+	+	−	+	Para.
7	34	+	−	+	−	Kata.
8	40	+	−	−	+	Para.
9	47	+	+	−	+	,,
10	31	+	−	−	−	Kata.
11	26	+	−	−	+	Para.
12	27	+	−	−	−	Hebe.
13	33	+	+	+	+	Kata.
14	47	+	−	−	+	Para.
15	33	+	−	−	−	Kata.
16	35	+	−	−	+	Para.
17	36	+	−	+	−	Kata.
18	33	+	−	+	−	,,
19	39	+	−	−	+	,,
20	30	+	+	+	−	Hebe.
21	40	+	−	−	+	Para.
22	33	+	−	−	+	,,
23	30	+	+	−	+	Kata.
24	35	+	−	−	+	,,
25	39	+	+	+	+	,,
26	32	+	−	−	+	Para.
27	48	+	−	−	+	,,
28	46	+	−	+	+	,,
29	31	+	−	−	+	,,
30	30	+	−	+	+	Kata.
31	..	+	−	−	+	Para.
32	27	+	−	+	+	Kata.
33	31	+	+	−	+	Para.
34	36	+	−	+	+	Kata.
35	33	+	−	−	+	Para.
36	20	+	−	+	+	Kata.
37	35	+	+	−	−	,,
38	27	+	−	−	+	Para.
39	26	+	−	+	+	Kata.
40	25	±	−	−	+	Para.
41	32	+	+	+	+	,,
42	34	−	+	−	+	Kata.
43	46	+	−	+	−	Para.
44	32	+	−	+	−	Kata.
45	33	+	−	−	−	,,
46	30	+	−	−	−	,,
47	25	−	−	−	−	,,
48	17	−	−	−	−	,,
49	19	−	+	+	+	,,
50	29	−	−	+	+	,,
51	24	+	−	−	+	Para.
52	34	+	−	−	−	Kata.
53	49	±	−	+	+	Para.
54	40	+	−	−	+	,,
55	37	+	−	+	+	,,
56	18	−	−	+	+	Kata.

TOTAL, 56.

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TABLE II.—*Male Patients with Signs of Dementia Præcox.*

No.	Age.	Crime.	Stigmata.	Hallucinations.	Delusions.	Type.
57	44	+	−	−	+	Kata.
58	43	+	+	−	+	Simp.
59	36	+	−	−	+	Para.
60	44	+	−	+	+	,,
61	37	±	−	−	+	,,
62	44	+	+	+	−	Kata.
63	42	+	−	+	+	Para.
64	34	+	−	−	+	Kata.
65	40	+	−	+	+	,,
66	32	+	+	+	+	,,
67	34	+	+	+	+	Para.
68	40	+	−	+	+	,,
69	34	+	−	−	+	,,
70	33	−	+	+	+	,,
71	36	+	−	−	+	,,
72	33	+	−	−	+	,,
73	31	+	−	+	+	Kata.
74	33	+	+	+	+	,,
75	32	+	+	−	+	,,
76	35	+	+	+	+	,,
77	30	+	−	+	+	,,
78	33	+	+	+	+	Para.
79	38	+	+	−	+	,,
80	29	+	−	+	+	Kata.
81	29	+	+	+	+	,,
82	29	+	−	−	+	Para.
83	43	+	−	−	+	,,
84	33	+	+	−	+	,,
85	52	+	−	+	−	Kata.
86	31	−	+	−	+	,,
87	38	+	−	+	+	Para.
88	32	−	−	−	+	,,
89	32	+	+	+	+	,,
90	27	+	−	+	−	Simp.
91	26	+	−	−	+	Para.
92	33	±	−	+	+	,,
93	32	+	−	+	+	Kata.
94	35	−	−	+	+	Para.
95	17	+	−	+	+	Kata.
96	34	+	−	+	+	Para.
97	23	−	−	+	−	Simp.
98	29	+	−	+	+	Para.
99	32	+	−	−	+	,,
100	25	+	−	+	−	Kata.
101	25	−	+	−	+	,,
102	27	+	−	+	−	,,
103	25	+	−	−	+	Para.
104	29	±	−	−	+	,,
105	29	−	−	+	+	Kata.
106	21	−	+	+	+	Para.
107	26	−	+	+	+	Kata.
108	14	−	−	+	−	Simp.

TOTAL, 52.

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TABLE III.—*Female Dementia Præcox Patients Certified, Section* 16.

No.	Age.	Crime.	Stigmata.	Hallucinations.	Delusions.	Type.
109	41	+	−	+	+	Para.
110	48	+	+	+	+	Kata.
111	35	−	−	+	−	,,
112	47	−	−	+	+	,,
113	..	−	−	+	+	,,
114	33	−	−	+	−	,,
115	40	+	+	+	+	Para.
116	40	+	+	+	+	Kata.
117	38	+	−	−	+	,,
118	45	−	−	+	+	,,
119	41	±	−	±	+	,,
120	31	−	+	−	+	,,
121	39	−	−	−	+	,,
122	34	+	−	+	+	,,
123	37	−	−	−	+	,,
124	41	+	−	+	+	,,
125	31	−	−	+	+	,,
126	28	−	±	+	+	,,
127	31	+	−	−	+	,,
128	31	+	−	+	−	,,
129	28	−	+	+	+	,,
130	..	−	−	+	−	,,
131	26	−	−	+	+	,,
132	34	+	−	−	+	,,
133	30	+	+	−	+	Para.
134	42	+	−	+	+	Kata.

TOTAL, 26.

TABLE IV.—*Male Dementia Præcox Patients Certified, Section* 16.

No.	Age.	Crime.	Stigmata.	Hallucinations.	Delusions.	Type.
135	52	+	+	+	−	Kata.
136	77	+	+	+	−	,,
137	50	+	−	−	+	Para.
138	51	+	+	+	+	Kata.
139	..	+	+	+	+	,,
140	36	+	+	+	+	,,
141	37	+	−	+	+	,,
142	37	+	−	−	+	Para.
143	39	+	−	+	+	Kata.
144	32	+	−	+	+	,,
145	33	−	+	+	+	,,
146	37	−	+	+	+	,,
147	42	+	−	−	+	,,
148	39	+	+	+	+	Kata.
149	31	−	+	−	+	,,
150	48	+	+	−	+	Para.
151	31	−	+	−	−	Simp.
152	..	−	+	+	−	,,
153	43	+	+	+	+	Kata.
154	29	−	−	+	+	,,
155	32	−	+	+	−	Simp.
156	28	+	−	−	+	Para.
157	37	+	+	+	+	Kata.
158	32	+	−	+	+	,,
159	29	+	−	+	+	,,
160	22	+	−	−	+	Para.
161	34	−	−	+	+	,,

TOTAL, 27.

TABLE V.—*Female Dementia Præcox Patients Discharged or Dead.*

No.	Age.	Crime.	Stigmata.	Hallucinations.	Delusions.	Type.
162	45	+	−	−	+	Kata.
163	41	+	−	+	+	,,
164	36	−	−	+	−	,,
165	34	−	−	−	+	Para.
166	47	−	−	−	−	Kata.
167	29	+	−(died)	+	+	Hebe.
168	55	+	−	+	+	Kata.
169	46	−	−	−	−	,,
170	37	−	−	−	−	,,
171	24	−	−(died)	−	+	Simp.
172	25	−	− ,,	+	+	Kata.
173	41	+	−	−	+	Para.
174	40	+	−	−	+	Kata.
175	42	+	+	−	+	Hebe.
176	47	−	−	−	+	Para.

TOTAL, 15.

TABLE VI.—*Male Dementia Præcox Patients Discharged or Dead.*

No.	Age.	Crime.	Stigmata.	Hallucinations.	Delusions.	Type.
177	38	+	−	−	+	Para.
178	40	+	−	−	+	,,
179	39	+	+	+	+	,,
180	42	+	+	−	+	,,
181	37	+	−	+	+	,,
182	34	−	+	+	+	Simp.
183	38	+	−	+	−	,,
184	38	+	+	+	−	,,
185	32	+	−	+	−	Hebe.
186	24	+	−	+	−	Simp.
187	40	−	−	+	−	,,
188	27	−	−	+	+	Kata.
189	31	−	−	+	−	Simp.
190	37	+	−	+	−	,,
191	34	−	−	+	+	Para.
192	31	−	+(died)	−	+	Kata.

TOTAL, 16.

TABLE VII.—*Female Patients with Signs of Other Psychoses.*

No.	Age.	Crime.	Stigmata.	Hallucinations.	Delusions.	Type.
193	39	+	−	−	+	Manic-dep.
194	37	+	−	−	−	Melancholia.
195	32	+	+	−	+	,,
196	31	+	+	−	+	Manic-dep.
197	34	+	−	−	+	Melancholia.
198	57	+	+	−	−	Mania.
199	40	+	−	−	−	Melancholia.
200	36	+	+	−	+	Manic-dep.
201	31	+	+	−	+	,,
202	43	+	−	−	−	Melancholia.
203	26	+	−	−	−	Mania.
204	21	+	+	−	+	,,
205	26	+	−	−	+	Manic-dep.
206	62	+	−	−	+	,,
207	39	+	+	−	+	Mania.
208	24	+	+	−	+	Manic-dep.
209	31	+	−	−	+	,,
210	23	+	−	−	−	,,

TOTAL, 18.

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TABLE VIII.—*Male Patients with Signs of Other Psychoses.*

No.	Age.	Crime.	Stigmata.	Hallucinations.	Delusions.	Type.
211	71	+	+	—	+	Dementia.
212	50	+	—	+	+	N.S. del. ins.
213	57	+	+	—	—	Dementia.
214	56	+	—	+	+	N.S. del. ins.
215	73	+	+	+	+	,,
216	51	+	+	—	+	Alc. dementia.
217	45	+	+	—	+	Manic-dep.
218	37	+	+	—	—	,,
219	39	+	+	+	+	Chr. mania.
220	41	+	+	—	—	Melancholia.
221	43	+	+	—	+	Juvenile G.P.I.
222	42	+	—	+	+	N.S. del. ins.
223	55	+	—	—	+	,,
224	57	+	+	—	+	Dementia.
225	43	+	+	—	+	N.S. del. ins.
226	50	+	—	—	+	Dementia.
227	26	+	—	+	+	? G.P.I.
228	48	+	—	—	+	N.S. del. ins.
229	14	—	—	—	—	Ins. c̄ epi.

TOTAL, 19.

TABLE IX.—*Female Other Psychoses Certified, Section* 16.

No.	Age.	Crime.	Stigmata.	Hallucinations.	Delusions.	Type.
230	43	+	—	—	—	Manic-dep.
231	..	+	—	—	+	Paranoia.
232	42	—	—	—	—	Manic-dep.

TOTAL, 3.

TABLE X.—*Male Other Psychoses Certified, Section* 16.

No.	Age.	Crime.	Stigmata.	Hallucinations.	Delusions.	Type.
233	62	+	+	—	—	Dementia.
234	60	+	+	—	—	,,
235	59	+	—	—	+	N.S. del. ins.
236	..	+	—	+	+	Ins. c̄ epi.
237	76	+	+	+	+	Dementia.
238	42	+	+	—	+	Obsess. ins.
239	28	+	+	—	+	Ins. c̄ epi.
240	36	—	—	+	+	Juvenile G.P.I.
241	..	+	+	+	+	N.S. del. ins.
242	55	+	—	+	+	,,
243	52	—	+	—	+	Ins. c̄ epi.
244	60	+	+	—	+	N.S. del. ins.
245	28	+	+	—	—	Ins. c̄ epi.
246	41	+	—	—	+	Manic-dep.

TOTAL, 14.

TABLE XI.—*Female Other Psychoses Discharged.*

No.	Age.	Crime.	Stigmata.	Hallucinations.	Delusions.	Type.
247	38	—	—	+	+	Manic-dep.
248	66	—	—	+	+	Alc. dementia.
249	36	+	—	—	—	Manic-dep.
250	34	—	—	+	—	Conf. ins.

TOTAL, 4.

TABLE XII.—*Male Other Psychoses Discharged or Dead.*

No.	Age.	Crime.	Stigmata.	Hallucinations.	Delusions.	Type.
251	74	+	—	—	—	Dementia.
252	72	+	—(died).	—	—	,,
253	63	+	—(,,).	—	—	,,
254	65	+	+(,,).	—	+	Melancholia.
255	23	+	—(,,).	—	—	Ins. c̄ G.B.L.
256	60	+	+	—	+	N.S. del. ins.
257	28	+	+(,,).	—	+	Ins. c̄ epi.
258	26	—	—(,,).	—	+	G.P.I.
259	45	+	—	+	+	,,
260	55	+	—	—	—	Dementia.

TOTAL, 10.

TABLE XIII.—*Summary of Results.*

	Male.	Female.	Total.
TOTAL ADMISSIONS:			
March, 1920–September, 1937	1290	1092	2382
PRESENT POPULATION:			
Mental defective	643	419	1062
,, ,, with signs of psychosis	71	74	145
,, ,, ,, dementia præcox	52	56	108
,, ,, ,, other psychoses	19	18	37
Total	714	493	1207
DISCHARGED OR DEAD:			
Mental defective	509	551	1060
,, ,, with signs of psychosis	67	48	115
,, ,, ,, dementia præcox	16	15	31
,, ,, ,, other psychoses	10	4	14
,, ,, ,, dementia præcox, Sect. 16	27	26	53
,, ,, ,, other psychoses, Sect. 16	14	3	17
Total	576	599	1175
TOTAL MENTAL DEFECTIVE	1152	970	2122 = 89%
Total mental defective			
,, ,, ,, with signs of psychosis	138	122	260 = 11%
,, ,, ,, ,, dementia præcox	95	97	192 = 8%
,, ,, ,, ,, other psychoses	43	25	68 = 3%
Total	1290	1092	2382

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In Table XIII the figures for the total admissions, apart from those for the present population, are not accurate owing to the inclusion of a number of re-admissions. This error causes the rough percentage figures for the psychoses to be slightly smaller than they should be. The psychoses have been divided into two groups, dementia præcox and other psychoses, owing to the preponderance of the former. Opinions may differ as to what is a dementia præcox, and while the figures for the present population are the result of my personal observations, those for the remainder are taken from records made by others. Only those cases in which the signs of psychosis were clear and definite have been included, and the details set out in the tables are only a part of the evidence. In the dementia præcox groups all the cases showed signs of dissociation at one time or another and many had mannerisms and other signs of the disorder. It will be seen that the proportion of psychotics to defectives in the present population agrees roughly with that in the remainder of admissions, as also do the relative numbers of dementia præcox, so that my own observations are more or less in accord with those of others in this connection.

Discussion of Results.

The results, summarized in Table XIII, show that in a large group of mental defectives with dangerous and violent propensities, 11% have definite signs of psychosis. Of these psychoses more than 75% are found to be dementia præcox and the remainder consists of a group in which no one variety is particularly prevalent.

These findings deserve attention and suggest either that psychosis is liable to supervene in a considerable number of cases of this special variety of mental deficiency or that there is a relatively large group of psychoses which begins at a very early age and is at first indistinguishable from mental defect. Furthermore, dementia præcox, from its remarkably high incidence, is the type of psychosis which merits most consideration in this connection.

Although dementia præcox is the most common type of insanity met with, and Tanzi (1909) says it includes at least 25% of the patients confined in mental hospitals, one would not have expected a figure of 75%. It is clear that the series of psychoses here recorded is not one that is commonly encountered. Dementia præcox is the earliest of the insanities in point of age incidence. Most cases begin during adolescence and it is estimated that two-thirds develop between the ages of fifteen and thirty.

Perusal of the early records of each case of psychosis shows that at the time of certification under the Mental Deficiency Acts there was little or no evidence of insanity. In almost all instances the rate of manifestation has been gradual. In the light of after developments it is easy to assume, on looking into the history, that the condition should have been recognized earlier. In comparison with the ordinary type of deficiency this might be the case, but

the difference between early insanity and this special type of deficiency is much less obvious.

Clinical Relationship.

The literature contains many references to the relationship between mental deficiency and insanity. Tredgold (1929*a*) remarks on the separation of the two from the point of view of the law, the institution, medical science and the medical services. He says that the differential diagnosis may be very difficult and draws attention to the relationship. He further points out certain facts which justify the view that mental deficiency and certain forms of mental disease are actually manifestations of one and the same underlying process. In making the comparison, he rightly insists that the issue shall be confined to primary mental deficiency and primary mental disorder. It is clear that the small but diverse group of inflammatory and vascular conditions which may give rise either to deficiency or to insanity does not deserve consideration in this connection.

Tredgold's classification of defectives into three groups from the aspect of social adaptability is important and practical. His first group of stable, placid, unemotional, contented and industrious people of various intellectual levels is naturally to be found in the certified institutions. The second group is that of the unstable, whom we term defectives with dangerous and violent propensities. He says it has long been recognized, and that instances of it are to be found in all Certified Institutions. He might have added that some 1,500 are to be found in the two State Institutions. His third group is that in which retrogression occurs, and it consists of defectives whose intelligence and behaviour deteriorates after reaching a certain level. Some become stationary at a low level and others go on to complete dementia. It is especially interesting to note that he says that in the majority the first signs of deterioration appear shortly after puberty and in some instances even earlier. This is surely a particularly precocious dementia.

Tredgold goes on to point out that the stable group merges with the normal, and the unstable merges with the primary psychotic. He considers that it is a class which he calls "temperamental defectives" that constitutes the link with insanity. These are persons who have marked mental instability without intellectual defect and are not certifiable. In comparing the normal oscillations of emotion with those that occur in the unstable, he makes a remark of much significance. He says that the unstable not infrequently go so far as a dissociation of the personality. What can this suggest but a relation with dementia præcox? He says that such instability usually reveals itself in early years, and his description of the condition in children is hard to distinguish from one of dementia præcox at the same age.

In tracing the development of the unstable after adolescence he mentions three principal forms which the instability may take. In the emotional

form introspection is a pronounced feature. In the ideational form he emphasizes bizarre thoughts and lack of insight. The third form is that which results in overaction of primitive instincts. No one will disagree with his statement that it is from the inherently mentally unstable as a class that the insane come, but I cannot agree with the selection of types of mental disorder he says are related to the different forms of instability. The mild emotional is related to hysteria, anxiety states, manic-depressive insanity and hypochondriasis; but if marked introspection is evident, why not include katatonic dementia præcox? The mild ideational form is related to obsessional states, delusional insanity and paranoia; why not mention paranoid dementia præcox?

In discussing his retrogressive group of defectives, Tredgold says that it merges into certain forms of dementia. Dementia appears in two clinical types. In one, there is gradual failure of the powers of the mind without instability. This type is met with in oligophrenics and corresponds in all essentials with the simple type of dementia præcox as well as to senile dementia. In the other the process is irregular and leads to a disharmony similar to that of inherent instability. This type is also seen in mental defectives and, allowing for the difference of intellectual development, is essentially similar to the hebephrenic and paranoid forms of dementia præcox.

I submit that, from the above considerations, the unstable group is shown to be related to dementia præcox in particular.

Tredgold concludes this part of his article by remarking that the period of life which presents the most difficult diagnostic problems is that of adolescence, and the greatest difficulty at this time is to differentiate between high-grade oligophrenics, innate or acquired mental instability, with their incipient primary psychoses, and the early stages of such degenerative processes as dementia præcox and adolescent general paralysis of the insane.

In his textbook, Tredgold (1929*b*) says, with reference to mental deficiency in general, that the commonest psychosis met with is the manic-depressive. I am inclined to doubt this. My experience of mental defectives in a mental hospital is that the psychosis seen differs considerably from manic-depressive insanity, and the result of this investigation of unstable defectives in the State Institution shows clearly that dementia præcox is the commonest type. Tredgold also remarks that in a comparatively short time, much shorter than in the non-defective, dementia makes its appearance. He says that well-marked auditory or visual hallucinations occur, whilst a majority suffer from delusions, and paranoid types and cases of stupor are met with. I think that all these points are in favour of dementia præcox. Onset early in life, early dementia, hallucinations and stupor are not features of manic-depressive insanity.

The literature also contains a number of references to dementia præcox occurring in childhood. Mott (1920) refers to the question and says that in his opinion it may begin in some cases before puberty, even long before puberty.

He goes on to quote Kraepelin as pointing to the fact that in many cases clinical symptoms occur in pre-puberal life, so that it is legitimate to conclude that there are a number of cases which might be termed dementia præcossissima. Mott also says that a certain number of cases of dementia præcox occur in congenital "aments" or imbeciles, and that these might be termed dementia præcossissima, and from early childhood there are clinical indications of a failure of the higher neural functions.

An article by Potter (1933) deserves particular attention. He reports six personal cases of schizophrenia in children, reviews the literature, and finds records of some thirty-six other cases occurring before puberty. Potter's six cases all appear to be typical katatonic præcoxes. Five of them were apparently normal in their early years, and all had developed the typical psychosis by twelve years of age, the earliest being at four years. He remarks that five showed no physical indications of puberty.

Potter observes that the central nervous system in the child is unable to respond with the complexity of the adult in its functional expression. Therefore one should consider the facilities which children possess for expressing any form of psychopathology, and hence the delusional formations in childhood are relatively simple and their symbolization is particularly naïve. He refers to the fact that in primitive races mental disease, and especially dementia præcox, is uniformly simple in its clinical expression. He quotes Kraepelin, who found in the Javanese a preliminary period of confused excitement which rapidly passed into simple dementia; hallucinations were rare and delusional formation elementary and of uncomplicated construction.

Potter concludes that a typical schizophrenic reaction may appear long before puberty, that delusional formation is slight, simple and naïve, and that the outstanding symptomatology is in the field of behaviour and there is a consistent lack of emotional rapport. He says there is a superficial resemblance of schizophrenic children to certain so-called unstable mental defectives, that the schizophrenic child often appears to be mentally defective, and that a study of mental defectives in institutions would show that schizophrenia in children is not so rare as is now generally believed.

Richmond (1932) describes a clear-cut case of dementia præcox in adult life with some doubt as to whether the patient was a defective during adolescence. She thinks that the question of the existence of psychosis in children has been lightly passed over by the average psychiatrist. She says that one of the types of personality consists of actual cases of dementia præcox, existing from birth or an early age, showing certain characteristics, and passing through a certain course of development. She refers to Kraepelin and his statement that a certain proportion of dementia præcox cases showed mental peculiarities in childhood, and that about 7% of his dementia simplex cases were weak-minded. She describes the type of behaviour-problem child, sometimes called potentially feeble-minded, whose intelligence appears normal in early life, and

who is, therefore, not defective. She says that some are cases with frank psychosis and others are more defective than psychotic. She concludes that there are cases of dementia præcox in children, often thought to be mental defectives, but fairly easily differentiated from them, and that they have a normal intelligence at first, but a poor one later.

Blau and Averbuck (1936) have described a case of catalepsy or flexibilitas cerea in a child aged 3.

From these considerations it is clear that dementia præcox occurring before puberty gives rise to some difficulty in distinguishing it from mental deficiency. Cases which develop at three and four years of age must be very difficult to determine unless well-marked, and I consider it would be quite impossible to say whether a child was defective or insane before these ages. With the exception of Tredgold the writers are concerned only with dementia præcox, which goes to show that the important relation is that between mental deficiency and dementia præcox.

Pathological Relationship.

The macroscopic, microscopic and chemical pathology of the brain in primary mental deficiency has been studied by many investigators. Particular mention may be made of the work of Bolton (1907, 1914, 1933), Berry (1932, 1935, 1936), Berry and Norman (1934), Ashby and Stewart (1933, 1934 1935), and Ashby and Glynn (1935). It has been found that the cerebral cortex is thinner than normal, especially in the pre-frontal and parietal lobes. In this thin cortex the cells are fewer, under-developed, and arranged in a disorderly fashion. As there are fewer cells, naturally there are fewer fibres. These workers have also observed that the above-mentioned abnormalities vary with the degree of mental defect and are most marked in the lowest grades.

The pathology of dementia præcox has been investigated by Mott (1920), Bolton (1907, 1914, 1933), Southard (1914), Forster (1917), Tiffany (1920), Morse (1923), Dunlap (1924), Golla (1929), and many others. Reference to it is made by Tredgold (1929*a*), Hoch (1910), Kraepelin (1919), Dawson (1928), Davidson (1934), and Conn (1934). There is considerable disagreement in the views of these writers, particularly in the case of the changes in the endocrine glands, but to a less extent as regards the brain.

Mott (1920) concurs with the views of Nissl and Alzheimer that the essential neuronic change is one of nuclear decay. He observes that the greater part of the neurones are living, but are so biochemically altered that a progressive disorder and loss of function results. He adds that the process may begin, in some cases, long before puberty. He further remarks that many cases of dementia præcox are congenital "aments", as shown by the fact that a number of the higher cortical neurones do not develop. Bolton (1914) also found

degenerative changes in the cortical neurones. He pointed out that the changes in the dementia præcox brain are parenchymatous and not interstitial, thus differing from other dementias.

Davidson (1934), in reviewing the question, quotes Spielmeyer and Dunlap, who consider that the brain-lesions of dementia præcox are non-specific, and says that this does not exclude an organic basis for the disorder but indicates a lack of data for an anatomical differential diagnosis. He believes that the underlying cause is a progressive disease of the brain. Hoch (1910) admits the presence of structural changes in the brain in dementia præcox, but prefers to disregard them and to adopt the non-organic view.

The pathological findings in dementia præcox are different from those in other psychoses, and, in comparison with the picture in mental deficiency, are not incompatible with the view that the two conditions are the result of a common cause which is active after full development in one case and before it in the other.

Psychological Relationship.

This aspect of the question is a difficult one owing to the diversity of opinion of the numerous writers.

Tredgold (1929*a*), in discussing mental instability, concludes that it is due either to temporary neuronic impairment or, more often, to an incidental phase in the course of neuronic destruction and dementia. He says that it is therefore more reasonable to regard it as the result of a disorder or decay of the controlling functions of the mind, rather than as being due to any increase of function. He thinks that the constitutional mental instability would clearly appear to be due to an irregular and imperfect development; from which it follows that psychologically the condition is not merely related to mental deficiency but it actually is mental deficiency.

Meyer (1906), in his fundamental conception, insists that only a particular type of personality is liable to develop dementia præcox, and that the condition is largely due to ill-directed application of the instincts and unsuitable methods of adaptation.

Jung elects to disregard an unknown in favour of another unknown. He says that instead of assuming some hereditary disposition or a toxæmia which gives rise directly or indirectly to organic processes of disease, he inclines to the view that, upon the basis of predisposition, whose nature is at present unknown to us, there arises a non-adaptable psychological function which can proceed to develop into manifest mental disease, and this may secondarily determine organic degeneration with its own train of symptoms.

Campbell (1907) considers that in persons with a constitutional inferiority a long period of unhealthy biological adjustments will result in dementia præcox.

Hoch (1910), in adopting the view that dementia præcox is not an organic

disease, points to the fundamental differences between it and what he terms the plainly organic disorders, such as the other dementias and general paralysis. I agree that it is not to be placed in the same category as these, as is shown by its clinical features and morbid histology, but this is not to say that, therefore, it is not an organic disease. Hoch then describes the special personality which is predisposed to the disorder. He calls it the "shut-in personality", and says it recurs with striking frequency, as the personality existing before the psychosis or before the incubation period. He says that 68% of his own cases had it and that, among other workers, Kirby found 50% with it.

Hoch does not say what this special personality is, or what gives rise to it. It seems to me that it only begs the question, and that it would be just as reasonable to say that the shut-in personality is, in reality, undeveloped dementia præcox. He goes on to refer to the fundamental lack of sexual adaptability in dementia præcox, and quotes Jung on the close relationship between dementia præcox and puberty, the frequent importance of sexual conflicts in the development of the psychosis, and its diffuse and poorly adapted sexuality. He quotes Abrahams, who regards the lack of sexual adaptability as due to an arrest of sexual development. He concludes that the shut-in personality leads to an increase of day-dreaming, that is to say, faulty psycho-biological habits.

Jelliffe (1911), in considering the question of the pre-dementia præcox character, quotes Meyer as saying that children who later developed abnormal reactions of the type of dementia præcox were peculiar rather than defective. He emphasizes disorders of balance as the determining cause of the psychosis. He remarks on the unnatural proneness to fatigue in dementia præcox and quotes Kahlbaum to the effect that intellectual strain contributes to the katatonic variety.

Kraepelin (1919) says of dementia præcox that an essential factor is the weakening of those emotional activities which permanently form the main-springs of volition, so that the essence of the personality becomes destroyed, and that there is loss of the inner unity of the activities of intellect, emotion, and volition in themselves and among one another.

Mott (1920), in considering the results of his investigations, says that the suspension of neuronic function is variable and leads to alteration of symptoms and apparent recovery, and the suppression of neuronic function is incapable of any remission, is progressive, and represents the dementia simplex part. He thinks that the nature of the accompanying phenomena depends on the personality of the individual. He concludes that it is quite probable that in addition to a developmental deficiency of the brain there is a general deficiency of the *élan vital*.

Dawson (1928), reviewing the various psychological theories of the cause of dementia præcox, remarks that many cases appear to have been derived from a type of person which corresponds to the introverts of Jung. On the

other hand, he says the disorder may develop on top of definite congenital defect. In this case the subjects have always had a lower intellectual capacity than normal, and may have shown such propensities as extreme waywardness, obstinacy, and lack of amenability.

The constitutional aspect of schizophrenia has also been considered by a number of writers, including Bowman and Kasanin (1933).

All these psychological theories may well explain the mechanism by which the symptoms and signs of dementia præcox are produced, but they all introduce some unknown factor. Broadly speaking, I see little difference between the "weakening of emotional activities" of Kraepelin, the "deficiency of *élan vital*" of Mott, the "faulty application of instincts" of Meyer, the "non-adaptable psychological function" of Jung, the "unhealthy biological adjustments" of Campbell, the "shut-in personality and faulty psycho-biological habits" of Hoch, the "disorders of balance" of Jelliffe, and the "constitutional mental instability" which, as Tredgold says, is mental deficiency.

Causal Relationship.

It is of interest to review the various theories of the causes of primary mental deficiency and dementia præcox, and the latter will be considered first.

The school which emphasizes psychological causes in dementia præcox consists notably of Kraepelin (1919), Jung, Campbell (1907), and Pollock, Malzberg and Fuller (1934). Of these it may be noted that Jung even discards the factor of heredity. In addition, Meyer (1906), Hoch (1910), and Jelliffe (1911), also believe in the special personality factor. This school denies that the organic findings are of any significance except as a result of the disorder.

The organic theory is supported by Bolton (1914), Mott (1920), Golla (1929), and Davidson (1934). Bolton, in his great classification of mental disorders into two types, amentia and dementia, places dementia præcox in the latter. It is interesting to observe here that he includes manic-depressive insanity in the former. Mott insists on the histological findings of developmental deficiency, also thinks that there is deficiency of *élan vital*, favours the theory of an auto-intoxication which may have some remote connection with the sexual organs, and concludes that the essential cause is an inborn germinal defect. Davidson believes that the cause is a progressive cerebral disease and that the schizophrenic manifestations are biochemical in their source. Golla is the leader of the biological division of this school. He accepts the anatomical findings and his physiological investigations strongly support the organic view.

With regard to mental deficiency, it is clear from the findings of Bolton (1914, 1933), Berry (1932, 1935), Ashby and Stewart (1933, 1935), and others, that the state of the mind is closely related to the pathological findings in the

brain. The condition is present from birth, therefore the cause must begin to act before birth. The accepted theory of the cause is defect of the germ plasm.

Myerson (1933), in discussing the causes of mental deficiency, considers that the most practical working field is in the influence of environment upon the germ plasm and upon the mind and personality. He recommends the study of nutrition before pregnancy, and of the effects of infectious disease upon ovulation. He remarks that the emotional state is no longer considered to be merely mental, but it affects every tissue and the chemistry of the entire organism. He concludes that a likely cause of mental deficiency is a passing infection affecting the germ plasm.

In his admirable summing-up of the question of the relationship of primary mental deficiency, primary psychosis and dementia, Tredgold (1929*a*) remarks that mental deficiency results from imperfect development of cerebral neurones, and that the underlying cause of primary psychosis, in a very considerable proportion of cases, is constitutional mental instability. He has previously shown that this instability must be regarded as coming in the category of incomplete mental development. He says that in a small proportion the psychosis arises in the absence of any signs of instability and this is due to inherent lack of neuronic resistance and not to structural imperfection. Dementia, he says, results from progressive degeneration of cortical neurones. In the ordinary senile form this is due to age. In general paralysis and arteriosclerotic dementia it is due to acquired disease. In certain pre-senile forms, and particularly in dementia præcox, it is due to inherent defect of neuronic durability. The neurones undergo degeneration because their inherent vitality is insufficient to enable them to withstand the normal stresses and strains of life, and in many of the dementia præcox forms the defective durability is superadded to a defect of the structure.

Referring again to the three forms of mental disorder he says that the great majority of cases have in common that they are the result of inheritance, and are the offspring of a psychopathic or neuropathic stock. They are germinal, although not directly inherited or transmitted. He concludes that therefore the three forms are most closely related and are merely different manifestations of one and the same condition, namely, peculiarity of the germ material.

Conclusion.

I consider that Tredgold's view of the relationship of the three great classes of mental disorder, primary deficiency, primary psychosis and dementia, is most useful and helpful. I have tried to carry it a little further and to show that there is a still closer relationship between primary deficiency and the most important member of the second class, namely, dementia præcox.

Evidence has been produced of the extent to which insanity occurs in a

particular variety of mental deficiency and the predominant type is shown to be dementia præcox.

The work of others has been reviewed from clinical, pathological, psychological and causal points of view. Dementia præcox is shown clinically to have much resemblance to unstable types of mental deficiency. It is demonstrated that the pathological findings in the two conditions will bear comparison. The evidence for the organic nature of dementia præcox is seen to outweigh that for the psychogenic view, and thus it is not to be separated from mental deficiency on psychological grounds. Finally, the causal relationship is inferred from several sources and is clearly indicated by the work of Tredgold.

Some investigators have noted a relationship between mental deficiency and the biogenetic psychoses, but this paper shows that, of the two psychoses, dementia præcox deserves the more attention in this connection. Little light has been thrown on the all-important question of the cause of mental disorder, but it may clarify the problem to direct attention to the probable existence of a common cause for primary mental deficency and dementia præcox.

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